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DISCUSSION ON INFLUENZA.

Sir Arthur Newsholme, K.C.B., M.D.

When I promised to open this discussion it could not be forecasted that we were about to experience our national share of a pandemic of influenza unexampled both in distribution and severity. In present circumstances a discussion on influenza will necessarily include consideration, so far as our lack of knowledge will permit, of the conditions leading to the minor epidemics with which in recent years we have been fairly familiar, and those leading to the mysterious occurrence of the wider pandemics, such as those of 1890 and of the present year.

It is necessary, in my view, to consider these minor epidemics and the endemic prevalence of influenza in this country in relation to bronchitis and pneumonia, which year by year cause a much heavier toll on health and life than is demanded by influenza. I have little hope of success of any personal or public measures against influenza which will not influence even more favourably the incidence of bronchitis and pneumonia. It is not improbable, in fact, that the influenza problem may be most successfully attacked when influenza is not epidemically prevalent.

ETIOLOGY.

The first difficulty is to define influenza. Is it one disease or a group of diseases? And is the disease now prevailing the disease which prevailed in the spring, and still more in July, of this year? These
two questions cannot easily be separated. The symptoms were usually milder in the earlier than in the present outbreak, but that may be due to the very exceptional culmination of the epidemic in the summer months. War conditions prevented civilian bacteriological work on the disease on a considerable scale, but I am hoping that this discussion will elicit contributions as to the bacteriology of the July and the present outbreak, and that later the results will be available of the inquiries by the Medical Research Committee, who have been collating evidence from military as well as civilian outbreaks, and making independent inquiries.

The report of the Influenza Committee of the Advisory Board to the Director-General, Medical Department, France,\(^1\) shows that influenza has occurred at intervals among our forces in France, and that a serious epidemic occurred in the spring of this year. The known facts bearing on the position of Pfeiffer's influenza bacillus as the cause of the epidemic are well summarized in this report. This bacillus cannot be said to have established its position, nor can it be disregarded; for even if some other organism be the essential virus of influenza, the Pfeiffer bacillus is probably one of the chief sources of the damaging complications of this disease.

From this country, from France, and from America there is evidence that occasionally influenza kills rapidly by toxæmia, but more often by pulmonary complications; and that in producing these complications hæmolytic streptococci have been predominating organisms, the Pfeiffer bacillus and the pneumococcus also being found in a varying proportion of cases. The increase in virulence of streptococci in animal passage is well known. There can be no doubt that all these bacteria, often acting in conspiracy, have contributed greatly to the recent mortality from influenza, but whether there is in addition a hitherto undiscovered virus to which influenza is primarily due is still a moot point.

The occurrence of such mixed infections is well known in other diseases; it is a chief source of mortality in measles, and success in the sanatorium treatment of tuberculosis depends largely on the elimination of secondary infections; and this points to oral, nasopharyngeal, and dental hygiene, and life under improved sanitary conditions as at present among the most hopeful lines of action in diminishing the mortality from bronchitis, pneumonia, and influenza in the general population.

Influenza as a Clinical Entity.

In view of the uncertainty in the bacteriological diagnosis of influenza, can this disease be recognized as a clinical entity? In typical cases it can, but certainly not in a very large proportion of atypical and modified attacks. In France, for instance, it has been found difficult to diagnose influenza from trench fever when the latter is unaccompanied by relapses. The main diagnostic evidence of the presence of influenza is its occurrence epidemically or pandemically. Diagnosis by this means is not helpful in prophylaxis, and there is the further possibility that other catarrhal infections may—though there is little evidence of this—take on transcontinental travels. This probably happens with the meningococcus, causing in most recipients little or no evidence of disease.

But the facts remain that influenza is a specific disease recognizable in severe outbreaks, and that it is a disease which, with the exception of plague and cholera, has on occasion travelled farther and more rapidly over the world than any other recognized disease. It differs from these two diseases in that their pandemic course can be stayed by organized measures which are well within human capacity. I know of no public health measures which can resist the progress of pandemic influenza. It is noteworthy that the relative failures of preventive medicine and public health to secure control over disease have been concerned with diseases in which infection is received by the respiratory tract—especially influenza, bronchitis, pneumonia, measles, and whooping-cough.

Death-rates from the Chief Catarrhal Diseases.

We need further knowledge—epidemiological, pathological, and bacteriological—of all these diseases before they, and especially before influenza, can be brought within the scope of preventive medicine. Acute respiratory diseases, chiefly bronchitis and pneumonia, cause approximately one-sixth of the deaths from all causes; and this does not include the deaths in measles and whooping-cough chiefly caused by respiratory complications, and the deaths under the heading of other diseases in which respiratory complications turn the balance against recovery.

In fig. 1 is shown the general course of the death-rate in this country since 1881 from the three chief fatal catarrhal diseases. The death-rates are given as relative figures, all the rates being reduced to
the same magnitude so that a comparison may be made of the percentage variation under each heading. The variations in the death-rate from influenza are evidently greater than those for pneumonia and bronchitis, though in nearly every instance they are in the same direction in any given year. The decline in the death-rate from bronchitis without corresponding increase of pneumonia points to the conclusion that much of the decline under the first heading is real and not due to changing fashion in certification.

In figs. 2 and 3 this comparison between deaths in London from influenza, bronchitis, and pneumonia is pursued further, the four-weekly incidence of mortality from each of these diseases being shown on the graph, for the four years 1889-92, in three of which influenza was epidemic, and for the four years 1915-18, in each of which some rise of the influenza curve occurred. In three of these last years the rise followed the almost universal rule of winter or spring incidence, the exception being the larger peak of July, 1918, and the still higher.

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1 It should be noted that, even in years in which influenza is epidemic, bronchitis and pneumonia cause several times as many deaths as this disease. Probably many of these deaths are secondary to influenza.
present peak. Several important points may be gathered from these graphs. Pneumonia and bronchitis nearly always have their maximum mortality in the winter months. In 1891 excessive mortality from both bronchitis and pneumonia was prolonged into the late spring, coincidently with a spring epidemic of influenza. In 1918 the summer outbreak of influenza was not associated with marked increase in the pneumonia death-rate, though it is likely that the excessive pneumonia mortality characterizing the whole of 1917 and 1918 was associated with fairly widespread prevalence of influenza.

**Mortality from Influenza since 1847.**

The course of influenza in this country from 1847 can be gathered approximately from the annual death returns as displayed in fig. 4. A preliminary remark is required. Our national death statistics are the least imperfect in the world; but the accuracy must always necessarily be limited by the extent to which medical certification of the cause of death is satisfactory. In the earlier epidemics of influenza, including the outbreaks of 1890-93, there is strong reason for concluding that deaths from influenza were certified under the heading of the pneumonia or bronchitis secondary to influenza in a much higher proportion than in recent years. This probability is increased by the large mass of endemic influenza revealed in fig. 4 from 1890 onwards, but almost
absent in earlier years. The difference is in part probably a real difference, though probably not entirely so. Although much of this increased mortality registered from influenza may not really be this disease, we must, I think, assume that conditions since 1890 have become more favourable than in earlier years for the continuous prevalence of influenza endemically on a considerable scale.

Influenza, Cerebro-spinal Fever, and Pneumonia.

I have introduced into fig. 4 (scale five times that for influenza) a further curve showing the annual death-rate from cerebro-spinal fever since 1881. It is suggestive that this disease scarcely maintained a footing in England during 1881-1914, but since then has prevailed each winter epidemically, although each case as a rule occurs sporadically, in the sense of not being traceable to any other case of the same disease. This difference in the annual curves of influenza and cerebro-spinal fever does not appear to support the seductive hypothesis of Dr. Hamer that both these diseases may be diverse manifestations of the same virus.

On the other hand, fig. 5 may be examined. This gives a comparison between the four-weekly incidence of cases of cerebro-spinal fever and of pneumonia in the British Army in the United Kingdom during the four years 1915-18. This curve is introduced by permission of the Director-General of the Army Medical Department. The two
diseases are plotted out on the same scale, the scale being omitted. The cases are enumerated as first notified without subsequent correction, but this does not detract materially from the general value of the curves for comparative purposes. On the same diagram I have plotted the number of deaths from influenza in four-weekly periods in London. The two sets of figures, although derived from different sources, may be regarded as roughly comparable.

![Diagram showing the course of influenza mortality in London]

There is shown a close correspondence in the curves of these three diseases during three out of the four years, and there can be little doubt that the same winter and spring conditions favoured excess under all three headings. The experience of 1918 is instructive. In this year influenza had an epidemic peak in July, and the cases of pneumonia in the Army showed a corresponding peak. Cerebro-spinal fever, however, did not follow the two other diseases.

**Course of Influenza Mortality in London.**

The course of influenza in this country from year to year has been shown in figs. 1 and 4. Its weekly course, judged by weekly deaths from influenza registered in London, is shown in fig. 6A, B, C. Prior
to 1890 deaths from influenza scarcely figured in the death returns. From this year the course of events may be briefly summarized thus:—

1890.—An epidemic culminated in the third week of January. The epidemic peak occupied about five weeks, the excess of influenza

![Fig. 6a.](image1)

gradually tailing off subsequently in this and in the later epidemics shown in fig. 2. Between the peaks of this and of the next epidemic seventy weeks elapsed.

1891.—An epidemic occurred culminating in the twentieth week of

![Fig. 6b.](image2)

![Fig. 6c.](image3)
the year. The epidemic peak occupied eight to ten weeks. Between
the peaks of this and of the next epidemic thirty-five weeks elapsed.
1892.—An epidemic occurred culminating in the third week of
January. The epidemic peak occupied six to eight weeks.
1893.—No great epidemic occurred, but some excess of mortality
from influenza for several weeks in the early spring. A smart epidemic
culminated in the fiftieth week of the year, the epidemic peak occupying
about seven to nine weeks.
1894.—No epidemic, except the one beginning in 1893.
1895.—A large epidemic culminated in the tenth week of the year,
the epidemic peak occupying six to eight weeks.
1896.—No epidemic.
1897.—No considerable epidemic.
1898.—An epidemic occurred which reached its rather low maxi-
mum in the fifth week of the year. Its duration was somewhat
protracted.
1899.—A somewhat similar epidemic to the one in 1898 culminated
in the eleventh week of this year.
1900.—An epidemic occurred in December, 1899, and January, 1900,
the greatest mortality occurring in the second week of January, 1900.
The peak of this epidemic occupied six weeks, the mortality slowly
tailing off. The interval between this and the preceding peak was
forty-two weeks.
1901.—No epidemic.
1902.—A small epidemic reaching its maximum in the eighth week
of the year. The epidemic lasted six to eight weeks. An interval of
over two years had elapsed between this and the last previous epidemic.
In 1903, 1904, and 1905 no epidemic of influenza occurred, though
occasionally the weekly deaths from this disease rose slightly above the
usual inter-epidemic level.
1906-07.—An epidemic occurred in December, 1906, and January,
1907, of low magnitude and somewhat protracted duration.
1908.—A smarter epidemic of influenza occurred, culminating in
the ninth week of the year. Its duration was six to eight weeks, the
tailing off commonly seen in other epidemics being shown in the
curve.
1909.—An epidemic occurred with its maximum in the twelfth
week of the year, similar in course and duration to the epidemic in
1908. The interval between these two epidemic peaks was fifty-five
weeks.
1910-11-12.—No epidemics.

1913.—An epidemic of low range occurred with a maximum mortality in the twelfth week of the year.

1914.—No epidemic.

1915.—An epidemic similar to that of 1913 occurred, the low maximum being reached in the eighth week of the year.

1916-17.—A considerable low-ranged increase of mortality was visible in the early part of 1916, followed by a relatively small epidemic, culminating in the last week of the year and terminating early in 1917.

In the previous statement deaths registered as having been caused by influenza only are dealt with, and I have selected the metropolis because for it we have an uninterrupted statement of weekly deaths from various causes for a long series of years. It will be noted, however, that London is a densely populated province, and that its epidemic curves represent the smoothed-out results of many local outbreaks which have rapidly become general.

The Pandemic in 1918.

Reference to fig. 6c shows that influenza during this year has followed a course never previously experienced. Until towards the end of June the London curve showed scant mortality from influenza, the number of weekly deaths between the eighteenth and twenty-fifth weeks varying from five to ten. There is evidence that prior to this influenza had been fairly widely prevalent, as it could not fail to be, inasmuch as during the spring of this year influenza prevailed seriously among both the allied and enemy troops, and in Spain the epidemic of influenza had got well ahead in May. In July influenza was widely prevalent and was causing excessive mortality (see fig. 5) not only in England, but also all over Europe, in the United States, in the great cities of India and elsewhere.

I have not found another instance of a widespread epidemic culminating in the summer months (i.e., after June 22). The London curves, with this single exception, show that since 1881 epidemic peaks have always occurred in winter or spring. Even in the epidemic culminating in July last streptococcal infection had been noted by various observers; and it is noteworthy that in American accounts of outbreaks of influenza in camps and in the civilian population of America during both 1917 and 1918 there were reports of this same
preponderant occurrence of haemolytic streptococci in the lesions and secretions. During the spring and throughout the summer of 1918 the usual endemic influenza probably was being almost constantly replenished by more virulent infection on a very large scale from overseas from both the American and European continents.

The further course of influenza in this year presents altogether exceptional, if not unique, features.

The curve shows clearly that in London after an epidemic causing excessive deaths during some six weeks in July and August—weekly deaths from influenza 10 (twenty-fifth week), 67, 218, 287, 192, 86, 38 (thirty-first week)—the weekly deaths declined, the deaths in each of the following weeks being 21, 20, 12, 12, 12, 14, 9, 17, 17.

In the forty-first week occurred a sudden increase of deaths to 80, and in following weeks the deaths numbered 371, 1,256, 2,458, and 2,433.

Reference to fig. 6c shows that this experience, as already stated, is different from that in any preceding year:

The interval between the peak of the July epidemic of 1918 and of the smaller epidemic in 1916-17 was eighty weeks; the corresponding interval between the July and the October to November epidemic peaks in this year was about sixteen weeks. This last-named interval is less than half of the interval elapsing between any two previous epidemic peaks of influenza in the metropolis, the nearest approach being thirty-five weeks interval between the epidemic peaks of 1891 and of 1892.1

THE QUESTION OF A SECONDARY WAVE.

The Secretary of the Medical Research Committee in the British Medical Journal of August 10, 1918, p. 139, at the end of an annotation headed "Bacteriology of the Influenza Pandemic," asked bacteriologists in this country to send results of their observations during the epidemic—

"In order that the results gained here may be collated, with a view to the organization of such co-ordinated work as may be found possible for the study of the secondary waves of infection that are to be expected."

1 I notice that Captain Greenwood, in the Lancet of November 3, 1918, quotes the solitary experience of a small agricultural district in Weardale in 1891 as a further example of a July epidemic followed by an October outbreak. The scattered character of this district is more consistent with the view that two independent outbreaks occurred; furthermore, we are concerned not with village outbreaks, but with the general trend of national events.
This statement was, I fear, overlooked by many bacteriologists as it was by myself; otherwise many who did not know would, I am confident, have sent contributions for the common stock. But in some journals an attempt has been made to show on the strength of the above quotation that public health authorities, central and local, did not take all possible steps in anticipating and mitigating the present major epidemic of influenza.

Furthermore, in an answer to a Parliamentary question, the above cautious statement has been asserted to have included also a forecast that (a) the secondary waves would occur in the autumn, and (b) they would be more severe in character than the "primary wave of last spring."

No such forecast as (a) was made, and it would have been a foolishly wild guess, inasmuch as the present secondary wave occurred more than twice as early as any previous "secondary wave" recorded in the history of the metropolis. (I know of no other place for which equally minute and accurate statistics are available.)

That this is so is further shown by the following statement taken from the monthly Medical Supplement compiled by the Medical Research Committee, dated October 1, 1918:

"It is, unfortunately, only too certain that a second and more severe wave of influenza will spread over Europe in a few months."

No special foresight is needed to anticipate that influenza, if it recurs in winter, will be a more serious disease than in July; but when this last statement—which, it may be observed, is inconsistent with other published statements—was made the "secondary wave" already was well on its way to an unexampled maximum, having caused during September serious mortality in South Africa, the United States, India, and some parts of Europe.

If the forecast in the last quotation is to be verified it will mean a further epidemic of influenza which has not yet materialized.

**Difficulty of Preventive Measures in War Time.**

The point would not be worth pursuing, except for the implication that more could have been done to avert the present pandemic; and it is only to elucidate this point that I make a further observation. Warnings of possible "secondary waves" in the presence of much endemic influenza, or immediately after a superadded epidemic has
declined, would be useful if a prophylactic were available (none except one in the early experimental stage has been suggested), or, if by issuing advice the progress of an epidemic could be stayed. Neither of these conditions can be fulfilled. We are at present unable to prevent the spread of influenza by communal means, and the experience of every family invaded by influenza demonstrates the difficulty, amounting almost to impossibility, of preventing the spread of infection in the domestic circle.

Notwithstanding this, I did in my official capacity prepare in July last a memorandum for public use, but on the balance of considerations its distribution was not considered expedient at that time. There are national circumstances in which the major duty is to "carry on," even when risk to health and life is involved. This duty has arisen as regards influenza among the belligerent forces, both our own and of the enemy, milder cases being treated in the lines; it has arisen among munition workers and other workers engaged in work of urgent national importance; it has arisen on a gigantic scale in connexion with the transport during 1918 of many hundreds of thousands of troops to this country and to France from overseas. In each of the cases cited some lives might have been saved, spread of infection diminished, great suffering avoided, if the known sick could have been isolated from the healthy; if rigid exclusion of known sick and drastic increase of floor-space for each person could have been enforced in factories, workplaces, barracks and ships; if overcrowding could have been regardlessly prohibited. But it was necessary to "carry on," and the relentless needs of warfare justified incurring this risk of spreading infection and the associated creation of a more virulent type of disease or of mixed diseases.

The same problem has arisen in connexion with crowded trains, trams, and omnibuses. These, doubtless, are prolific sources of infection, but the service cannot immediately be increased, and meanwhile the vast army of workers must not be impeded by regulations as to overcrowding of vehicles in their efforts to go to work and to return home, and I have had no hesitation in recommending the Local Government Board to advise police authorities to this effect.

The Problem of Preventive Measures in Times of Peace.

But we may consider the preventability of influenza from the standpoint of possibilities in time of peace, and ask if we are prepared to pay the heavy price in personal restrictions which its prevention—
if even then it be possible—will necessarily imply until further means of prevention, so far undiscovered, become available.

The problem of preventing influenza is part of the wider problem of the prevention of the entire group of catarrhal diseases, with the added problem as to whether the occult causes of pandemicity of influenza can be discovered and their action averted. Hitherto we have no clue to these, and our only hope of improvement lies in means to be devised for preventing catarrhal diseases, by diminishing infection, by reducing susceptibility, or both.

The measure of our present impotence is given by the inability of medical science to ensure us against acute nasopharyngeal catarrh or a "common cold." I do not overlook the occasional "hits" of autogenous inoculation. A "common cold" does not appear in our national death statistics; but probably it is a more frequent cause of diminished efficiency, if not also of death, than any of the acute infectious diseases, with the possible exception of measles and whooping-cough, not only by occasionally initiating ear abscesses, purulent infections of the bony cavities of the skull and face and meningitis, but especially because of its common ending in chronic bronchitis, and because it opens the way for meningococcal, pneumococcal, streptococcal, and tuberculous infections. And yet we have no entirely trustworthy means of preventing common colds. The nearest approach to a general truth in regard in these colds is that life continuously in the open air secures relative immunity from attack.

An initial practical difficulty in such prevention is that the patient for several days may not fully recognize his condition, and it seems likely that infection is chiefly spread during these earlier stages. It remains true, however, as stated in my recent official Memorandum, that—

"If every person who is suffering from . . . catarrh . . . took all possible precautions, the present disability and mortality from catarrhal epidemics would be materially reduced."

**Unknown Causes of Pandemics.**

I have referred above to the unknown causes which, for instance, in 1803, 1833, 1837, 1847, 1890, and again this year, have been responsible for world-wide spread of influenza, limited in its rapidity only by the speed of human travel, and multiplying so rapidly as to create the illusion of almost simultaneous origin in many countries.
One somewhat improbable hypothesis is that nearly simultaneous enhancement of the potency of endemic influenza virus occurs throughout the world. A fair working hypothesis is to regard influenza as a specific infectious disease which is always present sporadically in urban communities, widespread epidemics of it only occurring when a more exalted strain of the same virus is introduced from without.

The source of the more exalted virus can only be guessed. If we were to confine our outlook to the present pandemic it would be easy to blame the vast meeting of men of different nations under circumstances of overcrowding in large masses in this and other countries, some of whom had had no previous opportunity of acquiring even temporary immunity. But such an explanation fails to explain the earlier pandemics, which spread apart from exceptional world movements.

In the 1890 epidemic foreign origin and rapid spread of imported infection undoubtedly occurred in this country; it is highly probable that in the present year events have pursued the same course.

It has been surmised that the accidental contact of the hypothetical influenza virus with a native population which has not had the opportunity of acquiring relative immunity from attack or death leads to an enhancement of the virulence of the contagion, and that it is thus enabled to commence its travels. This hypothesis does not appear to me to be satisfactory. The initial experience of the Fijians in regard to measles will be quoted in support; but those who re-read Dr. Corney's original account of that outbreak, as I have recently done, will note that the early cases in this first outbreak in the islands of Fiji were mild in character, and that among portions of the population who were under hygienic and medical control the fatality was low at other stages of the devastating outbreak. Experience of measles in armies is that it is sometimes as serious as on "virgin soil." It was so in the American Civil War, and it has been extremely severe in the present war. It is possible that this has occurred especially among the New Zealand and other troops from the remoter parts of Britain and of Greater Britain; but the same troops were simultaneously suffering from massive invasion by Pfeiffer's bacillus, by streptococci, and by pneumococci. The excessive fatality of measles in warfare is explicable by the combined invasion of several viruses, without need for recourse to the hypothesis of a specially unprotected population.

The measles problem is very similar to the influenza problem. In both, so far as our present knowledge goes, the chief hope of saving life lies in preventing, or in diminishing, the mortality from the strepto-
coccal and pneumococcal pneumonia in these diseases, and research work in these directions is greatly needed.

It is evident that we are ignorant of the causes of pandemics of influenza at irregular intervals, and their occurrence has never, so far as I know, been anticipated.

We are still obliged to think of unknown factors; and I can repeat the admission of similar ignorance in regard to cerebro-spinal fever made by me elsewhere:1

In epidemic times "strains make their appearance which possess greater intrinsic capacity for invading the tissues than the strains current in non-epidemic seasons. As epidemics are intermittent, this emergence of more virulent strains must be due to intermittent causes; and it is still necessary to assume the action of some unknown influence, telluric, climatic, or other, which becomes operative under conditions of which we remain ignorant."

INFLUENCE OF THE WAR.

But although no satisfactory explanation is known of the causes exciting either the minor or the major epidemics of influenza, there can be no doubt that the movements of the recent war have been responsible on a large scale for its increased virulence. During the summer months of this year migration from country to country, from America, Canada, Australia, New Zealand, and South Africa to England and France was occurring each month to an extent of several hundred thousand persons, and migrations in the opposite direction were also taking place on a large scale. The voyages of these troops occurred at a time of submarine dangers with a necessarily limited supply of ships; furthermore, influenza and streptococcal pneumonia were previously prevalent in oversea camps; and it would have been surprising had this marvellously rapid and successful transportation of troops been carried through without a heavy cost in communicable disease. There is no reasonable doubt that through these months contagia possessing enhanced virulence were being brought into this country. The circumstances, in fact, bear a close resemblance to those under which cerebro-spinal fever first became considerably epidemic in this country in 1915.

In attempting to trace the course of influenza in the present year it must be noted that the story cannot be told completely until Army

Preface to Reports on Cerebro-spinal Fever (Local Government Board, N.S., No. 114).
and Navy statistics and civilian statistics for many countries become available, and any views now stated may need revision as the result of further national and international information.

**Increasing Control of Disease.**

It will be evident that we have a long road to travel before acute catarrhal affections can be brought within human control. The list of preventable and curable diseases has steadily lengthened. A first group of these comprises typhoid and typhus fevers and small-pox, which are entirely controllable and in large measure controlled. Dysentery, cholera, malaria, and plague also have their place in this group. A second group of partially controlled diseases comprises diphtheria and scarlet fever, although like rheumatic fever, these diseases are subject to cyclical changes in distribution related to years of relative drought. Tuberculosis and syphilis are in the same group, and there is no adequate reason against their early transfer to the first group.

Measles, whooping-cough, acute poliomyelitis, and cerebro-spinal fever form a group in which infection appears to arise similarly to that of ordinary catarrhs and influenza, and their prevention so far similarly is only possible to a limited extent. For measles, whooping-cough, catarrhs, and influenza the chief present hope consists in attempts to diminish attack by or death from pulmonary complications.

**Desiderata.**

I may in conclusion indicate a few early desiderata in the investigation of influenza:—

(1) It would greatly facilitate further investigation if epidemic catarrhs, whether influenzal or other, were studied bacteriologically, especially in their early stages in selected populations, e.g., of institutions—and if a similar study were made of cases of bronchitis and of pneumonia in different localities and for a series of years. The work already begun by the Medical Research Committee will doubtless be of value in this and other directions.

(2) If such collective information is to have its maximum utility it will be necessary to ensure that observations by different bacteriologists are made by comparable methods.

(3) The study of the epidemiology of influenza during the present war has been hampered by the necessary secrecy as to military and

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naval outbreaks, and by the failure of the ordinary international information coming into our official possession from consuls, foreign journals. &c. It will be possible partially to piece together the story of the present outbreak from the reports which medical officers of health have been requested to prepare; but information from the services will be needed to furnish a less incomplete story.

The civilian reports from different districts, so far as they have reached me, show a remarkable change in the age incidence of deaths from influenza during the present year; so much so as to have led to a not infrequently expressed opinion that we must be concerned with a different disease. Dr. Stevenson, in his contribution to the present discussion, will throw further light on this and other points.

Possibilities of Control.

The control of epidemic catarrhs may be considered from the point of view of the prevention of attack, or the prevention of complications. The possibilities of prevention of attack pending further medical discovery are limited; but under peace conditions the avoidance of aggregation of large units, especially for sleeping, are among the obvious indications. Whether persons affected with catarrh will in the future submit to wear veils or respirators is doubtful; but nurses of influenza patients should adopt this precaution. The utility of prophylactic vaccines remains to be demonstrated.

The prevention of pneumonia by vaccines is a more hopeful possibility; and it is possible that greater attention to the hygiene of the mouth and naso-pharynx both before and during attacks of influenza would have good effect. The gentle irrigation of nose and throat with a weak saline or antiseptic solution appears to have succeeded in diminishing pulmonary complications in measles, and might be equally beneficial in influenza.

The present pandemic will not have been suffered in vain if intensive investigation is pursued on catarrhal diseases in general, and if these great causes of national disability and mortality are brought more nearly under control.
Dr. T. H. C. Stevenson, C.B.E.

I wish to point out by means of a few diagrams relating to London and Paris certain points in which the present epidemic appears to me to differ from those of the past twenty-seven years.

The sum of the four columns relating to any one year amounts to 100 per cent. The two sets of columns relating to the year 1918 refer to the July and October outbreaks, the latter being covered up to November 2 only.

(1) The first diagram shows that its intensity is greatly in excess of that of any of its predecessors. The excess of deaths from all causes over the average for the corresponding week of the previous ten years, corrected for growth or decrease of population, has been taken as representing the total influenzal mortality. Probably this understates the facts, as the diagram shows that, owing to the steadily declining death-rate, the deaths before and after each epidemic were distinctly below the mean. It is important to get some measure of total influenzal
mortality, as the diagram shows that, except in 1918, only a minority of the deaths ascribable to influenza have been so certified. The excess mortality of the present epidemic is understated in the weekly returns, which refer only to civilian deaths, owing to the absence on military service of a large portion of the population at the ages at which mortality is greatest. Although the curves relate to numbers of deaths, not death-rates, they serve as a very fair comparison of the mortality experienced this year and in the four principal previous outbreaks (1891, 1892, 1895 and 1900) owing to the stationary character of the population of the county of London during the period dealt with.

(2) The second diagram, which represents the proportion of the total excess mortality ascribed to influenza, to respiratory, to circulatory, and to all other causes of death respectively in the four chief previous outbreaks and in the two outbreaks of the present year, illustrates a second difference of the latter from the former. In the earlier outbreaks a minority only of the deaths (27 to 41 per cent.) were ascribed to influenza, most appearing as excess deaths from respiratory diseases, but in the outbreak of last July 54 per cent., and in the present outbreak up to November 2,77 per cent. of all the deaths were returned as due to influenza. This may be because the victims of this year's outbreaks have been much younger than in the other epidemics, for it may be, as in the case of phthisis, that in certifying a slight attack is more likely to be passed over in favour of the bronchitis or pneumonia it has given rise to, in reporting the deaths of elderly patients.

(3) The third diagram shows that the present outbreak in Paris is of the same character in this respect as our own, 65 per cent. of all the excess deaths being ascribed to influenza.

(4) The next diagram (reproduced above) illustrates the sudden and startling change which has occurred in 1918 in the age-distribution of influenza mortality. The percentage of deaths at 0-25, 25-45, 45-65, and 65 and upwards respectively in each year from 1890 to 1917 inclusive is shown, as well as that in the two outbreaks of this year. It is seen that in all previous years the majority of deaths—generally about 70 per cent.—occurred at ages over 45. But in July of the present year only about 30, and in October about 20 per cent. of the persons dying were over 45 years of age. In this respect as in regard to forms of certification the July epidemic was intermediate in type between the present one and those of earlier years. Only 5·5 per cent.
of the deaths in this outbreak have been at ages over 65 as against an average of 37 per cent. for the years 1890-1917.

(5) The next diagram shows that in Paris exactly the same change in age-distribution has occurred as here. The extent of the change is of course under-represented in both cases owing to the absence of so many young men on military service.

(6-8) The next three diagrams compare the dates of appearance and disappearance of mortality ascribed to influenza, and its growth and decline intermediately, with the corresponding facts for the excess mortality from respiratory and circulatory diseases and other causes of death respectively. They show that in the four previous epidemics illustrated the excess mortality not ascribed to influenza appeared sooner and generally reached its maximum and declined earlier than influenza certified as such. The latter returns are apt to remain in excess for many weeks after the epidemic, as tested by excess mortality from other causes, has come to an end. In July last the mortality from influenza and the excess mortality from other causes appeared and grew almost concurrently, but the delayed fall from influenza is apparent. In October the other causes once more preceded influenza—the order of disappearance cannot yet be illustrated.

(9) The last diagram, referring to the present outbreak in Paris, shows that the experience there has been exactly the same. It also shows that the wave length in Paris has been somewhat longer than in London, the maximum mortality not being reached till the sixth week. As there was no previous outbreak in Paris during 1918, none at least seriously impressing the mortality returns, the present outbreak there compares in this respect with ours of July, which had a particularly short wave length, attaining its maximum in the third week as against the sixth in Paris, and the fourth in most of our previous outbreaks.

Mr. M. Greenwood.

The first point I wish to raise for discussion is as to how far the present epidemic can be regarded as in any sense unique, and to settle that point we must first compare the 1889-94 period with its predecessors. Sir Arthur Newsholme put forward the view that the sequence of events in 1889 and following years formed a new phenomenon in the history of influenza, and that view, first emphasized by Dr. Charles Creighton, has a great deal in its favour; but it is well to recollect that
in the earlier history of influenza there have been indications of excessive mortality in the year following the primary epidemic. An instance of this is the influenza of 1675, described by Sydenham; in the following year, the constitution of which is not given, there were excessive deaths, according to the bills of mortality, though small-pox was at a low ebb. The same remarks apply to the epidemics of 1679 and 1782. In 1782 there was one of the most famous outbreaks of influenza in this country, culminating in June. This was the first epidemic for which the word influenza was in general use, and it gave rise to the largest additions to medical literature on the subject previous to 1889. The mortality of 1782 was less than that of 1783. Then with regard to the form of primary influenza waves, it can hardly be demonstrated exactly on mortality statistics, and for 1889-90 the only available incidence figures are those for Munich. The diagram\(^1\) represents the course of events in Munich from December, 1889, to February, 1890, constructed from daily notifications. It is characterized by an almost complete symmetry. It is important to notice the difference between primary and secondary epidemics in this connexion: the primary is a quasi-symmetrical phenomenon, whereas the secondary is usually asymmetrical. In the next diagram the full line represents the deaths for the epidemic of 1889-90, and the broken line represents the secondary epidemic of 1891. They are plotted to the same unit, and the maximum week of each is taken as 100. It will be seen that the decline of the 1891 outbreak is decidedly slower than that of the primary influenza of 1889-90. We have now to consider the affinities of the existing epidemic. This next diagram is constructed from data which I owe to the courtesy of the Royal Air Force. The maximum is the number of cases, again put as 100, in the worst week, and the diagram is a correct representation of the incidence upon the Royal Air Force at home, careful allowance having been made for alterations in the numbers of men at risk. If one takes the left-hand part of that diagram and compares it with the Munich incidence curve reduced to the same scale, the worst week of one epidemic corresponding to the worst week of the other, there is from the epidemiological point of view an absolute coincidence between the 1918 summer epidemic and the primary epidemic of thirty years ago. There is nothing unique in the epidemiological form of the disease of last summer. I now put before you the experience of the national munition factories. A different

unit has been used here, the method being to plot the percentage of
time lost through sickness week by week and to note the period when
the sickness rose above the average level and again sank to it. The
diagram once more shows a symmetrical or quasi-symmetrical rise and
fall. This is brought up to date, and one sees in the new curve a slower
rise and also that the height reached is nothing like the same as in the
summer. It should be remembered that we are dealing with incidence,
and the case incidence of the current epidemic is less severe than
the incidence of the summer epidemic. From comparison with the
epidemic of the nineties, it appears to me that a complete formal
identity has been made out between the two occurrences. The primary
symmetrical form has come first, and, what we are now experiencing,
the secondary asymmetrical outbreak later. There are two points to be
considered. The first is the time interval between successive epidemics.
Sir Arthur Newsholme's method of taking the distance between the
two apices of the curves is open to criticism, because the shapes of
the curves are dissimilar. A better method is to take the interval
elapsing between reaching the endemic level in the first and rising
above it in the second period; doing so, we find that the present
interval is not very much shorter than what was found before.

With regard to the question of fatality, no doubt the fatality has
been much higher than in the previous epidemic, and the point is
whether or not this is due to special circumstances. Sir Arthur
Newsholme suggested overcrowding as a cause; fuel shortage also may
have helped. Food difficulties are very unlikely to have had anything
to do with the matter. As the Society is aware, the course of the public
health in any community before an outbreak of disease indirectly due
to food shortage is very well known: there is a slow, steady rise in
general morbidity until there is a specific outbreak of disease, the
character of which differs according to circumstances, as, for instance,
dysentery in the Millbank penitentiary, or pneumonia in the case of our
gallant fellow-countrymen at Kut-el-Amara, and so on. In any case,
before the specific outbreak there is a steady rise in morbidity.
My chart represents the time lost in national munition factories by
sickness over a year. The general course of these charts shows the
usual rise in the sickness curve, due to the autumn colds, and another
rise again due to the spring colds, but no steady upward trend, and the
same thing comes out both in the case of the men and of the women.
When one remembers that the incidence of influenza has been equally
severe upon our well-fed troops, their equally well-fed Allies, and the

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civilian population, we are entitled to conclude that rationing has had nothing to do with this epidemic. I suggest that it is congruent with previous outbreaks, while minor special factors from the epidemiological point of view are overcrowding, and, possibly, fuel shortage. I agree with Sir Arthur Newsholme that one could not have predicted this epidemic with certainty, but it seems to me, in view of the whole history of epidemics of influenza, particularly of the course of events since 1889, such an outbreak should have been regarded as likely to occur, and indeed was so regarded in the Ministry of Munitions which circularized the managers of such hostels as it had to inspect and advised them to make provision for the nursing or removal of cases to hospital as they occurred, in fact, gave them such advice as it was in the power of the Ministry to give. These seemed to be the only precautionary measures it was possible to adopt.

Dr. W. H. Hamer.

I should like to draw a distinction between two things: as to the first there is apparently agreement, it being now admitted that there is a close relationship between outbreaks of cerebro-spinal fever, poliomyelitis, polio-encephalitis, and outbreaks of influenza, bronchitis and pneumonia. As to the second, though some of us think it equally important, there is not perhaps quite the same general consensus of opinion; the point is that it is claimed not only that these diseases prevail in the same communities, either concurrently or in closely related sequence to one another, but also that two or more of them may in certain phases of epidemicity simultaneously affect members of the same household or other closely associated groups of persons.

All this is clear enough to students who have read between the lines of Sydenham, Willis and Creighton; but it is anathema to those who do not admit the right of appeal to the earlier epidemiological history. It is still, however, possible to invite the attention of these particular critics to work done since the epidemic diseases in question have been known by their present names. The facts are shortly as follows: The relation between poliomyelitis and influenza (their concurrent prevalence in communities and in members of the same families) was demonstrated by Brorström in Sweden, ten years ago. Similar relationships between bronchitis, pneumonia and influenza have of course been over and over again commented upon, particularly since the great pandemic prevalences in the early nineties.
The like relationships between cerebro-spinal fever and influenza have been worked out in London during the last four years. Thus, in 1915, of 462 cases of cerebro-spinal fever, thirty-three gave a history of influenza within fourteen days of the onset of the cerebro-spinal fever; sixty-one others gave a history of cold, cough, catarrh, &c., within a similar period; twenty-three more gave a history of contact (within fourteen days) with persons suffering from influenza, most of the original sufferers being inmates of the same house as the persons later attacked. The numbers, it has been shown, are far too great to be explained as resulting from mere chance occurrence; there must, therefore, be some close relationship between the diseases. Similar results were obtained for the individual years 1914, 1916 and 1917: though it should be noted that if epidemic and non-epidemic periods of cerebro-spinal fever prevalence during 1914-18 be compared, the comparative frequency (percentage of total cases) of concurrent development of the two diseases in question, in the same individual or in members of the same households, is distinctly higher in the epidemic than in the non-epidemic periods. Furthermore, close relationship between cases of cerebro-spinal fever and cases of influenza was found by Lieutenant-Colonel Dorgan to obtain in army camps. Finally, the relation between the Heine-Medin symptom-complex and influenza has been carefully examined by Dr. Crookshank in his recently delivered Chadwick lectures.

All this plainly points to a common cause operating in the epidemic diseases in question; but then comes a parting of the ways; for while some hold that this cause is the same living organism, others maintain that the nexus which binds cerebro-spinal fever, poliomyelitis, polio-encephalitis and encephalitis lethargica together with influenza, bronchitis, pneumonia and epidemic catarrh is merely predisposing (telluric, climatic, &c.), and not an actual causal influence exerted by one and the same infecting agency operating in all of them.

Sir Arthur Newsholme, moreover, adds to the group two other epidemic diseases, measles and whooping-cough; now it is very important that distinction should be made between the diseases first enumerated and the two last named; detailed evidence as to a very remarkable association of outbreaks in respect of time distribution and of the presence of the several disease types, in households and in groups of associated persons, has been adduced in the case of the former, but no similar intimate inter-relationship between them and measles and whooping-cough has been demonstrated.
An important argument in favour of a common infecting agency in the first named influenzal group of diseases is forthcoming on applying the principle known as the law of parsimony. In an influenza outbreak the cases of "influenza" are numerous, the cases of bronchitis and pneumonia less common, those of cerebro-spinal fever, poliomyelitis and encephalitis lethargica, comparatively speaking, rare. Let us take one of these for purposes of computation, say, cerebro-spinal fever. Sir Arthur Newsholme in an introductory memorandum, of last year, to "Further Reports on Cerebro-spinal Fever," dealt with the relation of case-rate to carrier-rate. He calculated for London during the year 1916, on a 2 per cent. "non-contact carrier-rate" basis, each carrier only circulating amongst ten persons during the course of a year (a basis which is admittedly too low), that the number of carriers of the meningococcus was about 800,000; hence on a 10 per cent. basis, which Sir Arthur accepts (though, he says, it is if anything an under-estimate), the entire population of London would, at one time or another during 1916, be acting as carriers of this organism. If this be agreed, it might then be inferred that similarly during the year the whole population (or something very little short of it) acted as carriers of the corresponding organisms concerned in spreading bronchitis, epidemic catarrh, pneumonia, influenza, poliomyelitis, and encephalitis lethargica. So that nearly every one harboured the several contagia of all these epidemic diseases during the year. It cannot fail to be appreciated, however, that the alternative hypothesis, that there is one common infecting agency, at once fits the facts and greatly simplifies the necessary assumptions. I venture therefore to plead for the application of William of Occam's razor—"Entia non sunt multiplicanda praeter necessitatem"—to the present case. The use of this instrument would not only reduce by some millions, in London alone, the number of carriers of the causal agent, many or all of whom are regarded, by whole-hearted supporters of the healthy carrier doctrine, as persons who should be segregated; but it would also enable a consistent workable theory of the case to case spread of influenza, now in pandemic waves and now in intercurrent trailing epidemics, to be formulated. It would, by focusing attention upon one single cause instead of upon a congeries of secondary invaders, give research workers and statisticians a chance of studying the laws of influenzal epidemicity, and—a summation devoutly to be wished—at the same time relieve that much harassed Sindbad, Preventive Medicine, of the need of carrying on its shoulders an old man of the sea in the shape of an impracticable working hypothesis.
Colonel A. B. Soltau, C.M.G., A.M.S.

Influenza of the "commercial" variety—i.e., a loose diagnosis to cover any catarrhal pyrexia—has always been a constant feature in sick returns. In an analysis of over 100,000 sick admissions to casualty clearing stations, it formed 3.7 per cent. of the total. A proved influenzal infection, confirmed bacteriologically, has also been present throughout, and was an important and serious factor in the broncho-pneumonia complicating trench nephritis each winter. But no true influenzal epidemic affected the armies until this year.

About the middle of April the first outbreaks occurred, being then confined to local foci of infection in the forward areas and at two of the bases. The first outbreaks to come under my own notice occurred in the ill-famed Ypres salient, an area where disease of all sorts always seemed to flourish. From these foci the epidemic spread to a general prevalence but it continued a very mild infection—three days' incubation, three days' fever, and three days' convalescence—and except from the numerical standpoint gave very little cause for anxiety. The first wave of infection died down about the end of May, but early in June a general reappearance was noted, and this in very rapidly mounting numbers. One army had over 36,000 cases in two and a half months, and from that figure some estimate of the total numbers can be made, when the five armies and the troops on lines of communications are considered. The epidemic reached its height about the third week in June, and the incidence thereafter gradually fell, though up to the present time cases are still occurring in considerable numbers. The spread of infection could easily be traced in the early stages, and men who messed or slept together were invariably affected in groups. Later on the distribution became practically universal.

As the epidemic increased in numbers, so did its virulence augment. In the first six weeks complications were hardly ever seen, and convalescence was rapid and complete. Later, an increasing number of respiratory complications were noted, and it was estimated in June that of the cases admitted to the special influenzal centres, some 2 per cent. developed serious pulmonary lesions, of whom a very considerable proportion died. Especially did this appear to be the case when the patient was suffering from any old-standing renal lesion. In such cases a rapid increase in the renal inadequacy and a profound toxanmia led almost invariably to a fatal termination.
The epidemic had important military bearings, as the following points will illustrate:

Whole units were sometimes put out of action. One army brigade of artillery had at one time two-thirds of its strength laid up, and was unable to go into action, though badly needed, for three weeks.

The German armies suffered heavily, and our Intelligence Branch had evidence that this was one of the factors which caused the postponement of a certain contemplated attack of very critical importance.

The prevalence of respiratory catarrh was, I think, one of the causes of the increased mortality in the gassed cases during the summer months.

Some evidence was collected indicating the importance of the "law of redistribution" enunciated by Major Zinsser, of the American Medical Corps, which must be considered in connexion with troop movements and drafting. A formation of troops may develop a certain immunity to its own organisms, but breaking up and re-distribution of such a formation may lead to epidemics, as such immunized men are brought into contact with other strains of organisms, or take to other formations strains which are new to the latter and therefore not guarded against.

General W. S. Thayer, M.C., U.S.A.

Our experience has been very similar to that of Colonel Soltau. In the latter part of April and May, almost simultaneously, in various regions occupied by the American Expeditionary Force, appeared this sudden epidemic, possibly first at Bordeaux, then at another point on the Marne, another in the Vosges, followed rapidly by its appearance in various camps, and then by a more or less general epidemic in the region of Bordeaux. At the outset it appeared to have several different foci—at least it was not recognized as starting from one individual—and spread rapidly in the camps, obviously by direct contagion. The symptoms at first were very mild, so that the condition rapidly earned the name of three-days fever. There were no complications, very few respiratory symptoms, and no mortality whatever in these early cases, with one exception: this was the epidemic which broke out in one of our divisions when the Germans made a phosgene attack, and in a group of men exposed to phosgene poisoning there were thirty or forty cases of pneumonia and rather a high death-rate. During May the epidemic, although it
appeared in various places, became much less extensive, though it did not disappear. The character of the cases, however, became more severe during the summer, the duration longer, and the complications much more frequent. This went on until the latter part of September, when a great number of troops were coming from America, where there was also an epidemic, and in many instances an outbreak occurred on the boats on the way over. With the preparations for an offensive there appeared a very sharp secondary epidemic with very serious respiratory complications. These were almost entirely pneumonia, and almost universally a pneumococcus pneumonia, quite a different story from the epidemic occurring in our camps last year in America, which was an epidemic due to a streptococcus in individuals convalescent from measles. Among other interesting facts from the epidemiological standpoint brought out in a meeting at the central laboratory at Dijon was the remarkable rise of the meningococcus infections. Meningococcus infection—meningitis—rose 400 per cent. with the September and October peak, and it is falling rapidly now.

Colonel Stock, S.A.M.S.

Owing to cable delays it has not been possible to obtain a complete account of the recent outbreak of influenza in South Africa. From a recent official cable, however, it appears that the outbreak began towards the end of August and beginning of September at Durban, and presumably the infection was brought from India or East Coast ports. Towards the end of September and early in October the disease appeared in Capetown, the infection having probably been brought from Europe and ports on the West Coast of Africa. In most of the towns and localities to which the disease spread, the epidemic lasted about three weeks and has now practically subsided except in certain remote localities. It is estimated that about half the population of the Union of South Africa was attacked, while over 7,000 Europeans and 40,000 natives died—mostly middle-aged persons. The severity and mortality varied considerably in different localities and it has been stated that monkeys and baboons in different parts of the country were severely affected but no official confirmation has been received as to the accuracy of this report.

There are several mutilated words in the cable message but it appears that a mixed vaccine prepared from organisms isolated during
the present epidemic was extensively used. The effect of the vaccine in preventing attacks is doubtful but the almost general opinion was that, if vaccinated, the attack was mitigated, complications prevented, and the mortality reduced. So far as is known, no person twice inoculated before an attack has died.

In view of the various hypotheses in regard to the recent outbreak in England it is noteworthy that the food supply in South Africa at the present time is practically normal and that the climatic conditions differ completely from those prevalent in England.

[The contribution of Surgeon-Lieutenant E. L. Sturdee, R.N., to the Discussion has been censored, and its reproduction in the Proceedings forbidden; but the MS. has been placed for reference in the Library of the Society.]

Surgeon-Captain P. W. Bassett-Smith, C.B., C.M.G., R.N.

The stress of the epidemic was first felt at Plymouth and caused a high incidence of the disease with high mortality. This was almost always associated with acute pleuro-pneumonia and empyema. The specific organism was almost constantly a streptococcus which was definitely haemolytic and very infective. The organism was isolated from the blood several times, and always from the pleural fluid. Cases of accidental inoculation occurred amongst the nurses attending the patients, and among the laboratory staff, from slight scratches in post-mortems, the organisms from the infected men being pure streptococci similar to those found in the patients and giving rise to a septicaemia. The boys of a training ship in the port used a sea-water bath, and this was looked upon as a possible source from which the infection could be spread. On bacteriological examination of the water I isolated a long streptococcus on each occasion when tested, an organism not commonly found in sea-water, but this was shown to be non-pathogenic to rabbits and probably not infective to boys. At other ports the streptococcic infection has also been common. Examination of the sputum of patients frequently showed organisms like Pfeiffer's bacilli and pneumococcus. In the Fleet some ships have been severely affected, the disease giving rise to 10 per cent. mortality of cases.
General W. S. Thayer, M.C., U.S.A.

At the beginning of the epidemic in May, Major Zinsser went to various points at which the infection had broken out with a view to making bacteriological investigations. In a certain number of instances he found the influenza bacillus in the nasal secretions, but these patients had very few respiratory manifestations, the majority of them no appreciable coryza or bronchitis. They had a sudden onset of fever, with rather well marked general symptoms lasting several days. Finally, in a group of cases at Dijon, seen very early, the influenza bacillus was isolated in a larger number than at first, and later on the respiratory symptoms were more common. In general it may be said that the influenza bacillus was found principally in the upper respiratory tract, diminishing on the whole down to the lung tissue. In certain cases, studied very early, an exceedingly small Gram-negative diplococcus was found, almost ultra-microscopic. It has not been thoroughly investigated, but it excited a good deal of interest in those early cases. The complicating organisms varied. In the earlier part of the epidemic, when pneumonia was so common, the organism as a rule was the pneumococcus, either in pure culture or in association with the streptococcus and influenza bacilli, but the evidence altogether is fairly definite that the great majority of pneumonia cases following this epidemic have been pneumococcal. There were isolated instances of small epidemics, largely streptococcal in character. In several places where the lung lesions were a streptococcal broncho-pneumonia, streptococcal empyema was relatively infrequent. In pneumonia, during the last few months, there has been very little empyema; a striking contrast to the conditions which prevailed in our epidemic in America following measles. In some instances, meningococci have been cultivated from the lung tissue, and in one instance pure cultures of what appeared to be meningococcus agglutinated Pasteur type C serum in the sputa as well. I merely mention this fact without attempting to interpret it. The general feeling has been that this is a secondary factor. These observations have been made at several points. The pneumococcus found in the camp hospitals was, for the most part, of types 4, 3, and 2; there were relatively few cases of type 1. In summary, one
may say that the pneumonia that has followed these cases of influenza—if we call it influenza—has been, for the most part, pneumococcus pneumonia, though isolated instances of streptococcus broncho-pneumonia have been seen: in other instances, streptococci have occurred in connexion with pneumococci—a mixed infection.

Sir Kenneth Goadby, K.B.E.

I have recently studied a number of acute cases of the prevailing epidemic of influenza in the wards the Royal Herbert Hospital, Woolwich. In all, about 1,000 cases were admitted, with a mortality of about 5 per cent. The majority were characterized by acute onset of malaise, with pain in limbs and frontal headache. Some of the cases had practically no lung symptoms but a large number developed an acute broncho-pneumonia commencing at the base of the lung and associated with blood-stained sputum of salmon-pink colour; very few showed the rusty sputum common in lobar pneumonia. Epistaxis was common, and a proportion of the cases showed a cyanosis of a curious type not directly attributable to consolidation of the lung.

I made a bacteriological examination of thirty cases at various periods after the onset, from the first to the seventh day. Seven cases had consolidation of the lung, twenty-seven epistaxis, twenty-seven broncho-pneumonia with red-stained sputum. For examination the sputum was collected directly into sterile bottles, taken to the laboratory and examined both by direct films and by culture. The films were stained by Gram and counter-stained with carbol fuchsin, and a second film was also examined by fuchsind, staining direct. Not a little difficulty is found in staining the short Gram-negative bacilli which were commonly found in the sputum, as the counter-staining by Gram, using the ordinary dilute counter-stain, frequently failed to bring the organisms into view. The sputum was plated out on blood agar, using either fresh normal blood or blood obtained under aseptic precautions and citrated. Sub-cultures were made from the plates on to blood agar. The citrated blood gave equally good results with the fresh human blood. The tubes were incubated for twenty-four hours before use.

The first cultures failed to grow luxuriantly, but when an alteration of the agar from 6 to 10 was made, no difficulty was experienced in obtaining cultures. In eighteen instances minute gum-drop colonies,
hardly visible without a lens, were present in considerable numbers, but in no case was the organism found in pure culture. The organism, which refused to grow on any other media but blood agar, was a Gram-negative, non-motile, extremely short diplobacillus which shows a tendency in individual cultures to considerable alterations in morphology. Streptobacilli containing seven or eight elements and even threads have been noticed as well as shorter, somewhat swollen forms of an oval clostridial type. The organism corresponds with the description and characters originally given by Pfeiffer. The organism was present in the films of the sputum in twenty-seven cases and was isolated in pure culture in eighteen. In addition to this bacillus, two other types of organism were noted as occurring with considerable frequency—the pneumococcus and the streptococcus. Gram-positive diplococci with capsules were found in twenty-two of the sputa examined microscopically and were isolated in twenty-one. The streptococcus seen in fourteen of the sputa examined microscopically was obtained in pure culture in nineteen. The streptococcus obtained was strongly haemolytic. A number of other organisms were observed, including various types of Gram-negative cocci of the Micrococcus catarrhalis group and in the last series of cases examined a few showed the presence of Gram-negative bacilli of the Friedländer type. The Gram-negative bacilli of the influenza class were present in greatest numbers in the sputa examined in the early stages of a case, especially when blood-staining first appeared. Three cases in which a relapse occurred, with a return of the blood-stained sputum after the original attack had quietened down and the temperature had been normal some five or six days, showed considerable numbers of the minute Gram-negative bacilli. Blood culture was attempted in six cases, two of which proved positive, the organism isolated being in both cases the streptococcus of strongly haemolytic type. No Gram-negative bacilli were found.

In the thirty cases I have examined, the bacteriology of the fatal and non-fatal cases appeared similar. The haemolytic streptococcus was recovered from the sputum of six of the ten fatal cases and was not present in the remaining four. A blood examination was made in eight of the cases (three fatal). In addition to the red and white cell count, an Arneth’s differential count was made. The outstanding feature of the blood counts is a leucopenia which has been already described as occurring in influenza by Levy and others. In no case examined was a leucocytosis observed. In the differential count there was a
relative polymorphonuclear leucocytosis in three cases, two of them fatal and one which recovered, the recovering case being an extremely grave one but one in which vaccine was used, which may possibly have complicated the issue. A differential blood count of Arneth's type shows a large increase in the horseshoe and rod-shaped nuclear cells (juvenile type) and a diminution in the numbers of tri-lobed nuclear. There is no appreciable increase in the quantity of large mononuclear or small mononuclear cells, except in one fatal case. The high relative proportion of rod nuclear and horseshoe nuclear cells appears of decided prognostic significance. The fact of the occurrence of plasma cells is also striking.

In the previous epidemic in June I examined a number of cases bacteriologically, but in no case succeeded in isolating the Gram-negative bacilli of Pfeiffer type, and in the two blood counts made, although there is a similar leucopenia, it is not so severe nor is the increase in rod nuclear cells so evident, but the general character of the count is the same. The cases were, however, much less severe, unattended by any bronchial symptoms, there was no sputum, and they recovered without the malaise commonly seen in the present type of case.

Whatever may be its pathological significance, there is no doubt that a short Gram-negative bacillus is associated with the early acute stage of the present epidemic in all the cases which I have examined.

Dr. Harold E. Whittingham.

(ABSTRACT.)

The following investigations were made to ascertain whether the present "influenzal" epidemic and the July "Spanish plague" were due to one and the same micro-organism or group of micro-organisms.

The difference in the signs and symptoms shown by cases in the two epidemics points to a difference in the causal organisms: pharyngitis and laryngitis, with nervous prostration, distinguished the July from the present epidemic, in which the prevailing complications are bronchitis and broncho-pneumonia with septicæmia.

The cases investigated in this communication are comparable with

1 This contribution is published in extenso, in collaboration with Dr. Carrie Sims, in the Lancet, 1918, ii, pp. 865-71.
those reported on in July—the patients in both instances being Royal Air Force officers and cadets.

**Blood-cultures.**—These were performed on fifty cases; positive results were obtained in seven (five streptococcus, one pneumococcus, and one resembling *Diplococcus mucosus* of Stephan). Of the fifty cases, death occurred in seven, five of which gave positive blood-cultures during life (streptococcus in four, *Diplococcus mucosus* in one). Amongst the patients giving a negative blood-culture two died. Withdrawal of 10 to 20 c.c.m. of blood in early stage of the disease produced a fall of temperature—a point of clinical importance.

**Throat Swabs and Sputum.**—Over 100 cases were examined. The chief micro-organisms present were streptococcus in 80, pneumococcus in 66, *Micrococcus catarrhalis* in 72, staphylococcus in 54, *Bacillus influenzae* in 46, *Bacillus septus* in 20, pneumo bacillus in 16, and meningococcus in 8 per cent. of the cases.

**Post-mortem Examinations.**—Out of fifty cases there were seven deaths—a case mortality of 14 per cent. The average age of patients treated was 25, of those who died, 23. The day of death varied from the fifth to the fourteenth of the disease. The post-mortem examinations showed lung lesions in all cases varying from congestion and oedema to pneumonia and pus formation. The micro-organisms isolated from lung, pleural and peritoneal lesions, blood and spleen, closely coincided with the mixed flora obtained before death. In one case only was *Bacillus influenzae* found in the lesions.

**Examination of Urine.**—The main features were the presence of albumin and renal casts in many, acetone and excess of urates in some cases—especially at the height of the fever in pneumonic cases. The acetone disappeared with the fever. Albumin was present in 100 per cent. cases on the second day of the disease. Casts were present in 50 per cent. cases on the third to fifth day. The albuminuria gradually diminishes, but if men are returned to duty too soon after mild influenza there is risk of nephritis developing.

**Leucocyte Count.**—(a) Influenza type: Leucocyte count little below normal at first; leucocytosis increasing from fifth to seventh day. (b) Pneumonic type: Distinct leucocytosis present from onset. (c) Lethargic form: Decrease in leucocytes from first, becoming more pronounced in fatal cases; some increase where recovery takes place. Generally speaking, leucopenia is of bad and leucocytosis of good prognostic import.

**Vaccine Treatment.**—Vaccine was prepared from micro-organisms
isolated from severe cases. \( (a) \) Prophylactic administration: Total number of persons in institution, 305; 156 were inoculated and 149 not inoculated. The case incidence in the inoculated was eight in 156; amongst the uninoculated eighteen in 149. The prophylactic vaccination should be done on two, preferably three occasions. It will probably abolish or modify the toxic type of the disease. \( (b) \) Curative administration: The dosage in all cases was 1 c.c.m., containing streptococcus, 5 millions; meningococcus, 2 millions; pneumococcus, 5 millions; Bacillus influenza, 2 millions; Micrococcus catarrhalis, 5 millions. The first injection was given as soon after admission as was possible, and repeated daily till the temperature fell to normal. In cases with higher temperatures than 103.5° F. polyvalent antistreptococcus serum was given subcutaneously (dose 20 c.c.m.) as soon as possible, followed by daily dose of 10 c.c.m. for three or four days.

Vaccine treatment exercised a distinctly beneficial effect on the symptoms, and cases cleared up rapidly under it. Serum therapy is efficacious if the injections are made before the sixth day, but not if made later.

The conclusion derived from a study of the foregoing results is that the malady under consideration is a “compound” not a simple disease.

Major T. R. Little, C.A.M.C., M.B.

The dominant feature in the post-mortem examination of seventy-three cases of pneumonia supervening on influenza was that of a lobular or bronchial pneumonia. The mucous membrane of the trachea and bronchi was swollen and congested, and covered with a sero-muco-sanguineous exudate. The lungs showed patchy areas of consolidation, varying in size from that of a chestnut to a hen’s egg. In most cases there was semi-consolidation of one or more lobes that might well be called a pseudo-lobar type of pneumonia. On section these areas projected somewhat above the general surface. They were from dark red to light red, and in places bordering on a grey colour. This was chiefly marked about the larger and smaller bronchi. On pressure of the cut surface a sero-muco-sanguineous exudate could be forced out of the lumina of the larger bronchi. Sixty per cent. showed a sero-fibrinous pleurisy. The heart was softer than normal, the right side being engorged. A few showed early pericarditis. The other viscera were somewhat congested, but exhibited no obvious
morbid changes. Fourteen per cent. showed active tuberculosis in the lung or bronchial lymph nodes. Thirty-one per cent. showed old pleuritic adhesions. Seventeen per cent. showed interstitial emphysema. Seven cases showed recent rupture of the rectus abdominis.

Microscopical preparations were made in a fair number of cases. Sections of lung were taken from consolidated areas. The quantity of exudate in the air vesicles contained little cellular débris. The vesicles were chiefly filled up with coarse fibrin, serum, exfoliated epithelial cells, a few leucocytes and red blood cells. The lining cells of the vesicles were vacuolated. The capillaries were congested, the walls of the smaller bronchi thickened and merged with the surrounding vesicles, which showed a much greater proliferation of the leucocytes. The lumina of the larger and smaller bronchi contained large numbers of exfoliated epithelial cells intermixed with leucocytes and red blood cells. Sections of the trachea and bronchi showed almost a complete loss of the epithelial surface, with a marked infiltration of leucocytes into the submucous coat. Sections of the bronchial lymph nodes showed a marked absence of the framework with the lymphoid cells widely separated and intermixed with polymorphonuclear and endothelial leucocytes. In sections of the kidney the tubular epithelium was stained poorly, and Bowman's capsules were widely separated from the glomeruli. Sections of the spleen, liver and other organs showed a fair amount of congestion, but were otherwise normal. Sections of the heart wall showed the muscle fibre somewhat separated, but were otherwise normal.

For the bacteriological examination I began with direct smears and cultures. The direct smears proved of little interest. Those examined from exudates of the trachea and bronchi were generally so overloaded with all kinds of bacteria as to make them almost impossible to differentiate. Those made from heart blood and splenic pulp in the great majority of cases showed no bacteria at all. I therefore confined my work to the examination of cultures. These were made from the cut surface of the lung, heart blood and small blocks of splenic pulp. The culture media used for the primary culture were neutral bouillon containing a little human blood and neutral agar containing 5 c.c. human blood per 100, on Petri dishes. Subcultures were made from blood bouillon to blood agar containing 5 c.c. of a suspension of dead staphylococci per 100 of media. I found that this medium stimulates the growth of Bacillus influenzae and many other organisms. Trypsinized blood agar was given a trial, but owing to
contamination and the amount of time involved in its preparation, it was discontinued.

The following organisms were found in the seventy-three cultures:

<table>
<thead>
<tr>
<th>Organism</th>
<th>Cut surface of lung</th>
<th>Small blocks spleenic pulp</th>
<th>Heart blood</th>
</tr>
</thead>
<tbody>
<tr>
<td>Growth</td>
<td>In all 62</td>
<td>In 11 9</td>
<td>In 23</td>
</tr>
<tr>
<td>Small Gram-positive diplococcus</td>
<td>62</td>
<td>9</td>
<td>23</td>
</tr>
<tr>
<td>Bacillus influenza</td>
<td>48</td>
<td>5</td>
<td>11</td>
</tr>
<tr>
<td>Streptococcus in short chains</td>
<td>41</td>
<td>6</td>
<td>15</td>
</tr>
<tr>
<td>Streptococcus albus</td>
<td>10</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Streptococcus aureus</td>
<td>9</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Bacillus coli</td>
<td>23</td>
<td>6</td>
<td>11</td>
</tr>
<tr>
<td>Pneumococcus</td>
<td>35</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>Gram negative diplococcus</td>
<td>22</td>
<td>6</td>
<td>27</td>
</tr>
</tbody>
</table>

The air-borne organisms are not included. The small Gram-positive diplococcus was found in pure culture from the lung in twenty-nine plates, heart blood eighteen plates, and spleen four plates. All other organisms were found mixed.

The three organisms found to be predominating—viz., small Gram-positive diplococcus, Bacillus influenza and streptococcus in short chains, were tested for their virulence on rabbits, guinea-pigs and mice. I began first with mixed cultures of the small Gram-positive diplococcus and Bacillus influenza, thinking that these two were the causative organisms and working together. Plate cultures containing these two were inoculated into the chest cavity of three half-grown rabbits, each receiving 5 minims of a suspension made from one streak of the colonies on a 3-in. Petri dish suspended in 2 c.c. of normal saline solution. All three died seventy-two hours later. Post mortem all showed a fair amount of cloudy fluid in the chest cavity, sero-fibrinous pleurisy and various sized foci of consolidation, dark red in colour, some just visible to the naked eye, others as large as the end of an ordinary lead pencil. One showed an extensive hæmorrhage into the abdominal cavity, the origin appearing to be the right lobe of the liver. Plates made from the pleural fluid showed a pure culture of small Gram-positive diplococcus. The same experiment was repeated on a second lot of three rabbits, one of which died in thirty hours. Culture showed Bacillus influenza as well as the small Gram-positive diplococcus. The other two died—one in forty hours, the other in seventy-two hours.

My next animal experiment was to test the virulence of each of these two organisms. They were cultivated in pure culture and suspensions were made similar to the above. Five-minim doses were
given to six rabbits, three receiving the small Gram-positive diplococcus, the other three *Bacillus influenzae*. Those receiving the small Gram-positive diplococcus all died within seventy-two hours. Post-mortem findings were much the same as in the animals receiving the mixed cultures. Those receiving *Bacillus influenzae* showed no apparent change. This convinced me that *Bacillus influenzae* played no part in the infection in animal experiment.

A third lot of four rabbits received 5 minims of a suspension of dead small Gram-positive diplococci, which were killed in the water bath at 58°C for one hour. Seventy-two hours later they received 5 minims of the living suspension of the same organism. All were living after twelve days' observation. Thinking at the time that the virulence had run out, as this particular culture had been subcultured daily for fourteen days, I repeated this experiment with six other rabbits, half receiving the living and the other half the killed organism. To my surprise, two receiving the living organism died within thirty-six hours, showing a well-marked pleurisy and areas of consolidation in the lungs. The third one lived for ninety hours, post mortem showing the same as the other two, except for one small portion of a lobe showing a grey coloured wedge-shaped consolidation much like a pyamid infarct. The animals receiving the dead organism were given 5 minims of the living suspension on the third day. All these animals are living.

Other experimental inoculations were tried on six guinea-pigs and six mice. All proved immune in 5-minim doses into the chest cavity, with the exception of one mouse which died in forty-eight hours. The post-mortem showed the chest cavity full of a dirty clay-coloured fluid, and recent pleural adhesions. Cultures made from the pleural fluid showed a small Gram-positive diplococcus and a motile bacillus resembling *Bacillus coli*.

Broth cultures of the small Gram-positive diplococcus were tried on other mice and guinea-pigs in 5-minim doses, which failed to kill. The streptococcus in small chains failed to kill rabbits, mice and guinea-pigs in 5-minim doses. One of the mice died on the eighth day; no morbid change was noticed.

Peritoneal fluid from rabbits dying from inoculation with the small Gram-positive diplococcus was inoculated into the chest cavity of rabbits immunized against this organism, with no apparent effect. Non-immunized rabbits receiving the same dose of the same fluid died in twenty hours. Post-mortem examination showed a fair increase in the peritoneal fluid and the pleura distinctly congested.
Filtrates from heart's blood, spleen, lung and sputum were also tested out. The filter used was a Berkefeld. Rabbits were given the filtrates in various doses. Six half-grown rabbits were used, all receiving from 1.5 to 4 c.c. into the belly cavity. Observation showed very little change, except for a slight roughening of the fur, and a slight rise in temperature during the first twelve hours. In twenty-four hours the temperature was normal, and all had their usual appetites. One animal, which died after seventy-two hours, showed post mortem an extensive venous congestion of the peritoneum, especially marked on the serous coat of the cecum, a slight excess of peritoneal fluid and a few tags of fibrin. Cultures made from the peritoneal fluid yielded a pure growth of the small Gram-positive diplococcus. This may be due to the fact that the filters used were new and leakage may have occurred, or too great a pressure may have been used in the filtering.

The agglutination reaction was not very marked. Five bloods were obtained from advanced and convalescent patients. These sera gave a very weak reaction with the small Gram-positive diplococcus, the highest being not above a 1 in 25 dilution, and the lowest a 1 in 10 dilution. The normal serum used as controls would only agglutinate as high as 1 in 10 dilution. Serum from immunized animals reacted in 1 in 20 dilutions. The control normal animal serum gave practically the same results. Three types of the known sera for pneumococcus used in the Rockefeller Institute, New York, were tested. None showed a reaction above a 1 in 10 dilution.

Characteristics of the Small Gram-positive Diplococcus: (a) Morphology. — Smaller than the pneumococcus on primary cultures. After sub-culture several times it becomes quite pleomorphic—some forms are rounded, some lancet-shaped, some oval, and in liquid medium short chains are formed. A fair number are larger than the pneumococcus.

(b) Staining. — It stains with all the laboratory aniline colours, but best with dilute aqueous of fuchsin. Gram-stained preparations showed a fair number taking up the counter-stain. Direct smears from exudate showed a halo or capsule.

(c) Biology. — The best medium for its growth is neutral blood agar or neutral blood broth. Dead staphylococcus added to the medium stimulates the growth. The colonies of the primary culture are very often small "pin-point." When growing with other organisms it is seen along the borders of the streak. Colonies of the cultures transplanted for twenty-five days were as large as a pin-head, widely
The cultures, if sown too heavily, appear as a semi-transparent film with a slight sagittal border. It grows poorly on ordinary agar, the colonies being just visible to the naked eye. In ordinary bouillon it shows a flocculent growth at the bottom of the tube. It only slightly haemolyses solid media. Grown in fermentation tubes containing any of the following sugars—galactose, dextrose, lēvulose, maltose, saccharose—and a little human blood, no gas is formed and it gives acid reaction. No acid in mannite, or dextrin or glycerine. There is slight coagulation in litmus milk. It exerts reciprocal and exhilarating action on the growth and life of Bacillus influenza. I have demonstrated this by subculturing these two organisms together as many as twelve times. My experience has been that Bacillus influenza dies out very quickly after the third or fourth subculture alone.

In conclusion I would say that the small Gram-positive diplococcus proved to be very virulent in animal experiments; it was the predominating organism and must be regarded as of aetiological significance especially in pneumonia supervening on influenza. On account of the large number of cases showing Bacillus influenza it is probable that the initial infection of the upper air passages is due to Bacillus influenza and the graver complications due to the small Gram-positive diplococcus superimposed. I look upon the small Gram-positive diplococcus as an intermediate type between the pneumococcus and streptococcus. It differs from both in animal experimental inoculations. It did not agglutinate the three types of pneumococcus serum in a higher dilution than 1 in 10; it was the only organism found in pure culture in cases studied by me. I am satisfied from animal experiments that a vaccine prepared from it proves of prophylactic value.

Captain Hallows, R.A.M.C.

In June, 1918, at Aldershot, we dealt with a large number of mild cases. The patients complained of sore throat and pain in the chest, and a few of these developed pleural effusions. In these cases I found what I termed a diplostreptococcus. The most characteristic feature of that organism to me was its pleomorphism. I subcultured it very carefully, and made slides of twelve different colonies; in every instance I found pleomorphism very evident. Next came the more serious type of this influenza epidemic, which started in September, and since then
one has been overwhelmed with material. I have been working on it practically alone, except for the help of Lieutenant-Colonel French, who enabled me to get post-mortem material for bacteriological examination. In the June epidemic, as the sore throat and tracheal pain were the only symptoms complained of, I took a certain number of West swabs of the posterior nares, and got the Bacillus influenzae in roughly 50 per cent. To turn to the more serious epidemic, I have again encountered the diplostreptococcus. I examined thirty-five or forty cases post mortem very carefully with Colonel French's help, that is to say, I took cultures from the spleen, blood, kidneys, the internal ear, sphenoidal sinuses, and from any other local lesions which presented themselves. In the majority of cases I obtained a positive culture from the heart blood, and these cultures were usually a diplostreptococcus, in a few cases the pneumococcus or Streptococcus pyogenes longus, and in fewer still both the pneumococcus and the diplostreptococcus. In the case of the spleen there were fewer cases in which one grew the diplostreptococcus or other organisms. In 15 per cent. of cases only have I got a growth from the spleen. From the kidneys I have not obtained a growth on culture on any occasion, nor in the urine, although in all cases examined I found albumin ranging from 0.4 to 0.02 per cent., and all these cases showed either casts or transitional epithelial cells. Again, with regard to this diplostreptococcus, I think this is the organism of which some speakers have been telling us to-night. Is it merely a pleomorphic form of a pneumococcus or a pleomorphic form of another streptococcus, such as the Streptococcus pyogenes? At first I was inclined to think that it was the former, but on the other hand, these organisms, in addition to other points of difference, were usually haemolytic, while the pneumococcus is generally regarded as not being so. Therefore I was rather driven to regard it as being streptococic, and the question was whether it was a pathogenic streptococcus or some milder form. I think it must be regarded as a pathogenic streptococcus. From lack of adequate facilities I was unable to carry out any animal experiments by which definite results would have been obtained. We are dealing here, probably, with one of the causative organisms of the complications in the disease—I would not say the primary cause of the disease, because I believe the Bacillus influenzae plays a very important part in that respect. These complications we regard as largely septicæmic in origin, and apparently this diplostreptococcus gives rise, with the pneumococcus and other streptococci, to the different complications,
such as confluent broncho-pneumonia, pulmonary oedema, and capillary bronchitis. The evidence for this diplostreptococcus being septicaemic is fairly well established, as it is grown from the heart's blood and from the spleen and lung and many other parts of the body. In some cases a very definite collection of pus was found in the sphenoidal sinus; the culture obtained therefrom yielded the Bacillus influenzae in some instances, and in others the diplostreptococcus, pneumococcus and Streptococcus longus. I think that the initial affection is due to the Bacillus influenzae, but the diplostreptococcus, the pneumococcus or streptococcus may give rise to a secondary infection, and it may be in the sphenoidal sinus or in the ethmoidal cells that these secondary organisms become septicaemic and give rise to other pathological conditions in the body. The lung condition seems to be largely secondary to the general septicaemia.

I have examined the blood in a good many cases with a view to the leucocyte count. I selected ten cases, some severe, some moribund, and some who were only slightly affected, and found that the total leucocytes ranged in all these cases from between 3,500 to 6,800. The lowest counts were from the most severe cases, but the highest count of the series was from a patient with marked purulent sputum. With regard to blood cultures taken during life I there again selected ten men, some moribund, and all severe cases. In only one case did I get any growth, and this was the pneumococcus. To return to this diplostreptococcus, I agree with what Major Little said about the sugar reactions. I put something like forty cultures of diplostreptococci isolated from different cases through sugar media; no fermentation of lactose or inulin was obtained, but dextrose is turned acid, mannite is not acted on, while in litmus milk a very definite clot is formed with very slight acidity, but very dense and firm, falling to the bottom of the tube.

To sum up then, it seems most reasonable to suppose that the Bacillus influenzae causes a primary infection, and in doing so prepares the way for a secondary infection by the pneumococcus, Streptococcus pyogenes longus or the diplostreptococcus: these organisms may then cause a septicaemia. The identity of the diplostreptococcus is as yet undetermined, but investigations are proceeding with a view to determining its relation with the pneumococcus and pathogenic streptococci.
Surgeon-Lieutenant G. Roche Lynch, R.N.

I wish to describe the bacteriological findings of a very serious epidemic of influenza which occurred at one of the great Naval depots in October, 1918. In my opinion this epidemic was primarily an infection of Bacillus influenzae, or Pfeiffer's bacillus, which was complicated by secondary infection chiefly of the streptococcus and of the pneumococcus, causing respiratory diseases, empyema, and in some cases pericarditis. Clinically this is borne out by the fact that many patients went sick with acute catarrhal symptoms which after a few days got well, the temperature dropping to normal, &c., or else after a short apyrexial period had a fresh rise in temperature with pulmonary symptoms. Of course in the majority of cases it was not possible to make this distinction.

At first, owing to suitable media not being at hand, I was unable to isolate Bacillus influenzae, but later on I was able to isolate it in several cases from sputa and in one case from the lung post mortem, further in numerous other sputa, bronchial exudates, and pus from lungs, I found an organism having the morphological characteristics of the influenza bacillus.

Post-mortem Findings.—Thirteen post-mortems were made. All showed pneumonia or broncho-pneumonia, two showed abscess of lung, eight empyemata, two pericarditis, and one jaundice, probably toxic in origin. The post-mortem appearance suggested that almost all would have developed empyemata had they lived.

Bacteriology of the Post-mortems.—I isolated streptococci eight times, Bacillus influenzae once, Micrococcus catarrhalis once, pneumococcus once. With the exception of two streptococci and Bacillus influenzae all the above were from the empyemata and in both the cases of pericarditis streptococci were also found.

Of thirty-eight blood cultures on patients with respiratory complications, seven showed streptococci, and one pneumococci. Of nine ante-mortem pleural effusions examined, eight contained streptococci and one was doubtful. The streptococci from the various sources appeared in the main to be similar, consisting of short chains capable of haemolysing blood rapidly, and in some cases the cocci showed indifferent tenacity to Gram's stain. Sugar reactions, as far as could be done, suggested the same type of streptococcus throughout.

During this epidemic, I have not found the meningococcus except in the cerebro-spinal fluid of certain cerebro-spinal meningitis cases.
At a base hospital in France we have had two groups of fatal cases. First, nine sporadic or endemic ones in the course of the year 1917. Broadly speaking, both clinically and pathologically, these cases were examples of acute purulent bronchitis, there being, generally, very few associated patches of broncho-pneumonia. Major L. J. Rhea and I isolated Pfeiffer’s bacillus in pure culture from the greyish beads of muco-pus in the terminal bronchi of all these cases. Higher up in the bronchial tree the exudate changed in character and became greenish yellow in colour, and though Pfeiffer’s bacillus was by far and away the predominating micro-organism, pneumococci, staphylococci, and Microoccus catarrhalis were also found. Smears of the sputum—nummular tenacious greenish pellets—showed also this association of a few other organisms with Bacillus influenzae. In two of these nine fatal cases acute endocarditis was found, and from the centre of the vegetations Bacillus influenzae was obtained in pure culture, and, once, this micro-organism was grown from the heart’s blood post mortem. In contrast with this group, there were eleven fatal epidemic cases of broncho-pneumonia in June and July, 1918, and during the past six weeks forty-seven fatal cases have been examined bacteriologically.

Bacteriological Results.—The most outstanding feature of the bacteriological work is the almost constant isolation of Bacillus influenzae whenever the necessary time and care can be given to the examination. In eight out of eleven cases examined in June and July, 1918, Bacillus influenzae was isolated. In the present epidemic, out of forty-seven cases examined bacteriologically, in thirty-six cases it has been isolated from the lungs, and examined in pure culture. In two other cases typical colonies and smears have indicated its presence, and in one more case we failed to get it from the lung, but isolated it from the heart’s blood. In the remaining eight cases the examination is incomplete at the moment of writing, but it is certain that in at least most of these the organism is present. So it may safely be said that this bacillus is universally present in the cases of pneumonia in the present epidemic. We select for preference one of the white drops or plugs of muco-pus which can be squeezed from the bronchioles. Smears from this material often show an enormous number of fine Gram-negative polar-staining bacilli, in some cases with no other
organisms, and the plates may present a profuse confluent growth of this organism with not more than a dozen other colonies on the whole plate. Where the muco-purulent material cannot be obtained, and where secondary infection is extensive, there may be great difficulty in isolating the organism, but with care and patience a few typical colonies can be found. We use for the original plating agar, to which fresh citrated blood has been added. The fresh blood has the advantage that streptococcus and pneumococcus colonies are easily distinguished by the changes they produce in the blood. For subculture we use Matthews' trypsinized blood agar, which gives a much better growth of Bacillus influenzae than the fresh blood. The colonies are like fine discrete transparent dew-drops, and produce no change in the surrounding medium.

Of the other organisms found, in forty cases where the examination is complete, thirty cases or 75 per cent. have yielded staphylococcus, nearly always aureus. Nineteen cases or 47 per cent. have yielded pneumococcus, and eight cases organisms as yet undetermined, either pneumococcus or streptococcus. Nine cases have yielded Streptococcus viridans not fermenting inulin, and three cases have given hæmolytic streptococcus. Staphylococcus seemed to be more abundant in the cases showing definite peribronchial abscess formation. The diagnosis of pneumococcus was made from the presence of capsules and the fermentation of inulin. In the numbers given, three or four organisms of the Streptococcus mucosus type are included, and in these cases the lungs showed a very copious mucoid exudate from the cut surface of the massive lobular pneumonia. Organisms of the pneumococcus-streptococcus group appeared to occur with definitely greater frequency in the cases showing diffuse pneumonia either of the lobular or oedematous congested type. Thus, of twenty-five cases showing definite lobular pneumonia, nineteen yielded either a Streptococcus viridans or pneumococcus. In nine outspoken cases of the diffuse congested type of short duration, pneumococcus or Streptococcus viridans was present in eight, and a hæmolytic streptococcus in two, once in predominant numbers.

Cultures from the heart's blood at autopsy were taken in thirty-two cases, two of which yielded Bacillus influenzae in pure culture, and in three others its presence was probable, although it was not isolated. Four cases yielded pneumococcus, four pneumococcus or streptococcus, not yet determined, one yielded a Streptococcus viridans, two yielded a hæmolytic streptococcus and six staphylococcus.
The Royal Society of Medicine

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As regards the rôle played by the various organisms found in the production of the different lesions, and of the relation of these organisms to the epidemic as a whole, we agree almost entirely with Keegan, who described his results in the Journal of the American Medical Association for September 28, 1918, in a paper entitled "The Pandemic of Influenza." Like him we found the Streptococcus hemolyticus relatively infrequently.

We believe that Pfeiffer's bacillus must be regarded as causing the original infection either alone or in association with other organisms, and as such must be considered as the real causative agent of the epidemic. Its constant occurrence in practically every case examined as compared with the varying nature of the accompanying flora seems to establish it in this position. We think, also, that its action is chiefly confined to the production of lesions of the trachea and bronchial systems, although we have also obtained it constantly, though in smaller numbers, from the diffuse lesions. We may mention one case, however, showing massive lobular pneumonia, in which a specimen taken at random from the solid mass yielded a luxuriant and practically pure growth of Bacillus influenzae. This suggests that this organism may also cause diffuse pneumonic lesions. The staphylococci seems to play a part in the production of the peribronchial abscesses. Pneumococci were found most abundantly in smears and cultures from the diffuse lesions, particularly the acute type, with extensive congestion and oedema. Hämolytic streptococci, which seem to have played such a leading part in the pneumonias following measles amongst American troops, have been found in only three of our cases.

While holding firmly that Pfeiffer's bacillus must be regarded as the original causative agent of the epidemic, we think that the pathological lesions with their bacteriological data, and also to a large extent what we have observed and heard of the spread of the disease in its simple type and in its pneumonic type, point to the fatal condition being essentially a contagious pneumonia due to a variety of pneumococci and streptococci affecting patients already infected with Bacillus influenzae.

Morbid Anatomy and Histology of the Lungs.

There have been at a base hospital in France two groups of fatal cases to compare and to contrast. First, nine sporadic or endemic cases which occurred in the course of 1917, and, secondly, some twelve to fifteen cases which died in June or July, 1918, and together with
these, fifty-five fatal cases autopsied in the last six weeks. In the first group studied clinically and pathologically by Major L. J. Rhea and myself, after death and during life, the picture was one of a universal acute purulent bronchitis with only small patches of broncho-pneumonia about the fine tubules. The lungs were otherwise air holding, and not until the organs were very carefully examined was the extent of the lesion obvious. The gross picture is extremely characteristic; on squeezing the sectioned lungs, fine greyish-white drops of muco-pus appeared everywhere at the mouths of the fine bronchioles, which up to that time had been invisible. Microscopically, besides the acute bronchiolitis, purulent exudate filling the lumen and destruction of the living epithelium, there were seen numerous very small peri-bronchial abscesses resulting from the destruction of the wall of the bronchiole. In two cases there was acute endocarditis due to *Bacillus influenzae*.

In the second group of cases, the majority of the necropsies have been done by Major W. H. Tytler and Captain R. M. Janes of the Canadian Army Medical Corps, and I have merely had the clinical care of most of these cases.

The picture in the second group of cases is much more complex, and many processes are responsible for the various lesions seen. However, *purulent bronchitis* has been outspoken in forty of the last fifty-five cases, and indefinite or absent in fifteen.

*Peribronchial abscesses* evident in the gross, were present in twenty-one cases, and nodular patches of interstitial pneumonia (as described by MacCallum and Cole) in thirty-three. The peribronchial abscesses may be seen as isolated white spots of 1 to 3 mm. in diameter, and more often in clusters. They are found scattered throughout air-holding tissue, or in relation to areas of broncho-pneumonia. Microscopically, the bronchiole and its surrounding tissue are converted into an abscess bordered by pneumonic alveoli. Three cases showed *larger abscesses*, which were thought to be due to a conglomeration of small ones, but histological evidence shows that at least some of these are really the result of infarction caused by inflammatory occlusion of vessels. These larger abscesses may measure 1 to 2 cm. in diameter, and their formation may show itself clinically in the sputum, when this changes from a nummular green tenacious character to a thick brick-red purulent kind. The intense vascular reaction recalls the appearances seen in lung infarcts in staphylococcus pyæmia, and we have found this organism in these abscesses occurring in our epidemic influenzal cases.
Microscopically, there is a very diffuse necrosis with little cellular exudate.

The broncho-pneumonia seems to be the same as that which was described by W. G. MacCallum among American troops after measles as "interstitial pneumonia." This is an apt term, for both in the gross and microscopically, it is evidently due to an extension of infection through the bronchial wall to the surrounding lung tissue, not an infection of the alveoli by way of the natural air passages. This lesion may occur as small areas scattered indiscriminately throughout the lung tissue, recalling the appearance of a tuberculous broncho-pneumonia, or more commonly, as well defined wedge-shaped masses with the base to the pleural surface, and showing extensive formation of fine abscess cavities.

Nodular patches of interstitial broncho-pneumonia were found thirty-three times. Microscopically the peribronchial connective tissue contains numerous dilated blood-vessels, and occasionally a definite haemorrhagic process. There is profuse lymphocytic and leucocytic infiltration, and the surrounding alveoli are filled with purulent and fibrinous exudate. Later on the alveoli may be found to contain a considerable proportion of mononuclear cells—desquamated alveolar epithelium.

Bronchiecstasy may occur before death in some cases of longer duration—one case died on the seventeenth day.

Diffuse Lesions.—Lobular pneumonia, present in thirty-one cases, may comprise a single lobule, a group of them, or even those forming a third to the whole of a lobe. The tissue is swollen, firm, and grey on section, with a definitely granular surface, the borders of the lobules remaining distinct. In some cases a combination of the bronchial and lobular process is seen. Abscesses and cavity formation are common in this lobular pneumonia, pointing to the presence of an interstitial infection as well. The lobular pneumonia microscopically shows much less fibrin than would be expected.

Diffuse Congested and Edematous Type.—Twelve of the fifty-five cases have shown this and little purulent bronchitis. There is congestion with oedema of the lung tissue, either localized or diffuse, and probably this type often represents a pneumonic process. There was a distinct group of cases in which death occurred after a comparatively short illness. The tissue sinks in water and is almost airless. A thick bloody fluid, not pus, exudes on pressure, and in the late stages we believe it represents a true pneumonic process. Microscopically, the
lungs show extensive oedema, haemorrhagic exudate, and, usually, scattered leucocytes in varying numbers, and also localized areas of greater leucocytic concentration.

*Collapse* of small areas on the anterior borders of lobes was very common in the 1917 cases. It followed the plugging of small bronchioles. Broncho-pneumonia was generally present, and sometimes a dilated bronchieole full of pus. In this epidemic it was present eleven times.

*Pleural Effusion.*—In eighteen cases this varied from 100 to 1,000 c.c. in amount and was non-purulent. In fifteen cases in which the effusion was double, it was purulent on one side, and varied from slight turbidity to definite empyema formation, with thick fibrino-purulent exudate on the pleural surface. Fibrinous non-purulent exudate was present as patches or diffuse, and occurred twenty-seven times.

Captain J. G. Hopkins, M.C., U.S.A.

**Bacteriology.**

(1) The picture of the outbreak among the United States troops in England was rather clear cut, for the reason that the earlier cases all came from one transport. These men were taken sick on board, or shortly after landing, and there can be little doubt on epidemiological grounds that the infection was the same in all cases at the outset. They were distributed among a number of our hospitals. The fatal cases which came to necropsy showed a varied picture anatomically, and various micro-organisms were recovered from the pneumatic lungs. This varied rather strikingly from hospital to hospital. For example, at Base Hospital No. 29, Major Williams recovered pneumococcus, type 4, from the lungs of every case which came to autopsy, while at Base Hospital No. 33, Lieutenant Simmons found haemolytic streptococci in over 50 per cent. of the cases examined. At other hospitals, *Staphylococcus aureus* and pneumococci of other groups were also met with in a number of cases and in two instances the meningococcus was recovered from the lung, once in pure culture and once mixed with an influenza-like bacillus. When we consider that all these cases fell ill at about the same time and were apparently infected one from the other, we are forced to the conclusion that these organisms, while they may have been responsible for the fatal terminations, were
secondary invaders. The prevalence of a certain type of this secondary infection at certain hospitals suggests that the secondary infection probably occurred after admission to hospital. The results of several of these groups of autopsy examinations are summarized in Table I and Table II.

(2) It will be noted that in the earliest series of cultures which were made at the Base Laboratory an organism which we have called the influenza bacillus was found in the majority of cases, and in several instances was recovered from the heart’s blood or spleen. In the latter part of the epidemic this organism was recovered somewhat less frequently. However, a suggestive observation was made by Lieutenant Simmons to the effect that if cultures were taken from the most recently involved portions of the lung they showed Pfeiffer’s bacillus in pure culture, whereas in the older foci pyogenic cocci were found, either pure or mixed with a few colonies of Bacillus influenza.

(3) These data left us in some doubt as to whether the influenza bacillus was also a secondary invader, or was the cause of the original infection.

(4) In order to obtain further light on the nature of the original infection, a series of thirty-two cases from this same transport, only two of which showed signs of pneumonia, were studied during life by Lieutenant Stauffer. Sputum obtained at the bedside was washed and immediately injected into a mouse and also plated on blood agar. The results are seen in Table III, which shows that the influenza bacillus was obtained from the majority but not from all of the cases. The influenza bacillus was frequently obtained from the heart’s blood of the mouse after death, and the pneumococci, although frequently found in the blood or peritoneal cavity of the mouse, never grew in profuse and almost pure culture in the peritoneum, as is usually the case when mice are injected with the sputum from typical lobar pneumonia.

(5) These observations were not made until the epidemic in troops from this transport had begun to subside, and it was quite possible that the original infecting agent might be missed. At this time, however, a number of secondary cases developed among the hospital personnel and among troops in contact with the men of this transport at the Rest Camp. Forty-eight sputum cultures from such cases made by Lieutenant F. B. Harrington, showed the presence of the influenza bacillus in thirty-five, and in twenty-three of the cases it was found practically in pure culture, being mixed with only a few colonies of the organisms ordinarily found in the upper respiratory tract (Table IV).
### Table I.—Cultures from Lungs Post Mortem.

<table>
<thead>
<tr>
<th></th>
<th>Base Laboratory (first series)</th>
<th>Base Laboratory (second series)</th>
<th>Base Hospital No. 35</th>
<th>Base Hospital No. 29</th>
<th>Base Hospital No. 37</th>
<th>A.R.C.M. Hospital No. 1</th>
<th>Total</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Alone</td>
<td>Mixed</td>
<td>Total</td>
<td>Alone</td>
<td>Mixed</td>
<td>Total</td>
<td>Alone</td>
<td>Mixed</td>
</tr>
<tr>
<td><em>Bacillus influenza</em></td>
<td>13</td>
<td>24</td>
<td>37</td>
<td>0</td>
<td>8</td>
<td>8</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><em>Pneumococcus</em></td>
<td>3</td>
<td>12</td>
<td>15</td>
<td>0</td>
<td>7</td>
<td>7</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td><em>Streptococcus hemolyticus</em></td>
<td>1</td>
<td>5</td>
<td>6</td>
<td>3</td>
<td>4</td>
<td>7</td>
<td>25</td>
<td>8</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>4</td>
<td>6</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td><em>Streptococcus viridans</em></td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>4</td>
<td>4</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td><em>Meningococcus</em></td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

### Table II.—Cultures from other Viscera.

<table>
<thead>
<tr>
<th></th>
<th>Heart’s Blood</th>
<th>Spleen</th>
<th>Liver</th>
<th>Pleural Effusion</th>
<th>Pericardial Effusion</th>
<th>Sphenoidal Sinus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Base Laboratory (first series)</td>
<td>Base Laboratory (second series)</td>
<td>Base Hospital No. 35</td>
<td>Base Hospital No. 35</td>
<td>Base Hospital No. 35</td>
<td>Base Hospital No. 35</td>
</tr>
<tr>
<td>Number of cases examined ...</td>
<td>17</td>
<td>58</td>
<td>5</td>
<td>17</td>
<td>17</td>
<td>1</td>
</tr>
<tr>
<td>Number from which growth was obtained ...</td>
<td>5</td>
<td>13</td>
<td>26</td>
<td>7</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td><em>Bacillus influenza</em></td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td><em>Pneumococcus</em></td>
<td>2</td>
<td>6</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td><em>Streptococcus hemolyticus</em></td>
<td>0</td>
<td>6</td>
<td>23</td>
<td>0</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>
Table III.—Cultures from Sputum and Nasopharynx. Thirty-two Cases of Influenza without Pneumonia from S.S. "—.

<table>
<thead>
<tr>
<th>Organism found (number of cases)</th>
<th>Number of cases examined</th>
<th>Sputum (direct culture)</th>
<th>Sputum injected into mouse Peritoneum</th>
<th>Nasopharyngeal swab culture Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Bacillus influenza</em></td>
<td>29</td>
<td>22</td>
<td>22</td>
<td>24</td>
</tr>
<tr>
<td>Pneumococcus</td>
<td>9</td>
<td>10</td>
<td>10</td>
<td>6</td>
</tr>
<tr>
<td><em>Streptococcus hemolyticus</em></td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>7</td>
<td>7</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><em>Streptococcus viridans</em></td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Gram-negative diplococci</td>
<td>13</td>
<td>6</td>
<td>4</td>
<td>15</td>
</tr>
</tbody>
</table>

Table IV.—Forty-eight Sputum Cultures from Camp Hospital 35, October 10—October 31.

<table>
<thead>
<tr>
<th>Organism found</th>
<th>Number of cases</th>
<th>Sputum injected into mouse Peritoneum</th>
<th>Nasopharyngeal swab culture Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Bacillus influenza</em> alone</td>
<td>23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pneumococcus alone</td>
<td>7</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Bacillus influenza</em> and pneumococcus mixed</td>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Streptococcus hemolyticus</em></td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Bacillus influenza</em>, pneumococcus, and streptococcus mixed</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Streptococcus hemolyticus</em> and pneumococcus</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Streptococcus</em> and <em>Bacillus influenza</em></td>
<td>2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(6) To summarize the results of our bacteriological observations, it seemed evident that these patients died with a number of terminal infections due either to the pneumococci, which were of various groups, to the haemolytic streptococcus, to *Staphylococcus aureus*, or to the meningococcus, and that in the early part of the epidemic they died not infrequently from infection with the influenza bacillus alone. The organism most frequently met with, and met with the more frequently the earlier the cases were observed, was the influenza bacillus, but we cannot overlook the observations of Charles Nicolle at Tunis, as to the presence of a filterable virus in this disease. We have been unable so far to do any work on this side of the question. From our observations we can conclude that the primary infection was due, either to the influenza bacillus, or to a filterable virus.

Pathology.

(7) The post-mortem data have varied very much from case to case as might be expected from the different bacteria which we found in the terminal lung infections. In the earlier cases, the lungs showed
a fairly uniform consolidation, approximating typical lobar pneumonia, and differing from it chiefly in the hæmorrhagic appearance of the lung and the moistness of the consolidated areas. In the later cases the consolidation was very patchy and toward the end of the epidemic cases were encountered in which there was an intense bronchitis and little macroscopic pneumonia. Pleural exudates were common, pericardial effusions not uncommon, and the character of the exudate in these cases varied according to the infecting organisms. Abscesses in the lung were also rather frequent, and from these either staphylococci or pneumococci were recovered.

(8) The lesions varied so much that it is difficult to describe the pathological picture, but the most frequent change was a massive consolidation with a mottled appearance on section. The cut surfaces of these lungs were studded with lines or dots of opaque, yellowish grey consolidation, from the centres of which drops of pus could usually be expressed. These seemed to represent older areas of peribronchial consolidation, and between these areas the tissue was intensely red, moist and granular, and contained practically no air, apparently the result of rapid extension of the pneumonic process shortly before death. The older areas of consolidation were frequently pyramidal in form with the bases against the pleural surface of the lung, suggesting infarction. Occlusion of the blood-vessels could not be made out, however, and these areas probably represented a lobular pneumonia originating about the terminal bronchi.

(9) The trachea and larger bronchi in these cases showed an intense hæmorrhagic inflammation with relatively little exudate. Dilatation of the heart was frequently noted and many of the livers showed the nutmeg appearance of chronic passive congestion.

(10) In a number of instances there was well marked œdema of the meninges and injection of the cerebral cortex; and in few instances a true meningitis due to the streptococcus, pneumococcus, influenza bacillus, or to a mixture of two of these organisms.

(11) In the cases in which the nasal sinuses were examined they were found acutely inflamed and frequently completely filled with pus. Cultures from these sinuses usually showed the same micro-organisms as were recovered from the lung of the same case, a finding which suggested that these sinuses might be the portal of entry of the infection.
Dr. B. H. Spilsbury.

This contribution is based upon the post-mortem examination of twenty fatal cases of the disease occurring during the present epidemic, together with the completed histological examination of eighteen of them. Most of the cases, on account either of a rapidly fatal termination or of unusual symptoms or complications, have been the subjects of a coroner's inquiry.

Of the twenty cases, fifteen occurred in persons between 10 and 37 years of age; the remainder were infants. Two-thirds of the subjects were females. In the respiratory system, the most constant morbid condition was acute inflammation of the air passages, extending in three-quarters of the cases from the trachea to the small air passages: a few cases showed inflammation of the small air passages alone, and two cases showed no inflammation of the air passages at the time of death. The inflammation was the vivid "flaming" variety with abundant purulent exudate in the upper air passages and in several cases with the formation of a false membrane without ulceration; the membrane was undergoing calcification in one case. In the smaller air passages the exudate was abundant, varying from a purulent fluid to a thick muco-pus; some of the passages were dilated. Inflammation of the larynx with slight oedema was present in three cases. I found an organism having the morphological characters of Pfeiffer's bacillus in the air passages in twelve of the twenty cases; it being more readily obtained from the exudate in the smaller air passages than from the upper respiratory tract. Many other organisms were also present in the air passages, the most constant being pneumococci and streptococci, together with diphtheroid bacilli, and organisms resembling the meningococcus.

In the lungs the morbid condition showed greater variations. In two cases in infants there was general emphysema only; in two other cases, both rapidly fatal, intense pulmonary oedema was present. The remainder had acute broncho-pneumonia of which two varieties were found. The first was a typical acute suppurative broncho-pneumonia with a tendency towards abscess formation, though a large abscess was found only in one case. In this form yellow or grey areas were scattered through the affected portion of the lungs and were characterized microscopically by an intense polymorphonuclear cell accumulation in the alveoli. In this variety a streptococcus was the organism constantly
present, in some cases in very large numbers. Six cases exhibited this form of pneumonia. In the other, which I have called the hæmorrhagic variety, the pneumonic areas were firm and of a deep red colour; when small they bore some resemblance to pulmonary infarcts, but this form tends towards a more massive consolidation resembling in that respect a lobar pneumonia, although differing in the slight colour variations in different parts, which gave a mottled appearance to the cut surface of the solid lung. This form microscopically was characterized by hæmorrhage into the alveoli with little or no fibrin formation. In this form the organism constantly present was a pneumococcus, though not in such large numbers as were the streptococci in the previous variety. Nine cases showed this variety of pneumonia. In both varieties acute bronchiolitis was present in the affected areas, the exudate varying according to the characters of the exudate in the lungs.

With the possible exception of one case, I have not found Pfeiffer's bacillus in the pulmonary alveoli and I regard the pneumonias as caused by one of the two organisms described above. One case showed a mixed infection by these two organisms with gross and microscopical changes in the lung intermediate in character between the two varieties. Other organisms were found in the alveoli of the inflamed lungs, but apart from putrefactive organisms they were neither numerous nor constant in character. There was early pleurisy over the pneumonic areas in four cases and an empyema in one case. The glands in the mediastinum and lower part of the neck were enlarged and congested; in one case, the single instance of a large pulmonary abscess, a mediastinal gland had developed an abscess which extended to the surrounding tissue and burst into the trachea.

In the circulatory system the heart alone showed constant changes. In all cases there was cardiac dilatation; fatty degeneration was present in eighteen cases and was advanced in several instances. Acute pericarditis was present in two cases with pleurisy. Serous hæmorrhages were present in the pericardium and pleura in several cases. In the liver and kidneys cloudy swelling was found constantly; in several cases the liver also showed fatty degeneration, advanced in one case. In the spleen slight enlargement and congestion were frequently present, but no striking histological changes, and there were no acute septic spleens. In the genital organs miscarriage had occurred in one case, although the duration of the illness was less than two days, and in a second case miscarriage was developing. There were no changes in the alimentary canal, though in several cases the disease was of the abdominal type.
In the central nervous system there were degenerative changes in the nerve cells in several cases, especially in the cells of the motor nuclei in the pons, in some of which chromatolysis and pigmentation were found, but post-mortem changes greatly increased the difficulty of the microscopical examination. There was general congestion of the brain in several instances, but there was no inflammation in the brain or meninges. In one case I found what appeared to be a haemorrhage in the pons, but proved to be a cavernous angioma.

In conclusion, I regard the condition as a primary infection of the air passages by Pfeiffer's bacillus, the failure to find this organism in 40 per cent. of the cases being either because the search was not sufficiently thorough, or because the organism had disappeared before death. Following this infection an invasion of the air passages and lungs occurs by a pneumococcus or streptococcus, one of these organisms being responsible for the pneumonia. The changes found elsewhere in the body are due to acute toxæmia, and in no case have I found either a pyæmic or a septicæmic condition.

Captain Graham, M.C., U.S.A.

Out of 723 cases clinically diagnosed as influenza, 239 developed pneumonia, diagnosed as broncho-pneumonia or lobar pneumonia. These pneumonias were all of them characterized by an intense cyanosis, quite frequently by jaundice, high fever, and extreme prostration. The pathological changes in the whole series of cases showed an intense congestion of the lung and the respiratory tract, the pharynx and larynx, down to the very smallest bronchioles. The lungs on section were extremely moist, the congestion being mostly in the posterior portions. On section the bronchial lymph glands were large, moist, juicy, and pus or purulent fluid could be expressed. Out of the total number of necropsies (101) pleural effusion of one sort or another occurred in about thirty cases, or one-third of the total number, and the fluid was definitely purulent. In none of the cases did we find a definite thick creamy pus. The pus in nearly every case was a very thin, dark-brown fluid. In the 101 autopsies the heart was not dilated itself in any case. As to the bacteriological results, in thirty-three cases we obtained haemolytic streptococci, in sixteen cases the influenza bacilli, in four cases staphylococci, and in seven cases pneumococci of type 3. The intense congestion of the whole respiratory tract
approached in the lungs themselves almost the stage of haemorrhage, and the very frequent presence of the haemolytic streptococcus gives rise to our conception of the situation that the streptococcus is most commonly concerned.

[The Discussion was adjourned to the day following.]
DISCUSSION ON INFLUENZA.¹

Sir BERTRAND DAWSON, G.C.V.O., M.D.

CLINICAL ASPECTS.

I have certainly noticed one point, and I know that others have—namely, that the disease, although it generally begins acutely, need not do so; it may begin subacutely, but insidiously, and it need not begin with the involvement of the nose, it may start in the throat. Beginning with a sore throat it may perhaps in two or three days flare up into a bronchitis. But the graver forms are what interest us most. The grave form may follow close on the heels of the onset, that is, within forty-eight hours of the influenzal symptoms you may have the graver symptoms of pneumonia, or, on the other hand, this may develop as late as the sixth day. The gravity of the cases seems to me to be due to the relative dominance of two factors—the septicemic factor and the degree and nature of the involvement of the respiratory tract. Most of the severe cases seem to have both factors, the septicemic factor and the pulmonary factor, but occasionally the septicemic factor seems to dominate the picture. Perhaps it would not be amiss to express what I mean by a concrete example. The patient is lying in bed on the fourth day of the illness, flat, not propped up by pillows, mildly delirious, not perhaps fully aware of his surroundings, and certainly not aware of the nature of his illness, breathing rapidly—anything up to 40—and presenting a general cyanosis, a curious tint diffused all over the body, almost heliotrope in colour. Very often the vessels are injected. His

¹ At a meeting of the Society, held November 11, 1918.
condition is certainly not due to any respiratory involvement, for the
patient has no true dyspnoea; he is not conscious of any effort to get
breath, he does not require to be propped up, and yet his breathing is 40,
and his nostrils are dilated. Equally it is not due to the heart, for there
is no evidence, beyond the rapid pulse of fever, of any sort of cardiac
dilatation. The only conclusion one can draw is that it is a toxic
condition. The heart, as I have said just now, is rapid (120), but there
is nothing more to remark about it; rather a low blood-pressure if
anything. As to the physical signs of the illness, in this particular case
I have in mind there is practically no cough and no sputum; there is
the usual tongue, dry in the centre, brown, still moist at the edges—all
this on about the fourth day. Then there are two other features: there
are purpuric patches about the face, and in this particular instance one
on the back and two on the chest. In other instances in these
septicaemic cases there have been purpuric patches on the limbs,
sometimes purpuric blebs. These purpuric conditions are part and
parcel of the septicaemia. In this case and others in which the
septicaemic factor is in evidence there is a subcutaneous emphysema,
with pain in some instances over the chest, and in this particular
instance on the right side of the neck above the clavicle. I fail to see
the explanation. If it were a case in which the patient was coughing
a great deal, the condition might be due to an abscess or softening of
the lung near the surface, because this bronchiectasis extending right
up to the surface, it is conceivable that one of these patches of suppura-
tion may become adherent to the chest wall, and during an effort of
coughing the air may be pumped through. It sounds a little far-fetched
in so acute a condition, but it will not do at all in cases in which there
is no cough whatever. In this instance the lump to the right side of
the neck was very definite, and the patient was hardly coughing at all.
The other hypothesis is that it may be due to a gas-producing organism,
not a virulent gas-producing organism such as one is accustomed to
meet in the wounds of war, but some milder diplococcus or strepto-
coccus. It recalled to my mind the investigation of one or two cases
of gastric influenza some years ago, when I was very much interested in
infections of the stomach.

In what I have said up to now I have tried to give an example in
which the septicaemic factor is the dominant one. In most cases,
however, the clinical picture is due both to the septicaemic factor and
to the pulmonary involvement. There again the clinical picture varies
according to the nature of the pulmonary involvement. The cases we
have seen in the course of this epidemic really bear a resemblance to
the cases of purulent bronchitis which we had earlier in the war. I
should take the pulmonary varieties as follows: In the first place, the
acute broncho-pneumonic cases, sometimes with haemorrhage, sometimes
suppurative. There you get a dullness if it is sufficiently confluent,
tubular breathing and increased vocal resonance. In another type of
case where the bronchitis dominates, and where fairly early in the case
the bronchi after being intensely red and injected become blocked with a
purulent glutinous exudate, spreading perhaps into the bronchioles, the
physical signs are quite different. Then in my experience you will
often come to a case in which the patient looks obviously ill and suffering
from respiratory distress, yet examination of the chest reveals surpris-
ingly little amiss. There are scattered noisy rhonchi, but there are
large areas of silence over that chest. On percussion there is a high-
pitched resonant note, and it might appear that there is very little wrong,
yet careful examination shows that there is actual sucking of the
intercostal spaces, and this is probably because the bronchi are blocked
more or less with a purulent glutinous exudate. Now these physical
signs, although appearing quite different, are really fundamentally the
same; it is simply a change in the distribution of the lesion. In the
diffused broncho-pneumonia the distribution is amongst large patches
of alveoli and bronchi which run one into the other, whereas in the
second instance there are large areas cut off because the distribution is
mainly in the bronchi. The third type is the ordinary classical lobar
pneumonia, and then one goes on to what used to be called the influen-
zal type of pneumonia, which is simply a spongy condition of the lung—
a diffused congestion with impaired air entry. It has not the same
interest in this discussion as the two I have already mentioned. The
point I would like to make is that all these varieties are really funda-
mentally the same type of disease, and that they depend first on the
relative dominance of the septicaemic factor and the pulmonary factor,
and, secondly, on the particular nature of the lesion in the lung.

General W. S. Thayer, M.C., U.S.A.

A feature of these cases is the suddenness of the onset. Some men
can tell you almost the exact place and minute when they began to
feel ill—men practically drop out of the ranks while marching. The
onset is accompanied by severe headache, sometimes chilly sensations,
pain in the neck, general pain in the limbs, an aching pain throughout the body, and rapid rise of fever, usually to 103° F. or more. The respiration and pulse as a rule are not much accelerated. The pulse is often rather slow as compared with the fever, and the appearance of the patient is noteworthy, the general flushing of the face, the injection of the conjunctiva, and the rather heavy and dull expression, so that the patients with influenza can be picked out from the end of the ward by their flushed expression and their rather dull and heavy eyes. There was very little cough; nose-bleeding at the outset was very common, but the respiratory symptoms were often unimportant, though sometimes later on there would be a dry pharyngitis, and sometimes a little coryza. After two to four days the temperature usually fell by crisis to normal, and in these early cases there was no mortality, and there were few or no complications. That was a very familiar picture, at once recalling the epidemic of 1889, when I had a personal experience of its suddenness of onset. The illness was not like the very severe colds with coryza followed by bronchitis, so common among our patients every winter, and I have not often recognized that picture commonly since the epidemic of 1889-90. The epidemic subsided, but cases continued, and gradually grew rather more severe. In the first place, chills were more common at the onset. The nose bleeding was very frequent. Herpes was not rare. Coryza was much more common, and a dry pharyngitis was commoner. Cough, soreness of the chest, and general bronchitis were present in a large proportion of cases. Expectoration was scanty at first, and later it was blood-stained. Mild diarrhoea was not uncommon. The fever ran from seven to nine days, instead of from two to four days, and not infrequently there was pseudo-crisis; after several days of fever the temperature would drop, and the patient feel quite well, and then there would be a return lasting several days, with great prostration and considerable exhaustion. Most of these cases, if immediately sent to hospital, recovered quickly; if put to bed immediately without having to be evacuated there were few complications, though a good many had a persistent bronchitis. In those patients who had to be evacuated—that is to say, who were carried from the Front, or, being in small camps, were taken some distance to hospital—the proportion of complications was very much greater. All of us are agreed that the proportion of complications among the men who have to be carried even short distances is strikingly greater.

Major Moss brought some interesting figures at a meeting at Dijon
relating to the district round Bordeaux. In three base hospitals and two camp hospitals where the patients for the most part had had to be evacuated there were treated 3,227 cases of influenza. The proportion of pneumonia in these five hospitals varied from 15.58 per cent. to 40 per cent. In the same area there were two camp hospitals containing men who could be hospitalized immediately without being carried any distance, and here there were treated 7,877 cases with a proportion of pneumonia of 6.8 per cent. in one, and 7 per cent. in the other. I think there is a fallacy in these figures, because it is quite possible that some of those patients who were evacuated to the other hospitals were encamped there because they were ill already. Nevertheless, it is a very striking difference; the complications were much more numerous in the cases of patients who had to be moved. The complications were usually, apart from the simple bronchitis, pneumonia—in fact, they were almost always pneumonia—and came on usually in the common way. The fever did not fall; commonly there was no very acute onset of the pneumonia—there was no moment at which the observers noticed that the fever did not fall. Perhaps there were chilly sensations; the expectorations became blood-stained, sometimes extremely so, and then rusty. There were as a rule very few physical signs in the early stages. The respiration was somewhat accelerated, the pulse not much accelerated. As a rule cyanosis developed early. The patient became dull and apathetic. The physical signs in the chest usually consisted of here and there fairly good resonance throughout, perhaps with slight areas of dullness, and some modified areas of respiration; sometimes little areas of definite tubular breathing could be made out. Very often suddenly, a few hours or a day before death, the evidences of pneumonic consolidation rapidly spread. The first few signs, then, are indefinite evidences of what might be called wandering solidification, and then, very shortly before death, a widespread and considerable area of solidification. Patients with these symptoms, whose sputum as a rule was not very purulent, showed the picture so common in Berlin thirty years ago, which was well summed up by Beyer and Prudden, namely, that the striking feature was the frequent existence of an older focus of septic pneumonia, with evidences of sudden, explosive action, and the relative infrequency of suppuration, empyemas being found in two only out of seventy-five necropsies. There were, of course, other instances of similar epidemics in which the clinical picture was very much the same, except that the expectoration was more purulent, and the other
symptoms were perhaps even graver than in many of these cases, leading on to a streptococcus broncho-pneumonia with multiple small abscesses. The mortality in the cases of streptococcus broncho-pneumonia was very high, and the mortality in the other instances, which showed as a rule the pneumococcus of an indefinite type, was also sometimes very high, between 80 per cent. and 20 per cent. There were some instances, especially in patients at the Front, who were exposed rather severely in the region of Verdun, in which death occurred one or two days after the onset, most of these cases on necropsy showing a small central patch of solidification, the men having been ill a longer time, though the gravest symptoms had been noticed only a short while before.

In conclusion, this epidemic is analogous with that of 1889-90. I should like to ask what has been going on in the interval between these epidemics. Has the same malady occurred sporadically every year, or has there really been immunity? In connexion with the pneumonias following influenza there is one thing I have never seen before—namely, the occurrence of subcutaneous emphysema beginning in the neck and spreading sometimes over the whole body. A certain small percentage occurred in almost every considerable group of cases in the American Expeditionary Force. Yesterday I saw two cases at our hospital No. 29 at Tottenham. There is no evidence that these patients coughed especially hard, so that the emphysema is not due to efforts at coughing, such as occurred in the gas cases; it must be due to an abscess of the alveolar walls and of lung tissue which has allowed the escape of air into the interstitial tissue, but why it is so extraordinarily prevalent in this particular epidemic I do not know. I have been unable to find any reference to it as a common complication of pneumonia. As a common complication of pneumonia it is certainly very unusual.

Colonel Longcope, M.C., U.S.A.

Probably owing to the fact that I was entirely unfamiliar with the disease, it struck me as a most extraordinary and most interesting type. As it has occurred among the troops that I have seen in the American forces in France there were certain outstanding features that immediately attracted my attention, and there are a few points that I should like to emphasize. These are the flushed face, the swollen eyes, the tracheitis, the laryngitis, without any evidence of what the patient
called sore throat, great pain in the back, and very extraordinary pains which the patients complain of in the joints and bones and occasionally in the muscles, not only of the arms and legs, but even of the abdomen, so that quite frequently the patients have been brought to the hospital as cases of appendicitis. The muscle pains are particularly interesting. In the early cases almost all the muscles in the body may be tender, but the biceps and muscles of the leg are often exquisitely tender, and this is true of practically all the cases I have seen. But certainly in this epidemic it is extremely common to find that the abdominal muscles are also very tender. It is, of course, impossible to be certain, but I got the impression that we were really dealing with two infections, the influenza, and a complicating infection of the respiratory tract and particularly of the lungs, which manifests itself as a broncho-pneumonia. This is so similar to the epidemic seen last year in America that certain comparisons are unavoidable. There measles was the primary infection, and on top of that a streptococcus invasion. Clinically I think it would be extremely difficult to differentiate the complicating pneumonia in these cases of influenza from the pneumonia complicating the cases of measles. I have therefore briefly discussed the two separately. The pneumonia has been so well described that I shall only draw attention to two other features which have seemed of interest on going through the wards of the camp and base hospitals in France. I had been accustomed to see the cyanotic type of pneumonia throughout the hospitals in France, and then I went to Brest, where there was another type of case with which I was not familiar and with which those there were. The patients were brought off the steamers almost always delirious, often pale, collapsed and afebrile, and always died. It was ascribed to the fact that many of these patients, crowded on the deck of the transports, could not have had proper care or food, and that there was in combination with their pneumonia a condition arising from lack of food and nourishment which produced this particular picture. The other specially interesting feature, besides the emphysema, is in connexion with the subacute cases which have recovered from their acute attack of pneumonia, and the temperature falls, but the patients continue dyspnoeic and cyanotic, and the diffuse râles persist all over the chest: they continue to expectorate large quantities of purulent material, their general condition improves distinctly and they are physically very much better. X-ray photographs of such cases have shown scattered shadows throughout the lungs, and from a study of the pathology it appears
probable that many of these pneumonic patients recover with a bronchiectasis such as was found quite frequently amongst cases of streptococcus pneumonia, and that these prolonged cases represent the starting point of bronchiectasis. The fact that we have rather considered influenza as one disease, and the complicating pneumonia really as a complication and as another disease, has led to a form of segregation of these patients which has proved of distinct benefit.

General Thayer spoke of the importance of immediate hospitalization, and it might be added that isolation and segregation are equally important. The hospitals that segregated and isolated their cases of influenza immediately had many fewer cases of pneumonia than those in which this procedure was impossible. For instance, in one hospital where they went still further and tried to separate their cases according to the organism which was found in the sputum—the patients being separated in wards which were sheeted so that each patient was screened from the next—subcultures were made, and those patients who did not show a streptococcus or a type pneumococcus in their sputum were sent to one ward; after two or three weeks it was found that pneumonia was very uncommon in that ward as a complication, whereas pneumonia was a much more common complication in the wards where patients with the streptococcus or pneumococcus in their throats had been placed. An attempt was therefore made to isolate the cases of influenza, to separate them one from the other, although this was almost impossible in hospitals to which large numbers of cases of influenza were sent. One other experience, however, points out the possible value of this method of segregation. In one camp hospital which I saw there had been between 700 and 800 cases of influenza, and at the earliest sign or symptom of pneumonia—because, as several of the speakers have pointed out, early diagnosis of pneumonia is extremely difficult and often has to be presumed from the symptoms and temperature rather than the physical signs—it was found that among the cases so carefully segregated the incidence of pneumonia was extremely small. There were amongst 700 cases of influenza only twenty cases—not 1 per cent. In another centre where immediate hospitalization was impossible and a long isolation was impracticable, among about 5,000 cases there were 1,200 cases of pneumonia—or 24 per cent. Of course, there are other factors besides the isolation which come into play. In résumé, however, I should say that we have been impressed with the importance of considering influenza as a clinical entity if possible, presenting certain very interesting symptoms,
Mr. Herbert Tilley.

In my experience the present epidemic differs from many of the previous epidemics in that symptoms referable to the nasal accessory sinuses, the upper air passages and the ear are uncommon. In a previous epidemic I have seen seventeen cases of acute suppurative inflammation in the nasal sinuses in a period of three weeks, but I cannot recall a single case during the present visitation. Nevertheless, Colonel French has told me that at Aldershot he has found the sphenoidal sinus full of pus in twenty cases which came to post-mortem: it is possible that the symptoms which would be caused by such a focus of infection may have been crowded out by the severity of those due to the general infection. On the other hand the sinus infections may have been the cause of meningitis in those cases which died and in which cerebral symptoms were predominant. I wish to lay stress on the relation between suppurative lesions in the nasal sinuses and pneumonia, and in support of this view I would refer to a significant report of Dr. Darling (Pathologist, Ancon Hospital, Isthmus of Panama): "The Accessory Nasal Sinuses and Pneumococcus Infections" (Journal of American Medical Association, November 10, 1906, p. 1561).

Dr. R. Murray Leslie.

My chief purpose in taking part in this discussion is to refer to a very formidable late complication, or rather sequel, viz.: Post-influenzal pulmonary tuberculosis.

In regard to the frequency of respiratory complications, there is a very close resemblance between the clinical characters of the present October epidemic and those of the epidemic of 1890. The incidence of bronchitis and pneumonia has already been dealt with fully by previous speakers, but practically no reference has been made to pulmonary tuberculosis. During the 1890-92 epidemic, I was working in the pathological department of Edinburgh University, and amongst the large number of pneumonic lungs exhibited, there was quite a considerable proportion showing tuberculous caseous broncho-pneumonia. I have

and, like measles, predisposing to a secondary pulmonary infection, which with us in the American Expeditionary Force has been a highly fatal and unusual form of pneumonia.
already seen similar cases of acute tuberculous broncho-pneumonia during the past month. These cases of acute tuberculous pneumonia are comparatively rare, but there have been a large number of cases occurring some weeks or months after an attack of influenza, exhibiting the more usual manifestations of pulmonary tuberculosis, including positive bacillary sputum. During the past year I have had charge of tuberculous wards at one of the metropolitan military hospitals, and during the last few months some twenty tuberculous soldiers have been admitted under my care with a recent history of influenza. Not a few have had a tuberculous family history. These cases exhibited symptoms and signs of tuberculous disease of the lungs within periods varying from a fortnight to four or five months after an attack of influenza. In two instances an haemoptysis occurred almost immediately after the attack, tubercle bacilli appearing in the sputum later; and in one case the tubercle bacilli were found within a week of the onset. Some of the influenzal histories coincided with the April and June epidemics at the Front. A fair proportion of my other cases of tuberculosis were originally admitted to the F.A.'s and C.C.S.'s under the classification of P.U.O., some of these with a sudden onset being no doubt cases of influenza.

An obvious criticism of the so-called post-influenzal tuberculosis would be that the cases referred to were really tuberculous from the outset, in view of the fact that all transient and irregular febrile attacks occurring during an epidemic are apt to be put down to influenza, when they may be actually due in most cases to the flaring up of a concealed focus of tuberculosis. While admitting the cogency of such criticism, it is probably true that influenza often acts as the spark which lights up the conflagration. In any case, it is a wise precaution to be doubly careful in the convalescent stage of influenza in the case of patients with a constitutional or family predisposition to tuberculosis. I believe that influenza, although in most cases it merely finds out the weak spot and lights up an old quiescent or latent infection, does in a smaller proportion of instances actually determine the first onset of a fresh infection. This is not surprising, when one remembers the extreme prostration, and consequent great diminution in resisting power to both acute and chronic infections, which so often accompanies the convalescent stage of influenza, and also the frequent bronchial and respiratory complications which tend to cause abrasions and increased permeability of the mucous membranes. The action of influenza is comparable to that of measles and whooping-cough in children, which
are probably the most important of all predisposing causes to tuberculosis in early life. There is one important difference, however. In the child we have young healthy, activating bronchial glands, which act as fairly efficient filters for the tubercle bacilli, and so serve to protect the adjoining lungs; while in the adult these glands are largely functionless and afford but little protection against lung infection. There is, further, a much greater prostration than in measles and whooping-cough, with corresponding reduction of resisting power. Apart from the increased permeability of the mucous membrane, the congested and damaged bronchial and alveolar tissues no doubt form an excellent nidus for the ubiquitous tubercle bacillus. Sir Arthur Newsholme and Dr. Stevenson have exhibited a valuable and suggestive series of curves and tables illustrating the incidence of bronchitis and pneumonia during influenzal years. A similar series showing the incidence and mortality from tuberculosis, taken at monthly or three-monthly intervals after the recent epidemics of influenza, might throw valuable light upon the actual relationship between influenza and tuberculosis, and the degree of importance to be attached to this acute disease as a determining cause of tuberculosis. The same course might be adopted in regard to the measles epidemics occurring during the War. One would expect the tuberculosis figures to rise in from three to eighteen months after the epidemics. The present high incidence and mortality of pulmonary tuberculosis, which has been steadily rising since the onset of the War, may be partly due to the frequency of catarrhal conditions following the widespread epidemics of measles and influenza.

Among the rarer complications seen during the present epidemic of influenza were two cases of femoral thrombosis occurring in a series of 100 consecutive cases of influenzal pneumonia admitted to my civil wards during the past five weeks, the thrombosis occurring during the stage of convalescence of influenzal pneumonia. Acidosis has been a fairly common accompaniment. Reference has been made to a series of twenty-two cases of nephritis at the Front, most of which were fatal, which later post-mortem findings proved to be due to a recrudescence of chronic renal disease. Albuminuria has been extremely common in this epidemic. I have recently come across two cases of transient haematuria with tube casts, occurring as complications of influenzal pneumonia, and in each case the urine entirely cleared up within a week, indicating a purely temporary inflammatory congestion. In regard to streptococcal empyema—a grave complication—I have had a succession of these cases, in each of which one pleural cavity was
found on exploration to contain comparatively clear straw-coloured fluid, loaded with streptococci; the fluid only became turbid later. Each case recovered after resection and the withdrawal of over two pints of the infected fluid. In one of these cases bacteriological investigations by Captain Carnegie Dickson indicated that a large number of the streptococci were degenerated; many of them were found inside the phagocytes, showing that the resisting power of the patient, a young nurse, was beginning to get the upper hand, as indeed was indicated clinically by a gradual fall of the temperature and improvement in the general condition before the operation of resection was carried out.

In these streptococcal effusions it is advisable to evacuate the fluid before it becomes visibly purulent. A preliminary aspiration is often followed by a general improvement, and a lessening of the toxæmic symptoms. The rib resection may be done a day or two later and the cavity drained. A vaccine may be given if desired, although the condition usually clears up quite satisfactorily without inoculation.

Dr. F. G. Crookshank.

I should like to ask if any of the American officers present can give me any information concerning the epidemic which, at the end of 1915 and the beginning of 1916, according to the American journals, ran rapidly through the States. One was extremely struck to see the statement that it was a wave of influenza such as had not been seen for thirty years; while in subsequent papers it was stated that in May, 1916, there was a great number of outbreaks throughout the States characterized by severe pneumonia. Later authors, however, took up the position that this epidemic in the early part of 1916 was not a true influenza, but a "pseudo-influenza," because Pfeiffer's bacillus was so conspicuous by its absence. The epidemic was immediately succeeded by the poliomyelitis of 1916.

Major Fred. M. Meader, M.C., U.S.A.

I am unable to furnish any detailed information on the point just raised.

The Royal Society of Medicine

Major Fred. M. Meader, M.C., U.S.A.
(Sanitary Officer, Winchester Area.)

The following is an account of an outbreak of an acute respiratory disease aboard a transport.

General Features of the Outbreak.

The transport left the port of embarkation and arrived seven days later at the port of debarkation with 5,951 troops. On the day the transport arrived 571 cases of an acute respiratory disease developed among the troops. Including this period and up to three weeks later 1,668 cases developed, or about 28 per cent. of the total number of passengers. Of those who became ill 534 (32 per cent.) developed pneumonia. Of those who developed pneumonia 317 died; this is 19 per cent. of those who became ill and 59.3 per cent. of those who developed pneumonia.

Sanitary Officer's Report.

The Senior Medical Officer, Major Charles Bagley, Jr., M.C., U.S.A., reports that up to 2 p.m. of the date of arrival at the port of debarkation the sanitary conditions on the transport as applied to mess, ventilation, sleeping quarters, latrines and hospital facilities, were entirely satisfactory, but that during the previous forty-eight hours a number of cases having respiratory symptoms were reported. The onset was usually very sudden, and in several cases the temperature on admission to hospital was 103° to 105° F. These cases appeared entirely independently of the infectious diseases, of which the following developed during the voyage: Fourteen cases of mumps, sixteen cases of measles, four cases of scarlet fever, three cases of pneumonia, two cases of scabies, and several cases of an acute respiratory disease.

The report for the next day stated that the number of cases developing severe respiratory symptoms increased tremendously during the last twenty-four hours. The dispensary and hospital staff had been on duty throughout the night, during which time several hundred cases had been treated. The hospital was filled, and the men were placed in blankets on the deck immediately surrounding the hospital. The report continued: “In view of the fact that approximately 1,000 men
are now too ill to travel, and there is an additional larger number who will be seriously ill within the next twenty-four hours, it is strongly recommended that the troops be removed to an open-air camp."

A conference of the Senior Medical Officer with the officials of the debarkation medical staff was held, and it was recommended that all members of the Army Nurse Corps remain aboard the ship until further orders, and that as many nurses as necessary be placed on duty taking care of the sick troops aboard the ship. It was also recommended and approved that the medical officers aboard the ship, together with such enlisted personnel as was necessary, be placed on duty.

During the afternoon such portions of the troops as were able were disembarked to a rest camp located near by. During the next three days the sick aboard the ship were distributed to nine hospitals. A large number of cases developed from the camp, so that the camp was not cleared completely until about a week later. The hospitals above mentioned were filled to accommodate the sick, and the camp was crowded to its utmost capacity with troops and those who were sick. Through the courtesy of the British medical authorities wards were made available in two of their near-by hospitals for those who were seriously ill. There was considerable difficulty in obtaining adequate transportation, so that hospitalization was not accomplished as soon as it was desired.

**Epidemiological Data.**

It was felt that this outbreak should be studied as thoroughly as possible. Accordingly an officer was detailed to obtain as much information as possible, in order to suggest any corrections of defects that might be found. This officer prepared a list of about twenty-five questions which would indicate any debilitating circumstances which might contribute to the rapid spread of the outbreak. This questionnaire was presented to the patients in the various hospitals, and with the assistance of the local staff and convalescent patients it was possible to obtain the information quickly and adequately.

The first question which arose was as to the distribution of cases among the various organizations aboard the boat. It was found that in one organization 41.3 per cent. developed the disease before leaving the boat, and 6.8 per cent. after leaving the boat. The disease was distributed throughout all the organizations; a number of them had from
16 to 30 per cent. of the total strength of their organization ill with this disease before leaving the transport. Of the 300 nurses who were aboard the boat, in one group 6:9 per cent. were ill, in another 5 per cent., and in the third group 0:9 per cent. were ill before leaving the boat. They took an active part in caring for the sick troops, and after leaving the boat the percentage of nurses becoming ill by organizations were as follows: 28:8 per cent., 33 per cent., and 43 per cent. These nurses evidently contracted the infection from their patients.

Another question of interest was the kind of troops which were aboard the boat, in respect to their civil occupation and the period of time they had been employed in the military service. It was found that two-thirds of them came from communities having under 6,000 people, that 58 per cent. of them came from farms which were for the most part located in the north-west, 24:4 per cent. were from Wisconsin, 14:7 per cent. from Minnesota, 12:7 per cent. from Illinois, 17:2 per cent. were from Tennessee. Of course this last mentioned state is located more in the south-east than in the north-west of the United States. It was further found that 10:4 per cent. of the troops went to their training camps last May, 23 per cent. last June, and 41 per cent. last July, showing that for the most part the troops had not been long enough in military service to become hardened to it.

Information was obtained from each patient which would indicate the debilitating circumstances of the journey. Practically all the troops complained that the shower bath of sea water which many of them received twice on the journey, was a very severe ordeal. It was claimed that they were obliged to remain undressed without covering for a considerable period, and many of them date their colds from this experience.

The men further complained that the food issued in the ration was not seasoned sufficiently to make it palatable. Many men took only a few regular meals throughout the journey. They patronized the canteen, purchasing crackers, sardines, apples, "pop," canned salmon and candy. This food was eaten at too irregular intervals, and in quantities insufficient to afford proper nourishment. It was felt that their digestive system was very much deranged by this method of living.

Many of the men complained that they were obliged to sleep in hammocks in a room where the lights were on all night, and the air was very foul, so that they could not rest. The nervous tension in passing through the danger zone was such that it also contributed to their lack of sleep.
What effect had the matter of distance of the hospitals from the port of debarkation upon the incidence of pneumonia? The patients were transferred by hospital train to the hospitals which were the most distant. One hospital was located 180 miles distant. Three hundred and eighty-nine cases were sent to this hospital; of these 141 developed pneumonia, and 135 developed the disease by the time of admission. Another hospital only eight miles distant took 271 patients, and had only forty-five cases of pneumonia upon admission. These patients were all carried by ambulance. The inference that transmission a long distance was entirely responsible for this difference in the number of cases of pneumonia is not entirely warranted, because of the fact that several cases who were seriously ill were sent to the first mentioned hospital; but the general opinion of the men having this matter in charge was, that a long journey, even in a comfortable hospital train, is not without its injurious effects.

Another question arose: Whether very many secondary cases of pneumonia developed in the hospital to which these patients were sent? From a study of the data it was apparent that for the most part few cases arose within the next three or four days, but in one hospital there was a distinct outbreak of seventeen cases on the twelfth day and sixteen cases on the thirteenth day after admission to the hospital.

The mortality in the various hospitals was very high: it varied from 37.3 per cent. of pneumonia in one hospital to 65 per cent. and 77 per cent. in other hospitals. In two hospitals all the pneumonia cases died. This is explained by the fact that at one hospital they were all nearly moribund upon admission, and in the other hospital there were but a few cases, and they were all of the post-measles type of pneumonia.

**Experiences During the Next Journey of this Transport.**

One month later this same transport arrived at the same port of debarkation with an even larger number of troops. The incidence of disease was very much smaller, and the method of handling infectious diseases was very different. Troops were carefully examined before admission to the transport, and 138 excluded from embarkation. All were required to wear masks over the nose and mouth throughout the journey. The temperature of all persons aboard the boat was taken daily, and all having a temperature of above 99.5°F. were isolated in the sick bay. Again, the nose and throat of all persons aboard the ship were
sprayed with a solution of iodine and albolin. The result was that on the first day out at sea about ten persons were isolated, the second day forty-five, the third day thirty, the fourth day nine, the fifth day thirteen, the sixth day ten, and the seventh day twelve. Many of these cases were released before the journey was completed, so that on the day of debarkation only thirty-five patients were transferred to hospital, two of whom had pneumonia. From the above experiments it might be assumed that the method of control had much to do with checking the spread of the outbreak.

It happened that about this time another transport from the same port of embarkation arrived at the port of debarkation, on which there was a similar small number of cases of acute respiratory disease. The precaution which was taken in this instance was that temperatures were taken every day, and those having a temperature of 99.5° F. were isolated in the sick bay. This experiment seems to have been a control to that used on the first transport. It would seem that the important measure to prevent the spread of infection was promptly to isolate all with a fever.

On further investigation it was found that the troops on both these transports came from the same camp where this outbreak of acute respiratory disease had been prevalent for some time. Large numbers of men had been ill in their organizations. These troops were the unattacked survivors of an extensive outbreak, so that they might be considered as immune to a large extent from this disease. This fact would lead us to inquire how far any of these measures adopted had been instrumental in checking the spread of this infection. It should be further pointed out that at the time of the extensive outbreak aboard this transport there was a very severe outbreak of this acute respiratory disease in the camps from which these organizations came. This journey on the transport appears to have been an incident in the history of a comparatively small number of troops who were extensively infected with a virulent organism during the period of its increase in virulence. The outbreak was prevalent in the camps and aboard other transports with a similar degree of virulence. At the time of the second trip of this transport the incidence of the disease had already begun to subside, in the camps and aboard other transports. It is true that more rigid measures of control were in use at the time, but it is a question if the infectious agent had not already attacked those who were susceptible and accomplished its severest effects.
Conclusions.

(1) One of the most effective measures of checking the spread of respiratory diseases among troops is to space the men over a large area. The case-incidence aboard this transport dropped immediately as soon as the troops were transferred to the rest camp. It is true that they were crowded at the camp, but the crowding was not so extensive as it was aboard the boat.

(2) Probably the best method of dealing with cases ill with an acute respiratory disease is to treat the patient at the place where he becomes ill, by rest, warmth and good food, in other words, good nursing.

(3) The debilitating conditions aboard the boat, such as character of food, insufficient sleep, and poor ventilation, may have contributed somewhat to the extent of the outbreak. These, however, were probably no worse than on other trips.

(4) The appearance of this outbreak was probably due not so much to unfavourable conditions of environment, lowered individual resistance on account of fatigue, exposure, irregularity of food, immaturity and so forth, as it was to an extremely virulent infectious agent, which was prevalent not only aboard this transport but also aboard other transports, and in the camps from which these patients came. The actual number of cases may have been increased by so-called lowered resistance, but this alone will not explain the outbreak.

Mr. F. B. Turner.

I wish to speak on the clinical aspects of the epidemics of influenza of 1889, 1891, 1893, and up to 1895, as it fell to me—and I do not think it did to many of you—to go through the mill in the treatment of these epidemics. I believe that these epidemics from 1889 onwards were not quite the same as the one we are having now. The clinical symptoms certainly differed. Bacteriology was not then a power under the microscope, and we had to rely upon what we could gather from morbid anatomy and clinical observation. The cases began exceedingly suddenly. I was perfectly well on leaving one house one afternoon, and exceedingly ill before I got to the next, and my principal complaint was pain, exceedingly distinct, behind the eyes and down the back, particularly in the line of the spinal column, and at the back of the legs, and nothing much anywhere else. There was very little occipital or
frontal headache; it was behind the eyes. The temperature did not run quite so high as in this last epidemic. Seldom did one find anybody with a temperature much above 103.2° or 104° F. The general range was from 100.5° to 102° F. The pulse rarely tallied with the temperature; it was either a little too quick for the temperature or too slow, but without any of the ill-effects supervening which you generally find when there is that difference between pulse and temperature in other diseases. The eye had a curious appearance, which I can only attempt to describe as "foxy." The tongue was not moist or coated; in almost all the cases it was very like one of those dates which are seen dried on a stick; and there was a strange touch—I can only describe it as a sort of "tang"—in the skin, which was so characteristic that I believe I could have diagnosed a case of influenza by simply touching the person, after dealing with one or two thousand cases. It was quite a different feeling in the skin to what I have experienced in any other illness. We had many complications in those days—bronchitis, broncho-pneumonia, and pneumonia. Some of the cases of influenza absolutely simulated enteric fever. The first I ever saw, about October, 1889, I mistook for enteric and treated it as such for a fortnight. There was some excuse for so doing because this particular gentleman had come straight from Marseilles, and any person who developed a continued feverish temperature under such circumstances we looked upon as possibly suffering from typhoid fever, as this disease was then endemic at Marseilles. All the symptoms of that illness were present except the spots, until at the end of a fortnight it "fizzled out," and I had to console myself with the thought that it was "one of those fevers of miasmatic or malarial origin which were not yet thoroughly and efficiently classified." Afterwards, when I knew what it was, I saw many of these cases of influenza simulating enteric.

We wrote much about it and we discussed it as we are doing now, and we came to the conclusion that the specific infection attacked more particularly the nerve centres, and we considered afterwards that this opinion was justified to a great extent by the very pronounced nerve sequelæ which we observed. Many patients recovering from a case of influenza which had run a normal unchecked course frequently suffered for six or nine months, or even a year, from pronounced symptoms such as depression, neurasthenia, neuritis, and other ills which we could only describe as "nervous." It seemed that the pneumogastric nerve perhaps at its origin might be particularly affected, because the complications principally involved the stomach and lung. One of my
most difficult cases was a very bad influenza complicated by severe vomiting, and I could do nothing for that person until I had subdued the vomiting. The description I have given you of these cases differs very distinctly from what you have heard with regard to the present outbreak. Such cases ran on from 1889 to 1895 and then by degrees they "fizzled out." In those years when there was no actual epidemic, one used to hear of a very large number of cases of "influenza" which I steadfastly refuse to believe were true influenza. This state of things continued until 1900, when there was a very severe outbreak in January, which was somewhat akin to the epidemic we had last July. That passed off, and then again, for the long period from 1900 until this year, I have seen very few cases which, judged by what we called influenza in 1889-91, could be written down as such.

Dr. W. E. Carnegie Dickson.

Vaccine Treatment.

From practically the commencement of the War, at Fulham Military Hospital and at the Royal Hospital for Diseases of the Chest, we have treated cases of bronchitis, broncho-pneumonia, pneumonia and allied conditions of the respiratory tract with vaccines. At first I prepared autogenous vaccines for each case, but later, as my stock emulsions of these accumulated into hundreds, I mixed them into a multivalent stock vaccine, making a rough standardization of each as I added it, so that a given amount of the mixed emulsion would give on dilution a stock "mixed catarrhal" vaccine with a fixed total number of the contained organisms. My clinical colleagues found the use of this "mixed" vaccine sufficiently promising to warrant its routine use in such cases, and the majority of them have shown their faith in it by themselves undergoing prophylactic, and at times therapeutic courses of it. If in any given case the results were not sufficiently favourable, I made an autogenous vaccine, and usually added a proportion—usually one-third—of my mixed catarrhal emulsion, with on the whole good results. I adopted the same procedure as that detailed above in the present epidemic and my "mixed influenza" vaccine is now made from fifty cases (throat swabs from some twenty cases with high temperature before the onset of complications; and from washed sputum or throat mucus from about thirty cases with bronchitis, broncho-pneumonia, &c.), the routine media used being
agar, blood-agar, broth, blood-broth, blood serum and anaerobic meat-broth. The approximate composition of this vaccine, taking the whole as "twenty," is:

<table>
<thead>
<tr>
<th>Organism</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Streptococci</td>
<td>5 parts</td>
</tr>
<tr>
<td>Staphylococci</td>
<td>5 &quot;</td>
</tr>
<tr>
<td>Pneumococci</td>
<td>3 &quot;</td>
</tr>
<tr>
<td>Minute Gram-negative influenza-like bacilli</td>
<td>3 &quot;</td>
</tr>
<tr>
<td>Friedländer's bacilli</td>
<td>2 &quot;</td>
</tr>
<tr>
<td>Other organisms (Micrococcus catarrhalis, &amp;c.)</td>
<td>2 &quot;</td>
</tr>
</tbody>
</table>

For prophylactic use, 60, 100 and 150 millions total organisms were given to adult males at weekly intervals; 40, 60 and 100 millions to adult females, and the amount of reaction produced by these doses appeared suitable in the majority of cases, being either practically absent or only slight malaise and a very slight rise of temperature, and in some cases "stiffness" of the legs, neck, or elsewhere, being recorded. After fully testing the dosage of the vaccine on healthy persons (beginning with myself), we next proceeded to try it on influenzal cases with temperature, but with no pulmonary complications, with results that my clinical colleagues will be better able to assess than myself. They have all expressed themselves convinced that the results were beneficial, and that in most cases the disease was apparently shortened and the occurrence of complications prevented. Next we very cautiously approached the question of the treatment of cases with purulent bronchitis, broncho-pneumonia, pleurisy, empyema, &c., and I at first advised small doses (20, 40, 60, 80 millions, and so on), at intervals of three to four days, according to the nature and severity of the case. I am now inclined to advise rather larger doses, and the intervals may also be cautiously decreased. We have, perhaps, not yet a sufficient number of cases upon which to base a scientific opinion, but again my clinical colleagues have had a considerable degree of success in such cases when these were not actually moribund. I have myself given the vaccine prophylactically to about 500 persons, and so far as I can ascertain, only two of these have since developed anything resembling influenza—one girl who says she felt ill the day I gave her the injection but did not tell me at the time, and one man who also felt unwell at the time of his inoculation. The first case ran a mild course of a week, without complications. The second developed some purulent bronchitis, but was doing well when I last heard of him.

As my general findings, bacteriological and pathological, are on the whole very similar to those of the various official reports recently.
published, and also to those of the immediately preceding speaker and several others, I need only refer to one or two points where my run of cases appears to give slightly different results. First, in addition to haemolytic diplostreptococci, which were most frequent, and pneumococci which were next so, as organisms of secondary infection following upon, or in many cases coincident with, the primary infection with Gram-negative influenza-like bacilli, I should like to emphasize the comparative frequency of the presence of staphylococci, especially *Staphylococcus aureus*. I have found the latter in the cultures of the washed sputum of rather more than 50 per cent. of the cases with pulmonary complications, and in the lungs and pleuræ in about a quarter of the fatal cases, in addition to the usual strepto- and pneumococci and *Bacillus influenzae* itself. It has also been present in ten pleural fluids removed by chest puncture during life—four times in pure culture. It should, therefore, I think, be regarded as one of the important organisms of secondary infection, though it has not been included in the official vaccine. Friedländer’s bacillus also occurred sufficiently frequently to merit inclusion. It was frequently present in moderate or in small numbers, and in at least one case it was practically the only organism in addition to *Bacillus influenzae* found in the pneumonic sputum. Meningococci have not been found in this series in any case, nor have any cases of influenzal meningitis occurred, although Fulham Military Hospital is the centre to which the cases of suspected epidemic cerebro-spinal meningitis in the London district are sent, and one might therefore expect some, at all events, of the possible influenzal cases to come to us along with these. No cases of meningitis due to *Bacillus influenzae* have developed in hospital.

With regard to the occurrence of *Bacillus influenzae* itself—I have hitherto had little doubt that it was the probable primary infective agent concerned in the production of the epidemic, or that it is at all events one of the organisms most constantly found, and is certainly itself pathogenic. In the first few cases I had some difficulty in finding it, but latterly it has occurred in practically 100 per cent. of my cases—especially in throat swabs from cases without sputum, but also in the washed sputum of bronchitic and broncho-pneumonic cases—

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1 Since the above was written, the extremely important preliminary note by Rose Bradford and his collaborators upon the occurrence of a filter-passing organism in influenza and other diseases has appeared, and we must await further details of their investigation, the confirmation of which will, of course, have a very important bearing upon the subject of this discussion.
often in enormous numbers, cultures on blood-media sometimes giving an almost pure growth of the organism.

With regard to my post-mortem findings, there is just one point I would emphasize. The condition of the lung and pleura is sufficiently described in various published reports, but, in addition to the dilatation of the right heart, I have practically uniformly found intracardiac ante-mortem thrombosis—not merely the so-called "chicken-fat" agony thrombus—but definite, tough, adherent white thrombus, beginning in the appendix of the right auricle, passing through the tricuspid into the right ventricle, with firm entangling adhesions around the chordae of the valve and the columnæ carneaæ, and often passing a varying distance into the pulmonary artery. For some fourteen years I have taught the importance of this occurrence in lobar pneumonia. It is one of the most important fatal complications of the disease, and merits the attention of the clinicians in charge of these cases. Greenfield and Fraser, in Edinburgh, used to urge the value of Fraser's original tincture of strophanthus in such cases as a preventive of the occurrence of such thrombosis, and strongly deprecated the use of digitalis and strychnine.

The presence of leucopenia is an important phenomenon in a certain proportion of the cases and at certain stages of the disease in the present epidemic. Apart from cases of influenza, I have had in the past few weeks several cases of acute streptococcal infection, some of them giving the organism on blood culture, characterized by varying grades of leucopenia. In some of these cases I have given normal horse serum intravenously, and noted a marked polymorph leucocytosis within twenty-four hours—e.g., to 15,000, 20,000 or more—with apparent benefit to the patient. This means of stimulating the bone-marrow to react seems to be worth trying in influenzal cases with leucopenia.

One other point to which I should like to refer is the important question of the carrier of the Pfeiffer bacillus. At the Royal Hospital for Diseases of the Chest, which includes the tuberculosis departments of the boroughs of Shoreditch, Finsbury and South Islington, I have carried out the routine examination of many thousands of specimens of sputum. One of the most noticeable features of these examinations is the frequent occurrence of small Gram-negative influenza-like bacilli in many of the cases of chronic bronchitis, in which, clinically, the suspicion of the presence of influenza has not arisen. I have frequently separated these organisms from such cases. They grow upon blood-media and conform culturally and morphologically with Pfeiffer's bacillus, and they constitute, I am convinced, the reservoir of infection.
in the country—a point of great importance for the consideration of the Public Health Authorities in dealing with the whole question of such epidemics.

_Vaccine Treatment._—Returns are so far very incomplete. Dr. C. J. Harrison, physician to Fulham Military Hospital, writes:—

I used your vaccine No. 1 in thirty-one cases of influenza at Fulham Military Hospital. After admission each man had 3 minims as soon as possible. None of these thirty-one developed pneumonia. Seven developed purulent bronchitis. All did well. I came to the conclusion that the period of fever in the uncomplicated cases—i.e., twenty-four cases—was shortened by forty-eight to seventy-three hours. The bronchitis cases did well also. The temperature was frequently on admission over 104° F.; it soon dropped. There was little local reaction, only a little soreness at point of injection. In private I used it on thirty-seven people as a prophylactic; none of them so far has developed influenza. All had two injections, the second seven days after the first. I had three cases of pneumonia. I used your No. 3 (weak) vaccine on each. All three are doing well (3 minims for first injection, 5 minims five days after the first injection). The lung change was in each case a broncho-pneumonia: each has been followed by a pleurisy. None of the cases (five) on whom I am using your anticatarrhal vaccine has developed influenza. They have each, since October 1, had five to seven injections.

Dr. S. R. Schofield, physician to Fulham Military Hospital, writes:—

One case of influenzal pneumonia was admitted to one of my wards, and I gave prophylactic treatment with the vaccine to the six nurses and the other sixteen patients in the ward. None have so far developed influenza.

Major Charles Gray, physician to Fulham Military Hospital, writes:—

_Therapeutic Inoculation._—Sixteen cases of influenza inoculated on admission. In every case the temperature came down within forty-eight hours, without any complications. In one case with pneumonia on admission the temperature came down within forty-eight hours. Several severe cases with complications were treated with small doses, and I am inclined to believe that the vaccine assisted recovery in several instances.

The statistics of the other physicians are not yet available, but are on similar lines to the above.

1 This vaccine contained 600 total organisms per cubic centimetre.

2 This vaccine contained 260 total organisms per cubic centimetre; the organisms being as described, except that of Bacillus influenza there were two parts, not three; and of other organisms three, not two.—W. E. C. D.
Dr. Moreland McCrea, physician to the Royal Hospital for Diseases of the Chest, writes:—

Prophylactic Inoculation.—Twenty nurses and sixty "contacts"—i.e., the patients in the two adjoining beds to cases of influenza—were inoculated. Only one nurse developed the disease. She was taken seriously ill the day following her inoculation—i.e., she already had the infection when inoculated and developed bad broncho-pneumonia. She was the only case of this type that I have seen recover. She had a second inoculation on the fourth day, and next day the temperature dropped, and she is now doing well.

Therapeutic Inoculation. — Fifteen cases, including both cases of high temperature and cases with pulmonary complications, were inoculated on the lines suggested. Only one died, and was the subject of extremely severe nephritis, whilst all the other cases did well.

Surgeon-Captain Bassett-Smith, C.B., C.M.G.

In accordance with the recommendations of the committee of bacteriologists held at the War Office, and with the sanction of Sir W. Norman, K.C.B., Medical Director of the Royal Navy, a prophylactic vaccine was immediately prepared, in quantity, at the Royal Naval College, Greenwich, and distributed to the depots and to ships of the Grand Fleet to be given to all on a voluntary basis.

Composition of the Vaccine.

Bacillus influenza (3-5 strains) ... ... 60 millions per cubic centimetre
Streptococci (3-5 strains) ... ... 80 " " "
Pneumococci (3-5 strains) ... ... 200 " " "

Dose.—First inoculation, 0·5 c.c.; second inoculation, 1 c.c.

The Plymouth strains of streptococci, all haemolytic, were incorporated together with other blood culture strains. At one depot pneumococci were more frequently obtained from the blood than streptococci, showing that at different places the secondary types of infection also differed in frequency.

In sending out the vaccine directions were given that it was not to be used if acute catarrhal symptoms or fever were present. One medical officer reports that in the course of the inoculations ten cases occurred. Four of these had received one dose on the day of onset only. All ran a very mild course. Of the six not inoculated two were mild and four severe, with one death. This pointed to the value of the vaccine even in the early stage. At Greenwich 1,000 boys of the Royal
Hospital School have received both inoculations, and no case has occurred among them though the disease was prevalent in the district. Amongst the thousands that have been inoculated the reactions, with one exception, have been slight, and there has been no evidence of an increased susceptibility or negative phase.

The preventive value of the vaccine against influenza bacilli is not high, but it is probable that an increased immunity against streptococcic and pneumococcic infections will be acquired, preventing the most severe complications and fatal results.

A total of 132,475 c.c. of the vaccine has up to the present been distributed in the Royal Navy.

There has not yet been time to receive results of the controlled experiments of inoculated and uninoculated which have been carried out.

The President.

My own feeling is that the protective inoculation may be of use, particularly in young people, who are more susceptible at the present time, in preventing the severe complications to which the mortality is due.

Major Newton Pitt, R.A.M.C.

I wish to make a few remarks on the question of treatment because negative conclusions may be of value, and the results of only partially successful treatment should be put on record.

The whole question turns very largely upon the nature of the epidemic. The severity of the epidemic varies greatly in different localities. In some districts there may be no serious cases, the illness runs its course without any complication, and there is hardly any mortality provided the patient is kept strictly in bed, and almost all recover under any simple treatment. In other districts one feels that from the first the disease is so virulent that the patient has very small chances of recovery, especially in those cases where he is intensely cyanosed, dyspnæic and lethargic; his skin soon becomes yellowish, with almost a post-mortem tint, and he becomes steadily more and more toxic, often with a slow pulse, and in spite of any treatment the majority will die. The most we can hope for is to save the less
severe of these cases. So much do the cases differ that all statistics as to the result of treatment are fallacious.

It is agreed that in the present epidemic three or four different organisms may be present, and it is a question whether we can at present differentiate between the cases clinically. The sputum may be hæmorrhagic, rusty and viscid, muco-purulent or frothy. When the sputum is muco-purulent from the first, the organisms are streptococci—often mixed with Bacillus influenzae—some of the hæmorrhagic cases are streptococcal, and the rusty sputum is mostly, but not invariably, pneumococcal; it would be a great advance if we could determine the particular organisms from the clinical symptoms, and successful results will obviously not be obtained by giving the same treatment to all.

In the cyanotic septic cases I have been in the habit of administering subcutaneously, with some benefit, especially when it has been administered early, antistreptococcal serum 20 c.c. daily, rising occasionally to 40 c.c. The maximum total amount has been 140 c.c. I have seen cases more or less unconscious with incontinence become conscious under this treatment, and able again to take food, and ultimately to recover. The improvement has not always continued, and even after six or seven days the temperature may again rise suddenly, the patients again become lethargic and die. Other cases have been treated with intravenous injections of 10 c.c. (1 in 2,000) of hydrarg. perchlor. with temporary benefit, but at present no efficient treatment for the most toxic cases is known.

It is the massive collapse of the posterior part of both lungs, which become airless, splenized, and full of exudate, and the inability of the patient to open up the alveoli again, which ultimately cause death.

Lieutenant-Colonel C. E. Cooper Cole, C.A.M.C.

Serum Therapy.

During the past seven weeks I have had over 2,000 cases of this epidemic under my care in hospital. We had ample opportunities of trying the various lines of drug treatment (even salicin to 30 gr. q.i.h.) which are usually advocated, and our results have been far from satisfactory. Our cases could be grouped as (a) toxic, (b) pneumonic, and (c) toxic followed by pneumonic. The last two types were in several instances complicated by empyema, and direct smears of the
fluid usually showed streptococci, occasionally pneumococci. We, therefore, decided to try multivalent antistreptococcic serum in addition to ordinary symptomatic treatment.

Up to November 2 we had treated 125 cases with doses varying from 5 c.c. to 100 c.c. at one injection. The maximum given to one patient was 210 c.c. The serum was administered subcutaneously, intramuscularly and intravenously. Finding that an initial 5 c.c. dose produced no harmful effects, we usually gave 10 c.c. to 20 c.c. twice daily. Since the above date we have tried giving 20 c.c. intravenously on admission, and then 10 c.c. twice daily till a satisfactory result was obtained. It seemed usually to shorten the course of toxic cases.

Our observations seemed to show the following advantages:

1. Reduction of toxæmia, as shown by delirium clearing and a sense of wellbeing, even though physical chest signs showed no change.
2. Frequently profuse sweating with lessened fever.
3. Pulse usually quieter and of better quality.
4. The fight for life was prolonged, even though unsuccessful in many cases.

In no severe cases was a dangerous reaction noted, and in only a few already moribund patients did the toxæmia seem to be increased as evidenced by increased stupor. Of thirty-two extremely toxic cases nineteen showed immediate improvement, five none, and eight went on into the pneumonic condition. We therefore concluded that antistreptococcic serum should be given as early as possible in all cases, even pneumonic cases showed benefit, and after draining local foci—e.g., empyemas—the benefit was marked. We continued serum treatment till a sharp serum reaction developed. We also used normal horse, antidiphtheritic and antipneumococcic serums in short series, though the normal serum was not as useful as the other.

We have lately been using eusol intravenously in doses of 5 c.c. to 250 c.c. In most cases there has been a sharp reaction, as indicated by chill, rise of fever, and then a rapid fall towards normal. In a few cases results look satisfactory, but natural variations in the course of the disease are so manifold that the amount of influence which we can exert on them is very problematical.
Mr. E. B. Turner.

Treatment (Curative).

In a certain number of cases I have tried a vaccine consisting purely of the influenza bacillus, and out of those cases in which I tried it—all of them youngish school-children—50 per cent. have had influenza in an extremely mild form. All my cases have occurred in absolutely private practice. I have had no cases in institutions or hospitals. The only place which can come at all under the definition of an institution is a large hostel which I look after, and in which 120 young ladies live—business women for the most part. I have to take you back to the old epidemics. In 1878 I was house physician at St. George’s, and during that year we were very largely engaged in the treatment of rheumatic fever, which established the position of salicin and the salicylates in therapeutics. I had to look after those experiments under the direction of the physicians to the hospital at that time. To get any success we had to press the drugs very forcibly. In a short time we got up to giving 30 gr. of salicylate of soda every two hours, and giving it with very great success. I had a good deal of experience with salicin and salicylate of soda and other preparations derived from the willow tree. When these epidemics first began I had a large number of cases, and I was not at all satisfied with the way they went. I had bronchitic and pneumonic complications and nerve complications, and the cases hung about for a fortnight or three weeks and then got up ill. I came to the conclusion that probably if I could get a person thoroughly under the influence of salicin I might be able to cut short the course of the disease. I happened to get a sharp attack of influenza myself one afternoon (temperature 103°6' F.). I went straight off to Savory and Moore’s, in Bond Street, and swallowed in the shop 40 gr. of salicin, and went home with 20 gr. of salicin in powders, one of which I took every hour during the night. In the morning my temperature was normal, and although I had the feeling of having been bruised in all my limbs, I got up and did my day’s work without any harm whatever. Having done this, I came to the conclusion that the method was one to be pursued. That same day I went to some friends where three sons lived at home: one of them had influenza which was complicated with broncho-pneumonia, which was running a slow course. I was informed that both his brothers had started influenza that morning, and I found
that the temperature of one of them was a little over 102° F., and that of the other within a point or two the same. I gave them 20 gr. of salicin every hour, with the result that they were perfectly well, out and about long before their brother, who was treated with salines and quinine, had left his bed. I must have treated more than 2,500 cases of influenza in all up to and including to-day. Out of all those cases I have not lost one single person, and I have had no complications, no pneumonia and no bronchitis. I have not had to have a single nurse in to look after them. It is true that every one of them was a person who was in such a position that he could be properly looked after, could go to bed and stay there, and could call in the doctor at the very beginning. But I take the result in comparison with the cases which I had under the same conditions but which I treated with other drugs. In those results some had complications and sequelæ, but since I adopted this method I have had no complications nor sequelæ nor deaths.¹

Not only has it had this effect in London, but in the bad epidemics of 1890 and 1895 it had the same effect in the country districts. There was one case where a whole village was down with influenza. The doctor simply could not cope with the conditions. A patient of mine had gone down there and suggested to him that he should ask me about this mode of treatment. As a result, a pound or two of salicin was sent to him, and the district was at once in hand. I published my first 250 cases in the Lancet in 1891,² and I put a memorandum about it last August in the British Medical Journal³ after noting the results of this last July outbreak. What I have told you obtained up to the end of the July epidemic. In the July epidemic I had seventy-nine cases treated in this way. Not one of them lasted more than three days. They reacted and responded to the treatment in exactly the same way as the cases did in the former years. The particular set of cases we are getting now, however, behave quite differently, and that is what makes me pretty sure that this epidemic is not quite the same as those with which we made acquaintance thirty years ago. One speaker yesterday said that he found a difference in the bacteriology as between the July and the October attack. During this epidemic I have had up to now 157 cases, and I have treated them all very much in the same way, but they have not got well within the

¹ The late Dr. T. J. Machigan had exactly the same results in his cases, which he also treated in a similar fashion; and he also published his results about the same time that I did.
² Lancet, 1891, ii, p. 121.
short time that my other cases did, but every one of them has recovered without any complication; there has been no pneumonia nor bronchitis, although the cases have hung on for a little longer than they did on former occasions. In fifty-eight cases I managed to get them under treatment, probably within two or three hours of the onset of the disease, and none of these were ill longer than three days. In thirty-seven I got the treatment in hand later, and they were ill for four or five days. In twenty-five or so no treatment was given for the first twelve hours, and they were ill a little longer still. Some of them went for one or one and a half days before coming for treatment. But they all got right without any complication.

If you get a person early in the disease and get him drenched with salicin, you seem to destroy the infectivity of that patient. In the bad epidemics in London, from 1889 onwards, I hardly ever had more than two cases in one household, generally the person who was first taken ill and the attendant or maid who looked after him or her before treatment was administered. I got the time of incubation very accurately, it was eighteen to twenty hours, and I gave the attendant salicin immediately on the slightest rise of temperature, with the result that the attacks were confined to one or two only, without spreading through the household. In cases where this treatment was not followed it was not uncommon for every individual in a household to be ill. If you get your patient quite full of salicin it seems to destroy the infectivity. This I have also noticed in these last two epidemics. The hostel which I look after, and which I mentioned just now, houses 120 girls, each of whom has a bed-sitting room, while other rooms are shared in common, such as dining-room and drawing-room—an ideal place for the spread of infection. In the July epidemic there occurred here seven cases of influenza, every one of which I could trace to an outside infection. All were treated as quickly as possible, and there was no spread. In this last epidemic in the same place I have had eleven cases of influenza, in every one of which I have been able to trace the source of infection from offices, &c., where the girl may have been working, and in no case whatever has there been any spread of the illness in the house itself by infection from those girls who were ill there. It is not of the slightest use playing with this drug. If you try to give the salicin in small doses you will do no good at all.

As to the effects of the treatment, I have had several rashes—sudamina, and nothing more. Very often it sets up a profuse perspiration, and sometimes there may be buzzing in the ears and temporary
deafness. I give 20 gr. every hour for twelve hours, 20 gr. every two hours for the next twelve, and continue it for the next twelve hours as the case seems to require. I have not wanted any stimulants. In my own case I felt wretched and miserable, with agonies of pain, but in four hours' time this had practically subsided, or at any rate it was bearable. In these later epidemics I have found rather more headache. In some of the cases where there has been pronounced headache I have given citrate of caffeine, with a grain of antifebrin with good results. In this last particular epidemic, although there have been no complications, the treatment has not had the same effect in cutting short the disease. It has had a very marked effect nevertheless.

The earlier the salicin is administered the better. This is the one treatment I have used for this disease since 1890, and since then in all the epidemics I must have had more than 2,500 cases. I have tried it also in a good many cases which I used to describe as "pseudo-influenza." As a general rule it did good, but it will do good very often in an ordinary feverish cold, though it has not the pronounced effect such as it has on influenza properly so called. I had one fatal case in, I think, 1895, when a man who had had chronic bronchitis and emphysema for years, recovered from influenza, but on the Christmas Eve of that year, some three weeks after his illness, there was a ghastly fog, and he left his window open, with the result that he had a very acute attack of bronchitis and died, just as he would have done whether he had had influenza or not.

I have not tried vaccine or serum treatments. I should be disposed in any case that might be necessary to supplement my purely old-fashioned treatment in this manner, but so long as I can get the results I have obtained I intend to stick to the same methods. I have given salicin in cases of valvular disease of the heart, with nothing more than the most trifling inconvenience. I administer it in the cachet form, so that the patient can swallow it without tasting it. I tried salicylate of soda; it does not have the same effect, and is also more unpleasant, causing more buzzing in the head, and, as in rheumatic fever cases, it may cause delirium.
Early in 1917 I had under my care in France a large number of soldiers suffering from a grave form of purulent bronchitis, proceeding in some cases to broncho-pneumonia. The cases exhibited dyspnoea, a heliotrope cyanosis, purulent nummular expectoration, pyrexia, and a high mortality; delirium was not a marked feature. Healthy young soldiers were usually attacked; in a few cases the condition occurred in association with acute nephritis. In about one-third of the cases broncho-pneumonic consolidation was recognizable either by clinical or post-mortem examination. Pleurisy was present in about one-fourth of the cases, usually dry, but occasionally with scanty effusion, never purulent. It was noticeable post mortem that the pleurisy did not specially affect the consolidated areas.

The mortality was about 60 per cent. Death took place at two stages: early, with suffocative symptoms, and late, from exhaustion. In the most rapid cases, death from asphyxia took place within forty-eight hours; the protracted cases dragged on for weeks, presenting a close resemblance to acute tuberculosis. Cases showing a gradual fall of temperature with rise of pulse-rate were invariably fatal.

In most cases the sputum was purulent at the first examination; in some, however, it was at first mucoid and frothy, and later, purulent and nummular; during the transition both types of sputum were present in the same spit-cup. Bacteriologically, the sputum contained enormous numbers of an organism corresponding in form and culture to Bacillus influenzae. Cocci were also invariably present, usually of pneumococcus type, but occasionally in chain form.

Although the disease was specially prevalent at the time mentioned, it was by no means restricted to it; indeed, during the first winter of the war, Sir John Rose Bradford had directed the attention of medical officers to the condition. The bacteriological findings suggested that the disease was of influenzal origin; it must be stated, however, that it occurred apart from any definitely recognizable epidemic of influenza.
Dr. J. T. C. Nash.

In dealing with the treatment of influenza it is apposite to ask, what is influenza? A definition would need to be extremely wide to cover all cases which are accepted as cases of influenza. The disease is almost certainly of bacterial origin, but whether there is a more or less specific micro-organism such as the bacillus named after Pfeiffer, or whether the micro-organisms associated with ordinary catarrh (such as Micrococcus catarrhalis) are endowed with enhanced virulence is at present uncertain. The complications are almost certainly due to other associated organisms, such as the pneumococcus, the meningococcus, and various streptococci, but all these have possibly had their virulence enhanced by symbiotic relationship. The main point is that influenza is an infective disease apparently due to pathogenic organisms in symbiosis, several of which can be cultivated from the sputa and some from the blood, thereby enabling the scientific method of treatment to be adopted by means of a multivalent vaccine. Evidence already given has indicated the marked influence of such a vaccine when treatment was commenced within the first four or five days. Serum treatment, whether by plain horse serum or by antistreptococcic serum, is also indicated, having the virtue of inducing leucocytosis to deal with the pathogenic organisms.

In the treatment of septic throats in scarlet fever I have often obtained quickly beneficial results by the administration of antitoxinum serum as well as of antistreptococcic serum. If serum or vaccine treatment is delayed in the treatment of any infectious disease, it is not likely to be of much avail, as either (1) the patient will have formed his own antitoxins or germicides and will recover unaided, or (2) his tissue resistance will have already been overcome by excess of toxins which have entered into intimate relation with his cells and cannot be ousted.

The remarkable results obtained by Mr. Turner by the exhibition of heroic doses of salicin in about 2,500 cases of influenza are strongly suggestive of bactericidal action (whether by the formation of salicylic acid or other compound in the blood is a matter for chemical investigation).

Apart from drug treatment, the method I have adopted in the treatment of influenza in my own family has been gargling with izar or permanganate solution and early resort to bed. The drugs used
have been quinine and camphor and oil of cinnamon, and occasionally salicylates.

Relapses and repeated attacks have been very common in influenza, indicating causal organisms of unstable or less specific type, which do not readily call up in the tissues of the body an excess of response in the formation of natural antitoxins. I pointed out in 1906 that symbiotic relationship of micro-organisms in the human body had yet to be studied in much more elaborate detail than in my own small contribution in that year. I believe that symbiosis has much to do with the evolution of pathogenic properties in organisms which are generally mere saprophytes. The relationship of bacteria to moulds is another department of bacteriological research which awaits organized study, and which will open up inexhaustible and fruitful mines of information to be applied in the treatment of infective diseases.

Professor John Eyre, M.D.

In March, 1917, I had the opportunity of investigating an outbreak in the Aldershot Command where the disease commencing with acute catarrhal symptoms, characteristic of "influenza," in a large percentage of the cases passed to a profound toxæmia with involvement of the bronchi and bronchioles and of the immediately adjacent pulmonary tissue in an interstitial—not lobar or lobular—pneumonia. This was labelled by those of us concerned in the inquiry as "purulent bronchitis." In these cases the expectoration throughout the whole course of the disease contained *Bacillus influenza*, at first the predominant, if not the sole, organism present, whilst lung punctures, too, yielded material which on culture gave a growth of *Bacillus influenza* either pure or associated with staphylococci, but no pneumococci. In the later stages the influenza bacillus was associated in the sputum with pneumococci, streptococci, *Micrococcus catarrhalis*, &c. The final stage of these cases was the entry of the pneumococcus into the blood stream and death from pneumococccic septicæmia. *Bacillus influenza* isolated from these cases possessed a high degree of virulence, and I was able to produce a fatal septicæmia, in four days, in the rabbit, after intrapulmonary inoculation. Previously I have never secured a fatal result with this organism in this animal save by intracranial inoculation.


2 See "Evolution and Disease," by J. T. C. Nash, M.D. (John Wright and Co., Bristol.)
In February, 1918, I again had the opportunity of inquiring into an outbreak in the Southern Command, where the history suggested that Colonial reinforcements had, on their way to England, acquired infection from America, where, as we now know from the evidence of Cole, McCallum and others, similar epidemic disease was prevalent, also associated with *Bacillus influenza* and, in addition, haemolytic streptococci.

In this second investigation the early stages of the disease resembled those of the Aldershot epidemic, but in the progress of the disease broncho-pneumonia and even lobar pneumonia, often associated with pleuritic effusion (in which streptococci of the haemolytic variety abounded) frequently occurred, and the termination was by streptococccic not pneumococcic septicæmia, but in all *Bacillus influenza* was the predominant organism in the early stages.

Subsequent outbreaks in different parts of England that I have had opportunities of observing have consistently exhibited the characteristics of the second type, that is to say, a primary *Bacillus influenza* infection, and in later stages the addition of streptococcic infection.

Since early last year, then, I have been closely in touch with the epidemic form of the disease in all parts of the country, and in my experience its early bacteriology has never varied. At the onset of the disease, when pyrexia and malaise are the outstanding features of the attack, nasal discharge and expectoration are usually absent or scanty, and pharyngeal mucus, nasal and post-nasal scrapings are alone available as material for examination. Later, tracheal mucus and nasal mucus may be obtained, although perhaps with some difficulty. Up to this point the microscopical appearances are always the same, viz., a ground-work of mucus, showing numerous leucocytes and thickly studded with minute Gram-negative bacilli, many exhibiting bipolar staining and thus simulating minute diplococci; frequently examples of both forms crowd the interior of the polynuclear cells. The cultures prepared from such material yield a good growth of *Bacillus influenza*, associated with occasional colonies of *Staphylococcus albus*, *Streptococcus brevis* and other similar saprophytes of the upper air passages. After an interval the mucus becomes distinctly purulent and coincidentally shows a much more varied flora—Gram-negative and Gram-positive diplo- and tetra-cocci in profusion, in addition to the Gram-negative bacillus, which is now much less easy to identify. Cultures at this stage show chiefly *Streptococcus longus*, usually *haemolyticus* or *viridans*, or both, *Staphylococcus aureus*, *Micrococcus catarrhalis* and
pneumococcus; and *Bacillus influenza* is difficult, or almost impossible, to isolate except by the experimental inoculation of mice and rabbits.

In the severe and fatal cases all, or many, of the organisms already enumerated are present in the bronchial mucus and their presence may be shown by culture in the infected lung tissue. The organism most consistently present is *Bacillus influenza*, which is present also in the spleen pulp, post mortem, in association with the streptococcus or the pneumococcus which has caused the fatal septicaemia.

Personally I have not succeeded in isolating *Bacillus influenza* from the circulating blood during life, but I have had instances of its successful isolation brought to my notice by others.

My opinion, based upon the observations I have summarized, is that the epidemic is one of true influenza, due to *Bacillus influenza*, complicated in a large percentage of cases by secondary infections with pneumococcus or *Streptococcus longus*, var. *haemolyticus*—one or other of these secondary infections being responsible for the terminal fatal septicaemia.

I consider that the *Bacillus influenza* completely fulfils the conditions required of an organism accused of specificity—that is to say, with the exception of the comparatively rare “carrier,” it is absent from the upper air passages of the normal individual; it is capable of artificial cultivation and such artificial cultures are in turn capable of initiating the disease in man with recovery of the organism from the induced lesions.

The spraying of emulsions of *Bacillus influenza* into bottle cultures (for the preparation of massive growths for vaccine) has frequently provoked mild attacks of the clinical disease in those of us engaged in this work in my laboratory. (The production of specific antibodies in infected and vaccinated men and animals has also been observed, and I hope to make further reference to this point at some future date.)

In view of these results I fail to understand the attitude of those who, confronted with the available data, refuse to accept the obvious and seek some mysterious and elusive pathological virus, filter passer, or what not, to explain a pandemic which is the exact counterpart of the one which rather more than a quarter of a century ago prompted the investigations which led to the discovery of *Bacillus influenza*; and, on the other hand, readily accept the causative association of the pneumococcus or the streptococcus with the final and often fatal lesions.

Having thus clearly defined my conception of the bacteriology of the epidemic, obviously the next step was to inquire into the practicability
of prophylactic vaccination, not only against the prime cause of the disease but also against the associated bacteria responsible for the secondary infections. And here instant and sympathetic assistance was forthcoming from those in charge of the welfare of the N.Z.E.F., with the result that early in April, 1918, protective inoculations were instituted, and from the evidence at our disposal, seem to have been the means of conferring a considerable amount of immunity upon the New Zealanders in England through the primary wave of the pandemic in the summer months of this year. Our results were recorded by Captain Lowe and myself in the Lancet for October 12, 1918, p. 484, and need not be reiterated.

The serious incidence of the disease in a transport of colonial reinforcements which arrived in England at the end of September led to an even more extensive utilization of the prophylactic vaccine, but there has not yet been time to collect and collate the resulting figures.

I do not propose to discuss at length the dosage of the various components of the protective vaccine, since this is a matter depending almost entirely upon the methods of isolation, selection of strains and "distance of generation" from the human body.

This last factor is, in my opinion, of the highest importance, since by every subculture (or generation) a pathogenic organism is removed further from its original habitat in the pathological exudate, and this means a variable but distinct loss of virulence. I consider that if one obtains a pure growth of Bacillus influenzae in a tube or plate planted directly from, we will say, tracheal nuculus, a dose of 5 millions of the bacilli from that culture is equivalent in "antigen" content to a dose of 50 millions of the bacilli in the tube representing the fifth consecutive subculture from that original culture. And whilst I am aware that the 50 millions of bacilli from my fifth generation will provoke little, if any, more local and constitutional "reaction" than the 5 millions of bacilli from my original culture, I am not prepared to admit that the former will provoke as big an antibody response as the latter.

Whilst on this point I would further remark that the enormous doses suggested in connexion with commercial vaccines represent to my mind the large number of generations or subcultures that have intervened between the specimen from which the organism was isolated and the particular culture from which the vaccine was prepared—and coincidently the loss of virulence the organism has sustained.

Again, at the moment we have no accurate data as to the amount of
antigen that is utilized and the amount that is superfluous, and therefore wasted in any given dose of vaccine. Even the small doses I have advocated are ample to provoke a very smart local and constitutional reaction in weakly, though otherwise normal, individuals such as myself.

Major A. Abrahams, R.A.M.C.

My remarks relate to some clinical features of the cases which we have been able to study in the Aldershot Command. The Connaught Hospital, Aldershot, is devoted to the treatment of medical cases supplied by a very large number of troops in a highly concentrated area, and with a normal complement of 500 beds. We extended these to 850 to accommodate the large number of admissions during the recent epidemic.

I wish first to refer briefly to the condition known as "purulent bronchitis" in the light of the disease now under consideration: those of us who worked in Aldershot during the winters of 1915 and 1916 were struck by the existence of a peculiar type of lung affection with which we were hitherto unfamiliar. The patients manifested the signs more or less of bronchitis, broncho-pneumonia or lobar pneumonia; but the very special features were the expectoration of enormous quantities of purulent sputum, a most characteristic heliotrope-coloured cyanosis, and a very dreadful mortality.

It was not until Lieutenant-Colonel French was appointed consulting physician to the Aldershot Command that we began to see the proper significance of these cases. Hitherto I had been disposed to regard them as occurring for the most part in young men of feeble physique and of short service who were unable to endure the exposure entailed by ordinary military service; and the circumstance that the bulk of the cases occurred in one unit was speciously explained by the fact that this particular unit was at that time being largely recruited from men of low grade. Colonel French, however, objected very properly that such cases were by no means restricted to the type which supplied the majority, and further, that older men with good histories and comparatively long service were occasionally affected, and post-mortem examination supplied the puzzling evidence that the lungs were relatively free from disease. His alternative suspicion of some infective factor satisfactorily explained

1 My colleague, Captain Hallows (p. 11), has already dealt with pathological details.
the prevalence in one particular unit and his further investigations even narrowed the field down to a few special huts in one unit. It appeared indubitable therefore that we were dealing with a highly contagious disease and his communication to Sir Alfred Keogh led immediately to an investigation along these lines, in which we were assisted by Dr. John Eyre. In this way we quite unexpectedly made the discovery that the \textit{Bacillus influenzae} was a factor in the condition, which appeared to be a primary infection by this organism that exalted the virulence of other organisms: the pneumococcus, \textit{Streptococcus longus}, \textit{Micrococcus catarrhalis}, and, rarely, other organisms constituting the secondary infection. We were then under the impression that our cases were endemic, more especially as we had learnt to recognize various peculiarities of respiratory disease for which this district is notorious; but just before we published our results, Hammond, Rolland, and Shore, who had been working in France, published an article\textsuperscript{1} which clearly showed that they had encountered an exactly similar condition. Since then, various observers have published their experience of the condition in different parts of the country.

In our opinion these cases originally described by Hammond, Rolland, and Shore in France, and by Dr. John Eyre, Lieutenant-Colonel French, Captain Hallows, and myself\textsuperscript{2} in Aldershot, were fundamentally the same condition as the "influenzal pneumonia" of the present pandemic. At the time when we were investigating "purulent bronchitis" there was no generalized epidemic to lead to the suspicion that it was of influenzal basis, and identification of Pfeiffer's bacillus was in this connexion of the nature of a discovery. But we are convinced that we are now witnessing the same disease upon a widely epidemic scale, notwithstanding an objection which may be advanced that one dominant peculiarity—the copious purulent expectoration—has been conspicuously absent in the recent cases. But this enables me to correct a mistake which we ourselves had perpetrated. We were disposed to regard the heliotrope cyanosis and the copious secretion of pus as interrelated, so that a mechanical explanation for the cyanosis was forthcoming. This we now see to have been wrong; the presence or absence of purulent expectoration is only an incident in the disease and not an invariable accompaniment. In really essential features—the heliotrope cyanosis and high mortality with variable lung condition—"purulent bronchitis" and "influenzal pneumonia" appear to us to


\textsuperscript{2} \textit{Lancet}, 1917, ii, p. 377.
be one and the same thing—an influenzal pneumococcal or influenzal-streptococcal septicæmia.

In this Command the relatively trivial cases are treated at their own units and only the appearance of alarming symptoms or of pyrexia persisting over forty-eight hours sends them to hospital. The large majority of cases have of course been relatively trivial. Roughly speaking, of 1,000 cases, 800 perhaps have taken an ordinary simple uncomplicated course with fairly speedy recovery and without sequelæ. The remaining 200 have displayed pulmonary symptoms, of these perhaps eighty of moderate severity, and the remaining 120 have been desperately ill; and of this last category between sixty and eighty have died.

Of the symptoms encountered in this epidemic only a few stand out conspicuously to demand description. Signs in the chest have been remarkably variable, but even when what could be ordinarily accepted as unequivocal evidence that consolidation has been present the familiar features of lobar pneumonia with rusty sputum have been absent. The relative absence of expectoration has been a dominant and surprising feature of this epidemic. On the other hand, hæmoptysis has not been uncommon often when all alarming symptoms have disappeared and the case is convalescent. But hæmoptysis seems to be only an indication that the lungs have shared the general hæmorrhagic tendency in this disease, for epistaxis (in a very high percentage of cases) and hæmatemesis as well as hæmorrhages elsewhere have been noted. And a further indication of this tendency has been afforded by those cases which have developed a pleural effusion in which the fluid has been sterile and the preponderating cell the lymphocyte.

The colour of the patient is of particular interest in view of the baleful heliotrope element. In general one may say that once this colour has appeared the patient’s condition may be regarded as desperate. It is evidently bound up not so much with mechanical disturbances in the pulmonary circulation as with the condition of septicæmia. At one time we thought the colour might be due to methæmoglobinæmia, or even to the production of some other pigment in the blood, but the spectroscope has revealed no abnormal bands nor is there, judging from our experiments, any defect in the oxygen-carrying capacity of the blood. The condition is apparently that which Dr. J. S. Haldane has termed “anoxæmia,” precisely similar to what is seen in cases gassed at the Front, and possibly due to analogous causes, to judge from the histological changes seen in the lungs.
Abrahams: *Discussion on Influenza*

Although the *respirations* are rapid, orthopnoea is absent, and indeed the condition appears to be a polypnoea or tachypnoea rather than a dyspnoea. Nor is heart failure encountered. The pulse throughout is often quite regular and infrequent in comparison with the temperature.

*Nephritis* has been present in a large number of cases, and a striking feature has been the absence of oedema, and also of haematuria.

Despite the frequency with which *delirium* has been met we had no single case of meningitis, although the cerebro-spinal fluid was specially investigated in a number of cases.

Two rather peculiar features are worthy of note. In upwards of twenty cases we have seen spontaneous rupture of one or both rectus abdominis muscles, the immediate cause of which appears to have been the effort of coughing, but disease of the muscle must have been present. And in fifteen cases palpation has elicited widespread subcutaneous crackling of the deep tissues of the chest, neck, and back, the result of *subcutaneous emphysema*.

Another distinctive feature has been a characteristic stench which appears to exude from the body as a whole. This stench was remarked upon occasionally in sporadic cases of "purulent bronchitis," and so appears to be specifically related to the infection.

*Treatment.*—Despite the large number of drugs which have been recommended for influenza, our experience based upon a very extensive employment of the orthodox specifics and using a large number of controls, has led to the conclusion that not one has any unquestionable action on the course of the disease. Although common sense naturally dictates the greatest reasonable precaution possible, even for the mildest cases, yet the virulent septicæmic type appears to originate *ab initio*, and to develop in spite of early treatment. But I would add a warning that an attempt to belittle the condition and allow the patient to return to his ordinary duties after a short afebrile period has been poor economy. In very many cases—I am speaking of the present epidemic as distinguished from the cases in June, which took a much milder course—a recurrence has occurred on the third day after the patient has been allowed to rise, unless we have allowed a clear three days from the last pyrexia even in the mildest case to elapse before the patient leaves his bed.

When one turns to consider the virulent case, so called "influenzal pneumonia," I can without hesitation assert that there is hardly any sort of treatment which we have not tried, and that not one
single line of treatment can be credited with any real value. Early in the epidemic we were persuaded that the “blue” cases invariably succumbed. Later we were fortunately able to record that a certain, even if a small, number recovered, and yet among the recoveries were patients whose condition was regarded as so desperate that any special treatment then on trial was not undertaken, since failure in these instances could hardly be a reflection upon the adequacy of the treatment. Venesection, as one might expect, proved useless. Oxygen, even when continuously administered by the Haldane apparatus, appeared to yield no advantage. Subcutaneous saline infusions, with one curious exception, and intravenous infusions of saline and of glucose in varying strengths, were also tried without success. Antipneumococcal and antistreptococcal sera were employed, although not on a very large scale, but nothing resulted to encourage their continued employment. Vaccines have also been employed, but again not on a very large scale. As a consequence of one subcutaneous or rather intramuscular saline injection, suppuration occurred at the site of inoculation, with sloughing and the formation of an abscess, in the pus of which were identified Staphyllococcus aureus and the diplostreptococcus morphologically resembling the organism recovered from the heart’s blood in some of the fatal cases. This patient recovered from an apparently hopeless condition, and the happy accident encouraged us to attempt the production of similar “fixation abscesses” by further intramammary infusions, and later by the injection of turpentine and a live culture of streptococci. But in no other instance did any suggestion of suppuration occur at the site of inoculation. As one would expect, when empyema, that is to say Nature’s fixation abscess, developed, the prognosis was relatively very good.

Prognosis.—In the admittedly serious cases we have found it impossible to establish criteria. The “blue” cases which recovered were quite indistinguishable in any features from those which died. Cases regarded as beyond redemption recovered, encouraging the determination never to abandon hope while there was life, and we have even had the curious experience of thrice regarding as hopeless one man who recovered, relapsed, recovered, relapsed, and then finally recovered. An even more painful indication of one’s prognostic deficiency was afforded by cases which, not only upon admission but even for several days of treatment in hospital, appeared to run a comparatively trivial course, and to give rise to no legitimate anxiety; the patients then suddenly took a turn for the worse, rapidly developed cyanosis, and died within a few hours of being only trivially ill.
Prophylaxis.—The precautions we adopted were a routine gargle of dilute potassium permanganate or tincture of iodine for those in attendance on patients, and for those patients who had had a comparatively mild attack. In addition, all medical officers, nurses, and orderlies were instructed to use a gauze mask around the nose and mouth whenever they were in attendance upon patients. In the large venereal division of the hospital only two or three sporadic cases appeared—these had evidently entered the hospital with the disease; they were immediately segregated, and no instance of infection of other patients occurred. Our views upon the inefficiency of treatment, both as regards the aborting of a mild attack or the prevention of its development into the virulent form, to say nothing of the virulent form itself, may not be supported by others; but they are the expression of an opinion derived from a very considerable experience, and may help at any rate to emphasize the relatively great value of early segregation if more radical prevention cannot be achieved.
The Royal Society of Medicine.

President—Sir Humphry Rolleston, K.C.B., M.D.

Chairman—Sir James Galloway, K.B.E., C.B.

The Management of Venereal Diseases in Egypt during the War.


A brief reference is necessary to the history of the subject in Australia prior to the war. The efforts made in Australia to deal with venereal diseases on a comprehensive scale date from the Dunedin Session of the Australasian Medical Congress (1896) and culminate in the comprehensive investigation made by the Government of Victoria in 1910 and 1911. Particulars are set out in the Proceedings of Congress and in my own work, "The Twin Ideals."

From 1911 to 1914 a good deal of educational work was done. Clinics were provided, but the Government did not move as fast as the Advisory Committee to the Minister of Public Health (of which I was a member) thought necessary. Accordingly a committee of medical men and women was formed and took action on their own responsibility. They drafted the circular which follows and arranged with the leading chemists that prophylactic outfits should be sold and that each should be accompanied by one of these circulars. Before, however, actually taking action the circular was sent to every member of the medical

At a meeting of the Society, held January 20, 1919.
profession and to every minister of religion in the State. They were asked to express approval or disapproval of the step proposed to be taken, but if they disapproved they were asked to indicate a better method of controlling these diseases. All the medical men who replied expressed their approval and of the 800 ministers of religion about sixty replied. Of these a strong minority replied in dignified language expressing approval, but at the same time regret that such a step should be necessary; a majority disapproved, whilst a few were simply abusive.

**Warning! Venereal Diseases.**

Irregular sexual intercourse involves a considerable liability to infection with gonorrhoea, or syphilis, or both. Of those who are infected many are never cured; the consequences are serious so far as they are concerned, and still more serious as regards those to whom they may communicate these diseases later on.

Incontinence is not in any way necessary for purposes of health, and the advice the medical profession would give to all would be abstinence from intercourse other than in marriage, if they wish to have a healthy wife and healthy children. At the same time, if you determine to take the risk it is your duty, not only for your own sake but for that of other people, to avoid infection, and infection can usually be materially reduced in frequency or avoided by following the procedure indicated in the following:—

**Instructions for Men.**—Immediately after intercourse thoroughly wash the genitals with soap and water, pass urine, and then dry the parts, especially the opening of the passage. The washing must include the parts under the foreskin, which must be drawn back. Then dip the wisp of cotton wool into the liquid supplied and gently pass it into the opening by a twisting movement; pass it in for a quarter of an inch and allow it to remain in position for three or four minutes before removal. If the opening or "eye" of the penis is so small that this cannot be done, a few drops should be inserted with a medicine dropper. The ointment is then to be used by rubbing it in thoroughly and firmly over the whole penis, but more particularly the head, especially if a crack, abrasion, or small tear is noticed anywhere. The foreskin must be drawn back to allow of this being done properly. A small portion of the ointment should be allowed to remain in the opening of the canal. The value of these simple precautions depends on the rapidity with which they are adopted after intercourse. The sooner these steps are taken the less is the risk of infection. The applications should be made in the order indicated. The longer they are deferred the greater the risk.

**Instructions for Women.**—If there has been any risk of infection, as soon as possible after connexion douche well, using a quart of hot water and putting
into it two teaspoonfuls of tincture of iodine. Then smear ointment calomel round the outside of orifice and well up over the front.

The ointment, 30 per cent. calomel. The solution, 20 per cent. argyrol. (This may be prepared in the form of a jelly with glyco-gelatine.)

A deputation from the Council of Churches met the Medical Committee and a frank discussion ensued. The venerable President stated that he did not approve of the method proposed but that he had no alternative to suggest. The Committee asked him whether the Churches felt any responsibility for the wholesale infection of the innocent which goes on at present and whether he could withhold assent from measures which would save them. Finally, the Committee invited the Council of Churches to visit the asylums, the women’s hospital and the venereal clinic, to examine the facts and meet again. At this juncture and in the frame of mind indicated I left for Egypt on the outbreak of war.

On the way to Egypt, by direction, I gave systematic lectures to the 800 men on the S.S. Kyarra on the natural history of these diseases and on the modes of prevention and treatment. The attitude adopted was that now familiar—i.e., urging abstinence, but in its failure precautions against infection.

On arrival in Egypt in January, 1914, I found that the military authorities were much perturbed by the amount of venereal disease amongst the troops, and after consultation General Sir William Birdwood entrusted me with the organization of a moral and military campaign, so far as the Australian troops were concerned. He informed me, however, that he was unable to authorize the use of prophylactics and that any medical officer who adopted that policy must do so entirely on his own responsibility. I accordingly visited all troops arriving from Australia and handed to the officers the circular which follows:

For Private Circulation only.

DIVISIONAL HEADQUARTERS,
Mena, December 28, 1914.

The following letter written by Major-General W. R. Birdwood, C.B., C.S.I., C.I.E., D.S.O., Commanding the Australian and New Zealand Army Corps to Major-General W. T. Bridges, C.M.G., Commanding the First Australian Division, has been printed for private circulation.

V. C. M. Sellheim, Colonel, A.A. & Q.M.G.

JA—7a
HEADQUARTERS, AUSTRALIAN AND NEW ZEALAND ARMY CORPS,
Shepheard's Hotel, Cairo, December 27, 1914.

MY DEAR GENERAL,—You will, I know, not misunderstand me if I write to you about the behaviour of a very small proportion of our contingents in Cairo, as I know well that not only you but all your officers and non-commissioned officers and nearly all the men must be of one mind in wishing only for the good name of our contingents.

Sir John Maxwell had to write recently complaining of the drunkenness of some of our men in the Cairo streets. During Christmas time some small licence might perhaps have been anticipated, but that time is now over and I still hear of many cases of drunkenness, and this the men must stop.

I advisedly say "the men must stop," because I feel it is up to the men themselves to put a stop to it by their own good feeling. I wonder if they fully realize that only a few days' sailing from us our fellow-countrymen are fighting for their lives, and fighting as we have never had to do before, simply because they know the very existence of their country is at stake as the result of their efforts.

We have been given some breathing time here by Lord Kitchener for one object, and one object only—to do our best to fit ourselves to join in the struggle to the best advantage of our country. I honestly do not think all of our men realize that this is the case. Cairo is full of temptations, and a few of the men seem to think they have come here for a huge picnic, they have money and wish to get rid of it. The worst of it is that Cairo is full of some, probably, of the most unscrupulous people in the world, who are only too anxious to do all they can to entice our boys into the worst of places, and possibly drug them there, only to turn them out again in a short time to bring disgrace on the rest of us.

Surely the good feeling of the men as a whole must be sufficient to stop this when they realize it. The breathing time we have left us is but a short one, and we want every single minute of it to try and make ourselves efficient. We have to remember, too, that our Governments of the Commonwealth and Dominion have sent us here at a great sacrifice to themselves, and they fully rely on us upholding their good name, and, indeed, doing much more than that, for I know they look to us to prove that these two contingents contain the finest troops in the British Empire (whose deeds are going down in history), whom they look forward to welcome with all honours when we have done our share, and I hope even more than our share, in ensuring victory over a people who would take all we hold dear from us if we do not crush them now.

But there is no possibility whatever of our doing ourselves full justice unless we are, everyone of us, absolutely physically fit, and this no man can possibly be if he allows his body to become sodden with drink or rotten from women, and unless he is doing his best to keep himself efficient he is swindling the Government which has sent him to represent it and fight for it. From
perhaps a selfish point of view, too, but in the interests of our children and children's children it is as necessary to keep a "clean Australia" as a "White Australia."

A very few men can take away our good name. Will you appeal to all to realize what is before us, and from now onwards to keep before them one thought and only one thought until this war is finished with honour, that is, a fixed determination to think of nothing and to work for nothing but their individual efficiency to meet the enemy.

If the men themselves will let any who do not stick to this know what curse they think them in shirking the work for which it has been their privilege to be selected, then I know well any backslidings will stop at once, not from thoughts of punishments, but from good feeling, which is what we want.

I have just been writing to Lord Kitchener, telling him how intensely proud and well-nigh overwhelmed I feel at finding myself in command of such a magnificent body of men as we have here: no man could feel otherwise. He will, I know, follow every movement of ours with unfailing interest, and surely we will never risk disappointing him by allowing a few of our men to give us a bad name. This applies equally to every one of us, from General down to the last joined drummer.

Will you and your men see to it?

Yours very sincerely,

W. R. BIRDWOOD.

The matter was fully discussed with the officers and then each soldier was given the leaflet which is now set out:

**WARNING TO SOLDIERS RESPECTING VENEREAL DISEASE.**

Venereal diseases are very prevalent in Egypt. They are already responsible for a material lessening of the efficiency of the Australasian Imperial Forces, since those who are severely infected are no longer fit to serve. A considerable number of soldiers so infected are now being returned to Australia invalided, and in disgrace. One death from syphilis has already occurred.

Intercourse with public women is almost certain to be followed by disaster. The soldier is therefore asked to consider the matter from several points of view. In the first place, if he is infected he will not be efficient and he may be discharged. But the evil does not cease even with the termination of his military career, for he is liable to infect his future wife and children.

Soldiers are also urged to abstain from the consumption of any native alcoholic beverage offered to them for sale. These beverages are nearly always adulterated, and it is said that the mixture offered for sale is often composed of pure alcohol and other ingredients, including urine, and certainly produces serious consequences to those who consume it. As these drinks are drugged,
a very small amount is sufficient to make a man absolutely irresponsible for his actions.

The General commanding the Australasian Forces, therefore, asks each soldier to realize that on him rests the reputation of the Australasian Force, and he is urged at all costs and hazards to avoid the risk of contracting venereal disease or disgracing himself by drink.

Consequently every officer and every man arriving in Egypt was fully and adequately warned and a proper appeal made for his help. In my own unit, however, the First Australian General Hospital, by permission of the Officer Commanding, the practice begun on the voyage was continued—viz., education. In addition, prophylactic outfits of calomel ointment and argyrol jelly, together with the Victorian circular, were made available for anyone who wanted them, with the intimation that whilst I should be better pleased if they were not required, still, if exposure was contemplated, they should be used in the interests of the Service. It may be that in other units similar measures were taken at that time, but of such action I have no first-hand knowledge.

What was the result?

Before the troops left for the Dardanelles 800 to 1,000 men were known to be suffering from venereal disease at any one time—i.e., 2.5 per cent. to 3 per cent. of the force. The average stay in hospital of a venereal case was sixteen days, so that the number of infections was large. In addition, 1,344 men were sent back to Australia and 450 were sent to Malta. The cost of sending a soldier to Egypt and back to Australia, together with his equipment and pay, has been estimated at £300. If so, the cost of these cases to the State must have been in the vicinity of £500,000, without any commensurate return.

At this juncture a medical officer in conference challenged our estimate of the value of prophylaxis, and a test examination was immediately made of all the men in our unit who had been supplied with outfits whenever they wanted them. Only one man had been infected and he had not used the outfit. The number of men examined was under 200.

The number of infected was so large and the damage so great that renewed efforts were made and the moral campaign was pushed harder than ever. By arrangement, the Y.M.C.A. opened the now celebrated Esbekieh Soldiers' Club, the Central at Alexandria, and many others.
We recognized the fact that men cannot be made moral against their will but they can be given a reasonable and healthy alternative. A full description of this splendid work on the part of the Y.M.C.A. will be found in the book published by Lieutenant Deane and myself, entitled "The Australian Army Medical Corps in Egypt," and in a forthcoming work of my own, "The Y.M.C.A. in Egypt." Furthermore, as time passed by, the men were treated to personal exhortations and were given printed documents such as "Women: a Word to Men" (Y.M.C.A.), "Consequences" (Y.M.C.A.), "A Warning to Men going Abroad" (The Association for Moral and Social Hygiene). Exactly when these papers commenced to be given I cannot say, but exhortations of the kind were more or less continuous.

During the latter half of 1915 I ceased to be in charge of the work and in November was invalided to England. I am inclined to think that for some time there was an interregnum in which no one pushed the work.

**The Result.**

The result was that an intense moral and restrictive campaign, together with constructive work in the establishment of magnificent soldiers' clubs (staffed by ladies in some instances), had produced a moderate result. It had secured tolerable decency; there were no obvious orgies, but there was far too much venereal disease. On the other hand, where prophylaxis had been properly taught and applied the diseases were practically non-existent.

At the end of 1915 the men returned from Gallipoli, and later moved to France, and during this period infections became more numerous. From January to May, 1916—i.e., five months—there were no less than 10,000 known cases in Egypt. The matter was regarded as so serious that a combined civilian and military committee was appointed to investigate and recommend. This committee, the Cairo Purification Committee, appointed sub-committees in other cities and took extensive evidence from medical officers and others. The conclusions have never been formally published, but in practice may be expressed as follows:

1. **Rigorous repression of public indecency, unnatural offences, the suppression of pimps, the control of advertisements, and the restriction of the sale of alcohol.**
2. **Continuance of the system of medical examination of prostitutes.**
(3) Provision of ablution rooms and the supply of prophylactics.
(4) Moral and hygienic lectures, guides to meet trains and the like.

These measures were adopted, and by the middle of 1917 the diseases were again under control, and the second phase of the Egyptian V.D. campaign was closed.

Again attention became less acute, the advance into Palestine took place, and another outbreak of venereal disease occurred, and another inquiry was held, this time by one combatant officer. After a full investigation a combined campaign was again instituted and conducted with vigour. The chaplains did their best on the purely moral side. The medical officers delivered lectures and were allowed to provide ablution rooms. For the third time the disease was checked and held. The lesson to be learnt, however, is that it would have been better to place the matter throughout in the hands of one competent senior officer, who should have made himself acquainted with every detail and every phase of the problem; his advice would have been indeed valuable.

Whilst these events were taking place throughout Egypt, a remarkable development had occurred at Port Said, of which the following is a summarized account:—

Lieutenant-Colonel Elgood, C.M.G., was appointed Base Commandant at Port Said at the outbreak of war, and has occupied that difficult and important post ever since. He states that before taking office he had no experience of the management of venereal disease and suddenly found himself in an uncharted sea of trouble. He had to deal with a city of mixed and varying nationality, with multifarious Consuls and with Consular law, with a centre of the white slave traffic, and with very large numbers of troops and sailors of all nationalities, European and Eastern. Martial law was proclaimed in November, 1914. He did his best, and the activities may be summarized as follows: The suppression of the white slave traffic; the closing of the brothels in the European quarter; the segregation of the women who would not leave Port Said in the Arab quarter, where they were placed under the control of the medical officers of the Egyptian Public Health Department, and were systematically examined. His realization from experience that the results of such medical examinations are misleading and dangerous; the formation of a sub-committee of the Cairo Purification Committee at Port Said to deal with the local aspect of the problem.
and to assist in the development of healthy social agencies for the benefit of the men; the punishment of pimps and touts; the restriction of the sale of alcohol to the hours of 1 to 3 p.m. and 6 to 9 p.m., and the difficulty of enforcing this order. In spite of all these activities he gradually realized that sexual intercourse and venereal disease continued on a large scale and he then followed the conclusion of the Cairo Committee and authorized the ablution tent plan. The result was that of 9,282 men who passed through the local rest camp, 4,580 reported exposure, and there were only thirteen infections.

The measures adopted in the ablution tent were:

(1) Washing with a solution of perchloride of mercury 1 in 1,000.
(2) Irrigation of the anterior urethra with a solution of pot. permang. (1 in 3,000).
(3) Application of calomel ointment and the use of bandages to avoid soiling the clothes.

Such in outline is the history of this remarkable effort, the results of which agree with those obtained elsewhere in Egypt. When Lieutenant-Colonel Elgood introduced prophylaxis he did not in any way relax any of the other precautions.

The lesson taught by the experience in Egypt is that all the repressive measures, all the constructive social measures, all the educational efforts, and all the emotional appeals result in only a limited amount of success, and only reduce venereal diseases to a moderate extent. It is evident that a very large number of men either find the sexual appetite overpowering or deliberately indulge, and unless some form of prophylaxis is adopted many infections are certain. Some of the men were quite candid, and stated they intended to indulge despite generals, doctors and chaplains, and with or without prophylaxis, though they preferred to be safe. The conclusions reached by me were that primary prophylaxis and ablution tents are both quite effective in preventing infection.

The objections raised by some people to primary prophylaxis are both moral and medical. The moralists say that the free use of primary prophylaxis would induce people to become more immoral. I am unable to follow this line of reasoning. The use of prophylactics at the time of exposure, say 9.30 p.m., cannot produce a very different result from the use of prophylactics half an hour later in the ablution tent. Medically the position as I understand it is as follows: The civil authorities in Great Britain contemplate establishing treatment centres
Barrett: *Venereal Diseases in Egypt during the War*

throughout the country. These centres will subserve treatment, prophylaxis by ablation rooms, and educational propaganda. If all these activities are carried out, nothing but good can result; but I still think many cases will arise owing to geographical difficulties in which primary prophylaxis will be requisite. If these centres are to issue the outfits, so much the better: if not, it is certain someone else will provide them.

The aim of all medical men, I submit, is not simply the reduction of the amount of venereal disease but the removal from this globe of the spirochāte and the gonococcus, a task which does not seem to me more difficult than the destruction of other noxious animals and plants.

So much is said from time to time by the so-called moralists that I venture briefly to review the problem. Medical men dislike vice rather more than any other class of people because they see it on the nauseous side. They are not, however, as medical men, professors of morality. Their business is to prevent or cure disease, and they have a right to object to their action being hampered by those who seem to think that venereal diseases were created to enforce chastity. For the benefit of those interested, may I state the case in the form of a number of questions:—

1. Is there any material difference from the moral point of view in preventing venereal disease by the use of measures adopted before, at the time of, or after exposure? He who exposes himself knows that in any event he is practically safe.

2. If morality is secured by fear of infection, why not forbid treatment altogether?

3. Is a morality which depends on fear of immediate consequences worth very much?

4. Is not the fundamental ethical fact which concerns us the wholesale infection of the innocent which goes on at present?

5. Is it not a fact that the *Lex talionis* cannot be enforced solely against the transgressor but is enforced with terrible severity amongst the innocent?

6. Finally, why not frankly recognize the fact that the world will not be rendered more or less moral by the extinction of venereal disease?

7. In practice does fear of infection deter many men?

8. If, as Mr. V. Warren Low put it, we discover to-morrow,
as we may, a vaccine which will protect those vaccinated completely against venereal disease, will anyone forbid its use in the interests of morality?

To me, the problem of chastity involves a much wider survey. Here we have a physiological instinct of great intensity round which has been built up the finer feelings which we value more than anything else in life. No sane man wishes to see the animal instinct divorced from these emotions, but he cannot fail to see that if the legitimate gratification in marriage is postponed to the summer of life irregular relationships are certain to be formed. And if you ask why should men and women marry so late in life, the answer is the high standard of external requirements set and, as a valued friend observed, still more largely the artificial assessment of those values. The matter lies largely in the hands of women. If they revise their standards of value most of the sexual difficulties will disappear. But any revision requires a complete knowledge of the fundamental data, a clear and sane vision, and good judgment. Until social arrangements are recast, irregular sexual relationships are likely to continue and whilst they do prophylactics are necessary for bodily salvation. To me, much of the value of the knowledge gained in Egypt was contact with the facts of the case, since we had to deal with an organized commercial system of vice provided with agents catering for the gratification by healthy men of this powerful instinct. We found that we could not succeed by social and moral means alone and that prophylaxis was an absolute necessity if the men were to be kept healthy. Without recasting social relationships I see no present prospect of sweeping away the whole of this hideous paraphernalia.
The condition known as "traumatic," "surgical" or "wound" shock, although well-recognized, is difficult to define. It may be said to be a state of general collapse, associated with low blood-pressure, and the various phenomena dependent on this low pressure, such as coldness, pallor, sweating and so on. Perhaps the most serious consequences of the deficient circulation are those due to the deprival of the necessary supply of oxygen.

The remarks that follow refer to what is usually known as "secondary shock," since it is this that demands practical interference. The sudden "primary" shock that occurs on the receipt of an injury is analogous to fainting and is doubtless of nervous origin. It has its interest and importance and will probably receive attention in the course of the discussion. While, indeed, nervous factors are not dealt with in my short account, their possible co-operation in the production of the vascular phenomena at the basis of secondary shock has to be kept in mind.

It would be unprofitable in the brief time available to discuss those causes which have been found, on investigation, not to be the agents responsible for the state. It will suffice to mention them: arterial or venous dilatation, heart failure, acidosis, vasomotor paralysis, suprarenal exhaustion.

1 At a meeting of the Society, held January 23, 1919.
The low blood-pressure is, in practice, the most important feature of the condition, since it gives a direct indication for treatment. Indeed one may say in general that, both experimentally and clinically, if the blood-pressure is restored the other symptoms disappear, some with rapidity, others more slowly.

In the course of investigation it has gradually come to be realized that the chief, if not the only really important, factor is a deficiency in the volume of blood in circulation. This applies not only to that form of shock brought about by actual loss of blood out of the blood-vessels, but also to cases where there is no reason to suppose that there has been any great loss of blood. Apart from the fact that the general symptoms of hæmorrhage and shock are very similar, estimations by the "vital red" method of the volume of blood in circulation has shown that it is decreased in both cases. The name "exanmia" has been proposed by Major Cannon for the general condition.

It is clear that blood must be accumulated or held up somewhere or other in dilated regions of the vascular system. Observations made in the course of abdominal operations give no support to the view that there is any significant degree of dilatation of the arteries or veins of the splanchnic area. The region in question must therefore be that of the capillaries. Observations by Major Cannon had already suggested that there is stasis here in wound-shock.

But what is it that brings about such a dilatation of the capillaries? At an early date in the discussion of the problem, it was pointed out by General Cuthbert Wallace that operations involving injury of large masses of muscular tissue were especially liable to produce shock. Experiments by Major Cannon and myself in the beginning of last year showed that crushing the thigh muscles of cats was followed by a progressive fall of blood-pressure and other signs of shock. We found that section of the spinal cord above the origin of the limb nerves did not prevent the result, whereas clamping of the artery and vein did do so. Some chemical product of the tissue injury must then be the responsible agent. This view was confirmed more especially by Major McNee's observations on wounded men in a state of shock, where it was found that excision of the injured parts, or even preventing by a tourniquet blood passing through them from entering the general circulation, was followed by marked improvement.

We thought at first that the toxic agent might be lactic acid, but experiment showed injection of this or other acid to be innocuous. In fact, "acidosis" turned out to be a negligible factor in the causation
of shock. It is the result, not the cause of the low blood-pressure. The real agent is a much more toxic one. Dale and Laidlaw have described the action of a base, histamine, which has the remarkable effect of powerfully dilating the capillaries, but not the arterioles, and which in larger doses produces a condition of profound shock. There is reason for believing that substances of this kind are produced in injured and disintegrating tissues.

It was found further that massage of the injured muscles in the experiments on cats resulted in accelerated fall of blood-pressure, so that we have an experimental justification for keeping injured parts as immobile as possible, as is done by the Thomas's splint in the case of fractured femur.

These injury effects are, of course, intensified by causes tending to depress the circulation, such as cold, anxiety, fatigue, thirst, haemorrhage and so on.

The indications for treatment seem obvious. The volume of the blood in circulation must be increased. While transfusion of blood itself is the natural means, it is clear that if a satisfactory substitute, in the form of an artificial solution, could be made, it would frequently be of value.

No simple saline solution, whether iso- or hypertonic, or containing sodium bicarbonate or calcium salt, has any permanent effect. The liquid introduced does not remain in the blood-vessels for more than half an hour or so. To insure that it shall not leave the circulation, the addition of a colloid is necessary. The function of this is to attract water by its osmotic pressure and so to counteract the filtration produced by the blood-pressure.

Of those colloids admissible, gum arabic is, on the whole, the most satisfactory. To correspond with the proteins of the blood plasma, a 6 or 7 per cent. solution in 0.9 per cent. sodium chloride is correct. Such a solution has been found in practice to serve as well as one containing bicarbonate, or excess of calcium.

The use of vaso-constrictor drugs is to be deprecated.

It has been found that the reserve of haemoglobin is sufficiently great for recovery to take place even when it is reduced to a quarter of its normal value. Hence, it would seem that gum-saline should be effective in the majority of cases. A reduction of the haemoglobin to the extent mentioned implies a loss of blood of nearly four litres in man. It has not yet been possible to state definitely what are the clinical signs showing the necessity for blood transfusion, apart from
actual determinations of the blood volume and haemoglobin percentage. Some surgeons state that no case failing to react to gum is amenable to blood.

If, however, the delay between injury and treatment has been great, no measures avail. The tissues, especially those of the nerve centres, as shown by Colonel Mott, become so far damaged by the low blood-pressure and want of oxygen that recovery is impossible.

In actual practice, the following general procedure may be recommended:—

If the patient is cold and fatigued by a long journey, try first the ordinary measures of resuscitation, such as warmth, water to drink, and rest.

If little or no improvement in half an hour or so, 750 c.c. of warmed gum-saline, slowly into a vein.

This may probably be insufficient in amount. Therefore—

If some benefit in half an hour, more gum-saline.

If no result at all from the first injection, transfusion of blood, if available.

But the evidence on the whole suggests that if gum-saline is ineffective, blood will be also useless, except in the rare cases where the loss of blood has been more than 75 per cent. of the total volume.

By Dr. H. H. Dale, F.R.S.

Professor Bayliss has put before you in brief outline the conclusions of the Special Investigation Committee, appointed by the Medical Research Committee to co-ordinate work on “Shock and Allied Conditions” during the war. It will probably be of assistance to those who have not been hitherto in touch with the Committee’s work, if I briefly trace its connexion with the investigations and speculations which led to the views current before the war.

We need not spend more than a moment over the period before the days of modern surgery. No difference was then recognized, of course, between the conditions which Crile and those who follow him have distinguished as “collapse” and “shock,” and which surgeons working in France during the war have taught us to call “primary” and “secondary” shock. Goltz, in 1870, gave his classical demonstration of “shock” in the frog, as a phenomenon of reflex inhibition. A blow on the exposed mesentery of the suspended animal caused arrest of the
heart through the vagus, and loss of arterial tone, so that the blood tended to accumulate by gravity in the splanchnic area. I do not suppose that any better explanation has yet been given of the mechanism of fainting, or of the condition distinguishable from fainting only by greater severity and duration, which is variously called “collapse” or “primary shock”; a condition setting in rapidly after severe injury, pain or emotion.

The condition of “secondary shock,” with which investigations in recent years have been almost exclusively concerned, was not readily distinguished from the “primary shock” or “collapse.” Superficially they are very similar, and, as surgeons working at the Front in recent years have pointed out, may run into one another, so that the secondary appears to be an extension or a more gradual recurrence of the primary condition. It was natural, then, that the earlier writers should regard the whole sequence as a single phenomenon, and should regard reflex depression of the heart’s activity as a central feature of shock in general. Attention was first diverted from the heart to the rest of the circulatory apparatus, in the search for a cause of secondary shock, by the publication of Crile’s first series of experiments in 1899. Crile showed that the small output of the heart in this condition was not due to primary weakening of its activity, but to deficient return of blood to its chambers. He likened the condition to that seen in haemorrhage—a comparison the justice of which all later work has emphasized. Crile regarded this defective diastolic filling of the heart as due to fatigue of the vasomotor centre by excessive sensory stimulation. There would ensue, he supposed, a gradual failure of arterial tone, with a resulting decline of arterial pressure. So far the conception is easy to follow. But Crile went on to maintain that this lowering of arterial pressure would cause the blood to accumulate in the great veins; the patient would “bleed into his own veins” and the output of the heart would fail as in actual haemorrhage. Though Dr. Crile’s eager interest in anything pertaining to his subject has caused his theory to put forth sprouts and tendrils in various directions, its central stock still remains, I believe, this conception of venous stasis as the result of arterial dilatation.

It has seemed to many that Crile and his followers have failed to show ground for the belief that arterial relaxation would have this result. Experimentally it has not. If the arteries are kept dilated, by a drug relaxing their muscle coats without weakening the heart, the arterial pressure can be held down to 40 or 50 mm. of mercury for
prolonged periods, without causing accumulation of blood in the veins, impairing the heart's output, or producing that retarded or inefficient state of circulation through the periphery, which is the characteristic of secondary shock.

Recent developments make it of interest to note that in the year which saw the appearance of Crile's first essay on shock, 1899, a very similar conclusion had been reached by Romberg and Pässler as to the cause of circulatory depression in bacterial infections. Bacterial toxemia, according to these authors, depressed the circulation not by poisoning the heart but by causing failure of vasomotor control and consequent loss of arterial tone. I think we have good reason now for regarding this idea of the effect of toxemia as erroneous, and Crile's conception of shock as vasomotor exhaustion had been challenged from various quarters before the outbreak of the war. J. D. Malcolm was apparently the first to state definitely, in 1905, that the arteries in clinical shock were constricted instead of being atonic. He also first brought into view, as an important factor in shock, the increased proportion of red corpuscles in the blood, which Sherrington and Copeman had observed experimentally many years previously, and had attributed to loss of plasma from the blood. Malcolm also attributed it to a loss of plasma into the tissues, and saw the importance of the implied reduction in the volume of the blood. The data at his disposal did not enable him to account satisfactorily for its occurrence, or to reconcile the association of general arterial constriction with a low arterial pressure in such a manner as to carry general conviction. The accuracy of his two main contentions, however, that the arteries are constricted rather than dilated in shock, and the blood concentrated and reduced in volume, have received repeated confirmation from experimental observers in America, such as Seelig and Lyon, Yandell Henderson and Mann, and, during the war, from surgeons and pathologists in France with whom the Committee have been in contact.

This was the position when the Shock Committee began their investigation. There were many who, following Crile, still maintained that lack of arterial tone was the central characteristic of shock. Lockhart Mummery, for example, still maintained that the arteries were dilated, the blood diluted, and the veins full. The Committee, however, as the result of direct questions put to surgeons at the Front, early came to the conclusion that the type of shock seen in clearing stations, which they were specially called upon to investigate, corresponded with
that described by Malcolm, Henderson, Mann and others, in which the low arterial pressure was not due either to arterial relaxation or to weakness of the heart's action, but to oligæmia—deficient volume of the blood in effective currency. At the same time they were bound to realize than no satisfactory explanation was yet available of the cause of this condition. It was a paradoxical complex from many points of view. Arterial constriction normally results in high, not low, arterial pressure; low arterial pressure normally causes dilution, not concentration of the blood. Further, the concentration was often obvious enough, but the apparent defect of blood volume was serious out of proportion to the loss of plasma which the concentration indicated.

I need not spend much time on the lines of inquiry which the Committee followed without result. I suppose it was the grip of tradition which led us long to search in vain for a nervous mechanism by which such a condition could be produced. Our failure does not, of course, warrant the conclusion that nervous effects never play any part in the phenomena.

Acidosis was next brought prominently to our notice as a suggested cause of this curious complex, and those who are interested can read the record of its trial and unanimous dismissal in the Report (No. 7) recently issued by the Committee. Meanwhile a clue of more value was lying in our hands. Professor Bayliss has alluded to the analogy provided by the action of "histamine." Dr. Laidlaw and I had been working at the action of this substance at intervals since 1910. Its action had interested us not merely for its own sake, but because it represented a type of activity common to a large class of protein cleavage-products, formed by partial digestion or bacterial action. Products showing this type of action have been extracted from almost all organs of the body; they are naturally present in great abundance in the contents of the small intestine; and they appear to be set free readily from almost any tissue as the result of injury or even of temporary stoppage of the circulation. We had realized some time previously that the action of histamine presented the same paradoxical features as had been met in the condition of secondary shock. Here was a substance which caused contraction of all plain muscle, including that of the arteries, which yet produced a fall of arterial pressure, not by weakening the heart but by vasodilatation. In larger doses it caused a profound shock-like collapse, of which the central feature was oligæmia. The heart continued to beat vigorously, but was almost empty of blood, as were the arteries and the great veins. Loss of
plasma into the tissues was indicated by rapid rise in the corpuscular content of the blood, but seldom to such an extent as to account for the apparent loss of volume. Somehow blood had passed out of regular currency through the heart and great vessels, and through a large part of the tissues the circulation was slowed almost to stagnation. A process of exclusion led us to locate the blood in the capillaries, and to conceive the action of histamine as due to a general loss of the normal capillary tone. We supposed that under normal conditions only a limited portion of the capillary network is serving at any one moment as a path for the blood, and that the simultaneous opening up of all the potential channels would lead the blood to percolate slowly through the tissues, which would soak it up like a sponge. The outflow into the veins, and therewith the filling and output of the heart, would fall rapidly away; the blood would accumulate more and more at the periphery, yet with so uniform a distribution that there would be no local hyperemia, but merely the slight general bluish congestion of internal organs, and the dusky pallor of the skin and mucous membranes, which is seen in the animal suffering from shock produced by histamine. Major Cannon, observing the condition of men suffering from shock at the Front, and meeting with the same problem of discovering the whereabouts of the blood which had disappeared from the heart and great vessels was driven by a similar process of exclusion to locate it, as we had done, in the generally relaxed capillaries. Later an investigation in which I had the privilege of co-operating with Professor A. N. Richards, of Philadelphia, provided direct evidence in favour of the view that histamine acts by relaxing capillary tone.

Note how such a conception of the action, as a poisoning of capillary endothelium, accounts not only for the accumulation and stagnation of blood at the periphery, but also for the anomalous association of loss of plasma with low blood-pressure. It will hardly have escaped your notice that this association of capillary dilatation with transudation of plasma, represents a generalization throughout the body of a process which, if locally limited, we should recognize as mild inflammation. The fact that a bacterial toxin, causing inflammation when locally limited, would cause intense prostration and shock-like failure of the circulation if it spread rapidly through the system, was already well known. Would not the same hold good for the products liberated from injured tissues, apart from bacterial action? Professor Bayliss has told you how this possibility was brought under investigation, and has shown how results of experiment and clinical observation, by Major Cannon
and himself, by General Wallace, Major McNee and others, have all pointed to the conclusion that such a process plays a part in shock. I may add that some of our French colleagues have independently struck the same trail and reached similar conclusions. Professor Bayliss has also dealt fully with the therapeutic measures which this conception of shock suggests.

I am sure Professor Bayliss will agree with me when I say that the Committee we represent makes no claim to have given a general or complete explanation of shock. The definition of shock is by symptoms rather than causation, and I imagine the term is applied to any condition of circulatory failure for which no adequate cause, such as hemorrhage or infection, can be found. Our Committee's claim is merely to have drawn attention to another such recognizable cause, which we might call "traumatic toxemia." There may be many others yet to be investigated. It is to be hoped, however, that the subsequent course of this discussion will show how far this particular conception is likely to prove helpful in elucidating the nature of the condition of shock as it occurs in ordinary civilian surgical practice; and how far the suggestions for prevention and treatment, which it has given to the military surgeons, are applicable to the conditions of peace. Fortunately there are those present who have had large experience of surgery both in war and in peace.

Mr. J. D. Malcolm.

I should like to thank Dr. Dale for the very kind way in which he has mentioned my views on shock. He dates these from 1905, but it was in 1893 that I first expressed the belief that the vessels contract in this condition, with the corollary that there must necessarily be a diminution of the blood in them. At that time it was also pointed out that certain poisons circulating in the blood create a widespread irritation of tissue, with contraction of the vessels, and it was suggested as "conceivable that death may be produced almost with the suddenness of shock" by such poisons. My contention is that, whether caused by an irritant circulating in the tissues or by direct stimulation of sensory nerves in the course of an injury, the vascular changes-

2 Loc. cit., p. 204.
3 Loc. cit., p. 211.
characteristic of shock are brought about reflexly through the nervous system.

The following observations are founded upon a study of surgical operations in civil practice unaccompanied by crushing of tissues or other cause of toxæmia, except carbolic acid used as a lotion in my earlier cases and the anaesthetic which, if unskilfully given, may bring about a condition undistinguishable from that of shock before an operation is begun. Shock, in its simplest form, is a state produced in a healthy man by a severe but otherwise uncomplicated operation or injury. If it does not kill it leaves no permanent effects, and is recovered from completely soon after the injurious influences cease to act. No permanent injury is a part of surgical shock. Such conditions as hæmorrhage, exposure to cold, septic infection, massive injury of tissues, starvation, thirst and disturbance of injured parts are complications, each of which aggravates all the others, and some of them, acting alone, may induce changes closely resembling or identical with those of shock. But conditions having only a resemblance to that state should be carefully distinguished. The effects of hæmorrhage, shell shock, and spinal shock are altogether different from those of surgical shock. Sometimes shell shock includes surgical shock, but its characteristic features—namely, hæmorrhages into brain and cord and prolonged functional disorders—are not found in surgical shock.

Stimulation of sensory nerves brings about a contraction of the blood-vessels throughout the body, and the simplest as well as the most complete explanation of the phenomena of uncomplicated surgical shock is found in the view that its primary change is a reflex contraction of the blood-vessels, proportionate in degree to the intensity of the irritation of sensory nerves. This contraction begins in the small arteries, capillaries, and veins, from which it may extend until the whole vascular system, including the heart, is tensely contracted. It follows that every operation and every injury causes the beginnings of the state of surgical shock even although there may be no indications of any such condition observable clinically.

A profound degree of shock may be brought about instantaneously, too quickly it seems to me to be explained as other than of nervous origin, for example when a limb is crushed, or it may be produced gradually in the course of a prolonged severe operation. It is only when slowly developing that the changes can be defined. During an operation the radial pulse may for a time show no diminution in volume or increase of pace. As long as the smaller vessels only are contracted,
the amount of blood in such medium-sized vessels as the radial may be increased, whilst the rate of the heart action may even become slower. If the stimulation of sensory nerves is sufficiently prolonged and severe the medium-sized arteries contract also and the radial pulse may be observed becoming gradually smaller until it is impalpable. As the pulse becomes smaller the heart action usually hastens, but occasionally the pulse may completely disappear from the wrist and from all the distal vessels while the heart-beats remain slow. In such cases the heart-sounds are loud and distinct and the carotid arteries may be felt beating forcibly. These pulse conditions are not very common and are easily overlooked because they cause no sign of danger beyond an extreme pallor, but if diligently sought for by examining patients who show no alarming symptoms they will be found from time to time, and a single case of this kind, if carefully observed, leaves no way of escape from the conviction that as shock slowly develops the peripheral vessels forcibly contract. In the state of profound shock, whether it is induced suddenly or gradually, the heart always beats rapidly and the pulse may be imperceptible everywhere, so that only by the use of a stethoscope is it possible to recognize that the heart is beating. Like the rest of the vascular system the heart then works with a narrower gauge and a smaller range of relaxation. An intense arterial and cardiac contraction, amounting in the heart almost to a condition of tetanic spasm, was described by Eugene Boise\textsuperscript{1} as the essential feature of the state of shock. That this occurs in a well-developed case seems to me beyond question, but those conditions in which a complete absence of the radial pulse is gradually brought about in association with a slow powerful heart action show that an intense vascular contraction may take place in spite of an unimpaired propelling force, and that the vascular contraction really precedes the hastening and limitation of the heart movements, although the pulse and heart changes usually develop together. The hastened heart action in the state of developing shock is in part at least, due to a relative cardiac weakness created by the greater resistance to the blood flow brought about by the narrowing of the arteries. The small output of the heart is generally attributed to a deficient return of blood from the veins—a view passed on from one man to another without any very obvious proof. Crile declared that the blood-pressure in one large vein—the splenic—was raised as shock developed, which is not consistent with a deficient supply of

\textsuperscript{1} Trans. Amer. Assoc. of Obstet. and Gynecol., 1893, vi, p. 129.
blood in the vena cava. As long as the heart action remains slow, if the cause of trouble is arrested there is no danger, and it is generally agreed that, in a healthy individual, death from shock does not begin at the heart.

The conception of shock as arising primarily from a reflex vascular contraction, extending from the periphery to the centre, fully explains every change that is known to take place in that condition. When the vessels become generally contracted the cubic capacity of the vascular system must necessarily be reduced and therefore the quantity of blood in the vessels must be diminished.

Recently the Medical Research Committee has shown that the number of red corpuscles in the blood is increased, sometimes by as much as a third of the normal, in the state of shock, showing clearly that the plasma, or perhaps only some portion of the plasma, is the first part of the blood to escape from the vessels.

The fluid which leaves the vascular system has not been discovered clinically in any part of the body, but one of the most pronounced and constant features in a case of severe shock is a profuse sweating. The bed upon which the patient lies is often soaked with perspiration over an area extending to three or four inches from the body all round it, and when the face is dried it immediately becomes wet again. It is certain that much fluid escapes, not only from the vessels but also from the body. The bloodless condition of the skin does not interfere with the action of the sweat glands, which may be brought about in the foot of a dead cat.

In the case of the kidney the quantity of its secretion depends in great measure upon the blood-pressure in the renal arteries, and as these contract with the other vessels the anuria of shock is fully explained.

The amount of the intestinal secretions is not easily observed, but when the blood becomes greatly concentrated and contains an unusually large number of blood corpuscles an excessive destruction of these cells is to be expected and a common symptom of shock is vomiting as recovery takes place. The vomit consists mainly of bile which is often of dark colour, and this indicates that blood cells are being destroyed in unusual numbers. This sign cannot become very conspicuous because death or recovery is never long delayed in an uncomplicated case of shock. It is said that the rise in the corpuscular content of the blood is not sufficient to indicate the apparent loss of volume, but a rapid destruction of red cells does not seem to have been allowed for.
The tensely contracted condition of the vessels fully accounts for the blanched appearance throughout the body in a case of well-developed shock. The tissues are for the time to a great extent deprived of blood, and the consequent want of nourishment offers a complete explanation of the inert, collapsed condition of the patient, the fall in his temperature, and his death if death occurs. The starvation of tissue is also a sufficient cause of the acidosis of shock. As numerous forms of death depend eventually upon a slow starvation of the tissues, it seems likely that many modes of dying will be found to be associated with an acidosis.

The view that the blood-vessels contract tensely in a well-developed case of shock has been considered irreconcilable with the great fall of blood-pressure which is characteristic of that state. It was formerly argued that the blood was incompressible, and that contraction of the vessels necessarily increased blood-pressure whilst relaxation as certainly lowered blood-pressure. Now a large loss of fluid from the vascular system and a corresponding contraction of the vessels are recognized conditions of shock. One tends to lower whilst the other tends to raise blood-pressure, and there is no contradiction at all in the idea that a fall of blood-pressure may be induced as the ultimate effect of vascular contraction, and especially of the stage of vascular relaxation when the effect of an irritation ceases.

The reduction of the amount of fluid in the vessels must be due either to a contraction of the vessel which squeezes the more fluid parts of the blood into the tissues or to something which causes a rapid absorption of fluid from the vessels into the tissues, with a consequent contraction of the vessels upon the blood remaining in them. Assuming for a moment that a general contraction of all the small and medium-sized vessels takes place, clearly this must have a limit if the patient recovers, for a complete occlusion of the vessels would certainly cause death. The vessels must remain in their state of greatest contraction at least for a short time, possibly only for a moment. The following changes must therefore occur: (1) There will at first be resistance to the contraction and a rise of blood-pressure. (2) The volume of blood will be reduced in proportion to the degree of vascular contraction, and this will more or less neutralize the rise of blood-pressure. (3) The circulation will be carried on through much narrowed vessels. Every practical plumber knows that in a large tube the pressure of a fluid flowing through it is greater than the pressure in a smaller tube when all other conditions are equal, and therefore the circulation
through vessels of much diminished size must be accompanied by a
lowered blood-pressure as compared with that of normal conditions,
if no other change takes place. Of course other changes do take place,
and in particular the heart strives to keep up the blood-pressure, but if
the vessels are sufficiently small the cardiac reserve power is relatively
slight. (4) The contracted vessels must relax whenever the patient
tends to recover. There then arises a further cause of fall of blood-
pressure due to an increased disproportion between the bulk of the blood
and the capacity of the vascular system. This disproportion continues
until fluids return from the tissues to the blood, or are artificially
introduced.

Changes (1) and (2) correspond to the stage described by the Medical
Research Committee as primary shock, which is characterized by
collapse of the body brought about by an exaggerated contraction of the
vessels and starvation of the tissues. There is no circulatory collapse or
feebleness of any kind. Changes (3) and (4) correspond to the condition
referred to as secondary shock, which is characterized by a diminished
amount of blood and relaxing vessels. My view is that the four changes
are essential parts of one complete process, recovery from which is fairly
rapid in uncomplicated cases when the cause ceases to act. But
exposure, movements of injured parts, hæmorrhage, and septic or
aseptic toxaemias may prolong the stage of recovery indefinitely, or
may induce a secondary shock in the sense of the effects of secondary
stimulations. It is obvious that if a powerful vascular contraction is
immediate and intense and the loss of fluid is sufficiently rapid, the
stage of rise of blood-pressure may be so transient as to be overlooked.

If on the other hand it is assumed that fluid leaves the blood-vessels
on account of some osmotic change, which seems to be the newest view,
whether this occurs slowly or quickly the initial rise of blood-pressure
characteristic of a gradually induced shock cannot be explained.
Therefore unless primary shock and secondary shock are altogether
different conditions they cannot be due primarily to a removal of fluid
in any way comparable to that of hæmorrhage, although the resulting
condition of depleted vessels is similar to that of hæmorrhage.
Moreover no account has been given of any mechanism by which fluid
is drawn out of the vascular system in large quantities. The conclusion
seems inevitable that the fluid is forced out of the vessels by vascular
contraction.

Considerable importance is attached by recent writers to the
observation that the number of red cells is greater in the capillary
blood than in that from the veins. But it is definitely stated that there is in health a slightly greater number of red cells in capillary blood than in venous blood and the increased difference in the state of shock is therefore merely an exaggeration of a normal condition. It seems probable that the narrowing of the capillaries and the escape of fluids from them in health lead to a holding up of the blood cells and it is obvious that an extensive contraction of the arteries and capillaries with an increased escape of fluid from the vessels, must lead to a holding up of the red cells in greater numbers, and therefore a vascular contraction explains the increased difference between the capillary and venous blood.

It is suggested by the Research Committee on Surgical Shock that the "absorption of depressant substances from injured tissues," causes "a general loss of normal capillary tone," and "leads to the accumulation of a large part of the blood in a semi-stagnant condition in the capillaries." The existence of a peripheral relaxation of vessels is constantly referred to, but the evidence in favour of this view does not seem very definite whilst the location of the bulk of the blood in the capillaries seems also to require confirmation. In Report No. 2 of the Committee, p. 75, it is said that "if in wound shock the lost blood is not in the arteries, and probably not to a great extent in the veins, it must be mainly stagnant in the capillaries." But why must it be in the capillaries? Why should it not be mainly out of the vessels altogether? In the same report, shock is described as an exsanguina. Surely if the bulk of the blood were in the capillaries and if the blood contains, as has been shown, a high percentage of red cells, the extreme pallor of all the tissues which is one of the chief characteristics of uncomplicated shock would not be found.

As regards treatment in uncomplicated shock the essential points are to do everything that is possible to induce the vessels to relax and to prevent further stimulation of sensory nerves by warmth, by opiates, by rest, by preventing disturbance of injured parts when a patient must be moved, and by adopting all the known methods to reduce secondary irritation during operations. These methods have been especially worked out by Crile, who properly lays stress on the selection of a suitable anaesthetic.

It seems to be the universal experience of the R.A.M.C. that in conditions of developing shock of a serious type no method of introducing fluid into the body is of any use, and Crile showed experimentally that in such cases saline fluid injected into the veins passed rapidly into the
connective tissues. Here again the facts support the view that when the condition described as primary shock is well developed the vessels are tensely contracted, so tensely that they will not yield to any known method of dilating them forcibly. It seems possible that attempts to do so may be harmful at this time. On the other hand in cases in which haemorrhage is the chief feature the greatest benefit has been derived from filling up the depleted vascular system, and the work of Professor Bayliss with a view to deciding the best composition of the fluid to be injected is of great value. As haemorrhage is one of the most serious complications of shock its treatment is indirectly of importance in combating shock. But it should be recognized that this method is mainly and most directly useful as a treatment for haemorrhage when the state of shock is developing.

As soon as relaxation of the vessels begins in a case of shock, which it naturally does when the primary irritation ceases, there is an urgent need for fluid to fill up the relaxing vessels. Without an introduction of fluid, the blood-pressure will fall still lower, and even greater danger may arise, especially if the patient is moved or if the condition is aggravated by some complication. But fluid is readily absorbed at this time, and it is best introduced subcutaneously. Its introduction in this way may with advantage be arranged even before an operation is begun. Again, of course, if the case is complicated by haemorrhage the treatment for that condition may be necessary.

After an operation recovery is greatly hastened by a continuance of the subcutaneous injection or by an administration of fluid by the rectum or mouth. Before this was known patients, after a severe operation, constantly showed signs of collapse on being moved from the operating table to the ward. The rapid absorption of fluids during recovery from shock, and the difficulty of recovery without an introduction of fluid, indicate clearly that the lost fluid is mainly out of the body altogether, and like every other point that has been considered, strongly support the view that uncomplicated surgical shock is a very simple condition, consisting of the consequences of a reflex contraction of the vessels throughout the body, beginning at the periphery and extending in intensity as well as towards the larger vessels in proportion to the degree of the irritation of sensory nerves until it may involve the whole vascular system.
Major-General Cuthbert S. Wallace, C.B., C.M.G., A.M.S.

About the time when Cowell pointed out that shock often came on some time after a wound and was in many cases preventable, another—namely the septic—conception of shock was coming to the fore. While the late appearance of shock was quite easily accountable on the old theory of exhaustion of the medullary centres by afferent impulses, its tardy appearance offered another possibility. There are some clinical facts that are pertinent to this question.

In the first place, a patient dying of peritonitis presents all the appearances of one dying of shock. Many soldiers with abdominal injuries were said to die of shock, but it was not so much the actual injury that the surgeon feared as the effects of haemorrhage and developing peritonitis. If the case arrived early, very extensive injuries could be recovered from without the patient exhibiting marked symptoms of shock, while late arriving cases died of what was called shock, but the cause of death was really sepsis or sepsis and loss of blood combined. A careful series of post-mortems performed by McNee and Dunn clearly proved that intoxication from gas gangrene was often the cause of death when the clinician had put down the fatal result to shock. Another striking example is furnished by the behaviour of patients obviously affected with gas gangrene. Here a patient presenting the appearances of shock is relieved in a few hours by the amputation of a limb, or still more dramatically by the ablation of an infected muscle. It is certain that patients rapidly improve after the removal of smashed limbs. In fact, such limbs are one of the most potent causes of shock when long ambulance journeys are in question. In one particular case that I remember a patient showed no sign of recovery from shock, although resting quietly in bed, until the foot was removed, when he at once began to improve. It is well known that operations involving large muscle section are accompanied by shock. Multiple wounds were accompanied by a high mortality, and these multiple wounds nearly always concerned a large muscle bulk when all the wounds were taken into account, though possibly no single wound was large.

The toxic theory of shock is a seductive one, but pushed to an extreme it seems to land one in a difficulty. If loss of blood, loss of heat, and toxæmia be excluded, can it be said that shock will be banished? On this point some other clinical details are interesting.

In the war we came across men with all the symptoms of shock
without any external wounds and very slight injuries, as was proved by post-mortem examination. These cases exhibited all the classic symptoms of shock, and the men died in spite of all treatment. In these cases there was apparently a concentration of the blood, but no observations as to the blood volume were possible. It would therefore seem that, in some cases, causes other than toxæmia must be sought.

There are one or two other points that are worth mentioning. Although most shocked patients have a low pressure and a rapid pulse there are some notable exceptions. There is a certain class of case which comes in acutely shocked. After a period of rest and warmth the men improve, and exhibit a high blood-pressure, but any operative interference, however, at once produces a collapse—usually fatal. A second class of case is the shocked man with a slow pulse. These cases almost always end fatally. This class of case has a superficial resemblance to the high spinal injury described by Gordon Holmes.

Blood out of Circulation.—There is no clinical evidence as to the site of the "lost blood." There is no engorgement of the intestines or dilatation of the visceral veins. Nor can it be said that there is any œdema of the tissues, subcutaneous or otherwise, though possibly the lost fluid would not be sufficient to produce such a change. The concentration of the blood described as taking place in the cutaneous capillaries does not lead, as a rule, to a coloration of the skin, which is usually white, though in a certain number of cases there is obvious stasis and a purple tint.

Treatment.—Besides rest and warmth the treatment consists of getting fluid into the circulation. Natural means are the best, but if fluids cannot be retained and the case is urgent some other method must be employed. There can be no doubt that blood was the best fluid under war conditions, as nearly all the shocked cases had lost blood to a greater or less extent. On the other hand, the 6 per cent. gum solution is amply sufficient in many cases (Drummond and Taylor). Keith's observations tend to show that few men who reached the casualty clearing station had a total hemoglobin value below the danger point. It cannot be said that any definite criterion as to the need of blood is forthcoming. Keith also brought out an interesting point. If a man's vessels retain the power of taking up fluid he will almost invariably do well. The progressive dilution of the blood is a good sign, so that a low Hb. value may be an indication that things are going well, and not an indication for blood transfusion. In many cases not only is
the power of absorbing fluid lost, but the power of retaining added blood or gum is also absent, and a fatal termination is to be expected. The time factor is an important one.

The importance of a full circulation raises the point of the so-called preparation for operation. It would seem that the old time purging is to be avoided in the future, and if purgation is necessary time should be allowed for the circulation to become full.

As to the point as to whether shock as seen in the War is the same as that met with after operation, it would appear that the two are for all intents and purposes the same. In war sepsis, cold, exhaustion and usually haemorrhage are more potent factors than in civil practice. If we subjected our civil patients after operation to the same conditions as prevail after a man has been wounded, we should see as much shock in civil as in military practice. The nearer a man's condition is to "pure shock," the less can anything beyond "rest" be done for him.

Dr. F. W. Mott, F.R.S.

The investigation of the brain in various conditions of shock is difficult, and the results I have obtained require further observations and experiments before positive statements regarding the existence of fine cellular changes of a biophysical or biochemical nature can be definitely associated with shock, either as cause or effect. The observations, so far however, show the desirability of further clinico-anatomical observations and experimental investigations.

The lantern slides illustrate the vascular changes in the brain in various forms of shock, viz.:—

(I) CLINICO-ANATOMICAL.

(1) Shell shock.
(2) Shell shock, with burial and probably gas poisoning.
(3) Extensive burn shock.
(4) Wound shock. Gunshot wound cases of compound comminuted fracture with much laceration of muscle.
(5) Shell shock, with cardiac contusion.

(II) EXPERIMENTAL—HISTAMINE INJECTION.

All the cases of shock were attended by a great fall of blood-pressure before a fatal termination. The burn shock and wound shock cases died pulseless, in spite of injection of gum solution.
Examination of the brain of shell shock cases was characterized by haemorrhages into the sheaths of the vessels, and minute scattered haemorrhages into the substance of the brain in various regions. These haemorrhages are not due to a hyaline thrombosis of terminal arteries such as occurs in shell shock with gas poisoning, but to rupture of very small vessels and escape of blood into the peri-adventitial sheaths or into the substance of the brain.

I do not find these haemorrhages from ruptured vessels in other forms of shock to the same extent, and I believe they are actually due to the commotion.

In the cases of wound shock, burn shock, and shell shock, there was evidence of engorged veins of the meninges, and in the substance of the brain associated with venous and capillary stasis. But besides this evidence of stasis there was marked evidence of anaemia; for empty collapsed vessels with dilated peri-adventitial spaces are as frequently seen as dilated capillaries and veins containing blood corpuscles, or more frequently so. The condition is not unlike that seen in the brains of animals after ligation of all four arteries. The engorged meningeal and other veins may be due to backward flow from the venous sinuses.

In two of the cases of gunshot wound fat embolism was found in the medulla and cortex, but I did not think it was sufficiently prevalent to account for the fatal shock, although it may have been a contributory factor of importance.

The cell changes may be considered under two headings:—

(I) Chromatolytic.—The Nissl granules consisting of blocks of basophil substance do not exist in the living cell, but serve to estimate a quantitative change in the cytoplasm. In secondary shock attended by a prolonged anaemia there is a tendency to a breaking up of the Nissl granules and to a relative loss of the basophil staining substance; the nucleus appears larger and clearer than normal. These cases of shock all show this to some degree.

(II) The Colour Reaction.—In hyperpyrexia, in necrobiosis from complete anaemia, and in acidosis, there is a change in the colour reaction as well as a chromatolytic process. The cell tends to stain diffusely a dull purple with a mixed basophil and acidophil dye—e.g., Giemsa fluid. In experimental histamine poisoning this condition is seen. It seemed to me, moreover, that in the cases of shock from gunshot wound with laceration of tissues, and in the case of burn shock, there is a biochemical as well as a chromatolytic change indicative of
a toxæmia. The cells do not stain a bright blue; the Nissl granules when present are a dull purple and there is a tendency to a diffuse purple-pink staining. This change in the colour reaction is seen in many of the cells of the medulla and the cells of Purkinje, but not usually in the cells of the spinal cord.

Captain Kenneth Walker, R.A.M.C.

I shall lay stress on those points connected with shock which have a bearing on civil practice. Of all the work actually done in France of a more exact nature in investigating shock, that which most impressed me was the work carried out by Robertson at the Base, and by Keith in the First Army, on blood volumes. Robertson showed that the drop in blood volume that occurred as the result of shock, hæmorrhage, &c., was very much greater than most of us had previously suspected. A man's blood volume often sank to only 60 per cent., or even to 50 per cent., of its original amount as a result of hæmorrhage and of shock. Robertson emphasized the importance of reinforcing the fluid reserves of the body in combating this condition of exæmia. From his results, and owing to having read his paper, I, personally, derived the greatest advantage in the actual treatment of cases of shock hæmorrhage.

One or two of my cases struck me very forcibly in this connexion. On one occasion I had transfused a man with 700 c.c. of blood, and had apparently gained very little advantage from doing this. The case was an extremely bad one; the man's leg had been blown off from the thigh, and my blood transfusion did not cause him to rally at all. I then continued running in saline (2 per cent. sodium bicarbonate), more as a forlorn hope than for any other reason. Much to my astonishment, after I had got in a litre of saline following the blood used, the man opened his eyes and recovered in a dramatic manner. For his recovery I felt that the saline had been as necessary as the blood, and that I could not have got the same results from the use of either alone. Saline alone had invariably failed in such desperate cases to produce more than a temporary improvement, a failure that Professor Bayliss has fully explained by his animal experiments. But given in conjunction with blood the fluid apparently remained in the circulation and no subsequent fall in blood-pressure was noted. The importance of the volume of fluid given (apart from its nature), was a matter which was forced upon me by many subsequent cases of a similar nature.
On the night of a Canadian raid I transfused a man with 600 c.c. of blood in a cellar just behind the trenches. As a result of this transfusion his pulse became just perceptible. I then ran into him 500 c.c. of bicarbonate, and left him an hour, at the end of which time he received another 800 c.c. of blood. Altogether he received a very large contribution to his fluid reserves, as he was able to take in addition a considerable amount of fluid by the mouth. He recovered in the most astonishing way from the shock, but, as a result of the very long journey back to the clearing station and in spite of further transfusion following operation I believe he eventually died. I quote this case merely as another example of the importance of the volume of fluid given in contradistinction to its nature, in the treatment of exæmia. Indeed it was only when I fully realized the importance of the fluid reserves of the body that I began to get good results in the treatment of these cases. A routine was then established, in which blood transfusion was supplemented by continuous rectal salines, and by the giving of as much fluid by the mouth as the patient could possibly take. A special orderly was detailed to the job of encouraging the patient to drink. When vomiting negatived this procedure recourse was had to subcutaneous or intravenous infusions.

I believe I was one of the first to call Professor Bayliss's attention to the fact that a great many of the men to whom we had given gum in the field ambulances of the Third Army subsequently required blood on arrival at the casualty clearing station, and I think the criticism he mentioned in his paper is a very just one, that possibly if, instead of giving a second injection of blood we had given gum as the second injection, the result would have been the same. I confess to having failed frequently with gum, and I regret now that, instead of giving only 500 c.c. of gum solution, which was the custom of the field ambulances in the Third Army, we did not try the effect of giving larger quantities. But, having failed with the initial injection, it was rather natural to jump to the conclusion that the gum was at fault, and to fall back on blood. Of the great superiority of blood over gum in the great majority of cases I have no doubt in my own mind, but I fully admit the justice of Professor Bayliss's criticism.

As General Cuthbert Wallace pointed out, the lesson to be learnt from this work of Robertson and Keith is that in civil practice we must pay more attention to the question of the fluid reserves, and thus assist the blood to dilute after operation. I think the condition of the patient who undergoes gastro-enterostomy is, clinically, very similar to that of
the badly-wounded soldier. In civil practice not only does one purge him and limit his fluids before operation, but some surgeons limit his fluids with perhaps unnecessary severity after operation. Small wonder that the shock of the operation acting on a body with depleted fluid reserves, and weakened still further in some cases by haemorrhage, may occasionally prove too great. In such cases it would seem wise to pay greater attention to the fluid reserves of the body, and where these are low to administer large quantities of saline by the rectum before and after operation. I do not know what is the latest work with regard to absorption of fluid from the bowel during a state of shock, but in all the cases of fatal shock in which I have performed a post-mortem examination, and in which I have given large quantities of fluid by the rectum, there was an absence of fluid from the bowel, it having, presumably, been absorbed into the general circulation. For this reason it was the custom to rely chiefly on rectal and oral administration in all except the most urgent cases. It was found that one could get into the circulation a much larger quantity of fluid in that way than by the subcutaneous method. In very urgent cases intravenous injections were given.

With regard to anaesthetics in shock cases, particularly must one be careful of the false impression of security conveyed by the use of ether. Seen on the operating table, a shocked patient who is receiving ether may appear to be in a fairly satisfactory state: but half-an-hour, or an hour, after operation there is, in many cases, a tremendous reaction. Therefore we relied, more and more, on gas and oxygen. The giving of oxygen seemed to be beneficial whatever the nature of the anaesthetic employed. Captain Milligan showed also that the giving of oxygen for half-an-hour, or an hour, or even longer, through a suitable mask after operation, was certainly followed by good results and a rise of blood-pressure. Therefore, to my mind, next to the importance of the fluid reserves in the blood in the treatment of these shock cases, comes the question of the anaesthetic.

Like several others, I tried the effect of applying a tourniquet in suitable cases, with the idea of cutting off toxins from the damaged tissue and preventing their entry into the general circulation, but, as a rule, the pain caused by the tourniquet was so severe, and so definitely brought in the element of nervous shock, that I came to the conclusion it was not practical as a routine treatment.

Before closing, I would like to say how much we, working at the Front under considerable difficulties, owe to the Research Committee
for their assistance and advice. We were in constant communication with the London Shock Committee, and we owe them much for their suggestions and for their help in supplying us with various instruments and with the gum solutions used in the treatment of shock.

Colonel C. J. Bond, C.M.G., A.M.S. (Leicester).

The first of the two points on which I shall speak concerns the treatment of shock by means of blood transfusion. It seems clear that the question of compatibility between donor and recipient has now been settled as regards the red corpuscles. By the classification of donors into groups the danger which formerly threatened the patient from the agglutination and destruction of the red cells during transfusion has now been overcome. But as the result of recent observations in which we have used the blood serums of many different individuals, and incubated with these serums the leucocytes from other persons, we have arrived at the conclusion that different blood serums vary very much in their action on foreign leucocytes when these are incubated together outside the body. If the same result holds good in the case of transfused blood, then we shall have to reckon with this question of compatibility not only in regard to the action of any serum on the red cells but also in regard to its action on the leucocytes of the donor. I cannot help thinking of this point when I realize the probable presence of a chemical element in the toxæmia of shock, especially in view of Dr. Dale's work on histamine. If we rapidly bring about a disintegration of the leucocytes of the transfused blood by a leuco-toxic serum then these products of leucocytic disintegration may have an important bearing on the curious symptoms which we sometimes get after blood transfusion.

The other point concerns the mechanics of the circulation. Whatever may be the difficulties in coming to a decision as to the essential cause of shock, the Shock Committee, and I think clinicians will agree with them, have come to the conclusion that the trouble as regards the heart is, that owing to a deficient volume of blood in effective circulation the heart is unable to fill properly during diastole. In this connexion I may briefly mention some observations which were carried out many years ago by the late Sir Victor Horsley and myself on sheep and other animals at the Brown Institution. The sheep is an animal which may

die with dramatic suddenness when it is "cast" as the farmer says, that is, when it is thrown on its back for any length of time. I am now alluding to the influence of bodily position on the intracardiac blood-pressure in the right auricle, and on the capacity of the heart to fill during diastole. These early observations have been confirmed by recent observations carried out on a soldier with a bullet embedded in the substance of the left ventricle, in whom we were enabled, by the use of the X-ray image of the bullet, to trace the heart movements antero-posteriorly, vertically, and from side to side when the patient was placed in the supine, prone, erect, and lateral positions. Bodily posture undoubtedly exercises an influence on the position of the heart in the chest, and on the capacity of the heart to fill with blood during diastole. This point has a practical bearing, because I am somewhat doubtful as to the wisdom of keeping shock patients in the "flat on the back" and partly inverted positions during treatment. When the patient lies flat on his back and partly inverted the heart tends to fall back on its own base against the spine, the heavy ventricular portion presses on the thinner auricular portion and the heart does not fill as easily in this position. Although the difficulty with the circulation is primarily due to deficiency in blood volume and not to the heart, yet any position which facilitates the refilling of the heart under these conditions will help to restore the patient. These observations on animals\(^1\) were confirmed by experiments on myself and other individuals in which an air tampon was introduced into the oesophagus down to the level of the right auricle. By connecting this tampon with an air tambour and recording drum, we were able to obtain a record of the "base" beat of the heart. We found that the erect, the prone, and the left-sided positions in which the heart can fall downwards and forwards away from its base enable it to fill more readily as shown by the base beat records. A further confirmation is supplied by the posture assumed by patients suffering from certain heart affections. We do not find that such patients want to lie flat on their back, they prefer a partly sitting up, left-sided, or leaning forward posture, in which the heart can elongate more readily and fill better.

In regard to the bearing of the facts which have been observed during the War on the treatment of shock in civil practice, I agree with General Wallace that we do not usually see a "juiciness" of the tissues, when operating on patients who are the subjects of shock, and I also

\(^1\) Published in the *Brit. Med. Journ.*, 1885, ii, p. 1109.
agree with him that everything points to the fact that shock in civil patients is essentially of the same nature, though perhaps initiated in different ways, as the shock met with in military practice.

One word about the use of gum solution. From conversations with officers serving in the Macedonian Army, and especially with those working in casualty clearing stations, I understand that gum has not fully maintained its reputation on this Front. This may be due, however, to one or possibly two things. It may be due to changes in a stock solution brought about by the hot climate, but it may also be due, as General Wallace pointed out in the case of one of the armies on the Western Front, to difficulties in getting the men back in the mountainous area where, owing to the delay, the patients were often in a desperate condition before transfusion could be carried out.

In conclusion, as a member of the Medical Research Committee who is not also a member of the Shock Committee (and I can therefore speak freely), I think we should all like to thank the Chairman for introducing the use of the gum solution, and the Members of the Shock Committee, and Dr. Dale, for the judicial and masterly way in which they have brought together the available evidence on the question of shock and for the valuable work they have done from the military and as I hope also from the civil point of view.

Sir W. Arbuthnot Lane, Bt.

The view I have always held about shock, and here I mean shock quite distinct from that which is produced by loss of blood, is that it is a condition of acute intestinal auto-intoxication. The factor that is responsible for it is the large quantity of toxic material that during the process of digestion is being carried from the small intestine through the portal circulation to the liver. This varies in amount with the quantity of chyme in the small intestine at the time. As, in a considerable proportion of people the contents of the small intestine are infected by organisms the portal blood must in them be extremely poisonous. Such infections are likely to abound in the conditions of warfare. Only those surgeons who have freed the liver from an enormous flooding with infected blood poured into it through the portal system by performing a colectomy in a toxic subject and have observed the marvellous results that follow so rapidly, can realize the amount of poisonous material that the liver has to deal with in these cases. In my experience in this war I have noticed that the amount
of shock and its consequences have borne a direct relationship to the
degree of infection of the gastro-intestinal tract. This is most manifest
in those sequelæ of shock which are grouped under the term of neurasthenia, since these sufferers are almost invariably demonstrably toxic. One can easily imagine that any very powerful mental stimulus may inhibit the function of the liver sufficiently to permit of the flooding of the general circulation with poisonous products in such quantity as to produce death. Certainly all the symptoms of shock are identical with those of acute auto-intoxication.

It seems to me perfectly clear that the poisonous products which are responsible for the symptoms of shock cannot always be derived from damaged structures, as has been suggested, since shock in all degrees of severity may ensue when there has been no obvious traumatism and certainly no coarse destruction of tissues. Even in the case of injury the degree of shock bears no constant relationship to the extent of damage sustained by the soft parts.

How rarely do we get symptoms of shock in the largest and most prolonged operative procedures for the results of chronic intestinal stasis, provided care be taken to evacuate the small intestine as a preparatory measure! This view does not militate against the supposition that acidosis may exist and may play an important part as an end result in the production of the symptoms of shock. Again, the shock which a patient, the subject of acute intestinal obstruction, sustains when operated on while the small intestine is distended with infected and stagnating material is familiar to us all. The surgeon is well aware that the only means he has of combating shock in these circumstances is to evacuate the distended small intestine as thoroughly and as quickly as possible, and to introduce six to eight pints of saline into the axillæ at the time of the operation. The effect of this saline is to free the circulation from toxins absorbed from the intestine, which, if not removed, would produce the symptoms classified as "shock." Dr. W. J. Mayo and Dr. Crile examined a number of colectomy cases at Guy's and were much impressed by the complete absence of shock in any of them.

Mr. J. P. Lockhart-Mummery.

The modern physiologist has too often been interested only in the scientific aspect of his investigations into animal physiology without regard to the practical application and value of his discoveries. This
is one of the natural results of physiology being divorced from practical application and becoming a special study. In an ideal state of things every surgeon should be a physiologist, as every physiologist should be a surgeon or physician. The War in this as in so many other respects, has forced us to co-ordinate our knowledge for practical ends. On these grounds I particularly welcome the opening paper emanating as it does from the laboratory of a pure physiologist.

The sequence of events when a new truth is discovered, or a new principle demonstrated, is much as follows: A clinical phenomenon is observed by doctors and an explanation is sought in the physiological laboratory. As the result, often of much work, an explanation of this phenomenon is found, and certain natural laws governing it are discovered. If the discovery is a true one the application of these principles and rules to clinical cases will enable us to control or abolish the phenomenon in question, in proportion as we correctly apply these principles. One of the best examples of this was the discovery of the principle of antiseptic surgery by Lister. The pure physiologist is apt to lose sight of the fact—because it does not come under his observation—that the best proof of the correctness of the new principle is that it enables the surgeon to control the phenomenon. The greatest proof of the value of Lister's discovery is the fact that the application of his principle does away with sepsis.

Now in the case of surgical shock certain laws were discovered, and what has been described as the vasomotor theory of shock, which was first propounded by Dr. Crile and later by myself, was formulated. Since then those principles have been widely applied as a means of combating surgical shock in the operating theatre, and as the direct result of the application of those principles surgical shock has almost disappeared in civilian surgical practice. When I was a house surgeon surgical shock was a common cause of death after any big operation, and as a house surgeon I was constantly having to treat shock. At the present time surgical shock is very seldom seen in any modern hospital as the result of an operation. Personally I have long ceased to fear shock from any operation, and I should now feel as much ashamed of a case of mine suffering from shock as I should of a clean case which developed sepsis as the result of my interference. We do not have to treat shock in these days because we do not cause it. This is the direct result of the application of a physiological principle, and is in itself the best possible proof that the principle was a correct interpretation of the phenomenon of surgical shock.
But four and a half years ago the War came and certain facts soon became evident. One of these was that the methods of combating shock which were so successful in the operating theatre at home failed when applied in the casualty clearing stations. Another fact also became evident even earlier, namely, that the methods of combating sepsis which were in universal use failed completely when applied to wounded men on the field. Almost all the wounds received by our fighting forces in this war have become septic. This has not, however, led us to abandon the principle of asepsis, or to doubt its essential truth. We have discovered that as the result of modern conditions of fighting new factors have been introduced into the problem which have rendered our methods of combating sepsis ineffective. The same has been the case with shock. Personally I have always recognized that there were certain cases which appeared to be shock, which did not respond to treatment in conformity with the vasomotor theory of shock. The notable exceptions were burns, and cases of peritonitis and intestinal obstruction. I pointed this out as long ago as 1905 in my Hunterian lectures, and observed then that these cases suggested a toxæmic cause for the condition. I think it possible that Dr. Dale and Professor Bayliss may be right in believing that chemical poisons, the direct result of massive injury of tissues, are one of the causes of the condition described as shock in men wounded in the field. I believe this condition of shock is closely related to that seen after burns, and that the same treatment will help both.

The trouble at present is that there is no proper accepted definition of shock. The term is one applied to a clinical state or phenomenon with which surgeons are well acquainted. Unfortunately we are not at present able to distinguish clinically any material difference between the clinical picture presented by, we will say, a patient dying from a severe burn, and one dying from a severe surgical operation performed by what my friend Dr. Crile has described as a "carnivorous" surgeon. The physiological causes are different, but there is very little difference in the clinical picture.

We must recognize that there are several different physiological conditions often described as surgical shock. It is essential that we should differentiate between them. Surgical shock, as it used to be seen in the operating theatres, is one thing, and I am convinced that the vasomotor theory of its causation is correct. It can be entirely avoided, and when it occurs it is best treated by morphia and pituitary extract, complete rest, warmth, the administration of fluid, &c. Toxic
shock, as seen in war and in cases of burns, is quite another thing (although both conditions are often associated), and while from the nature of its causation we cannot hope to deal with it as we have done with surgical or operative shock, that is, by prevention, we should be able to treat it by appropriate measures. Among these I should assign a prominent place to continuous saline infusion, as it has been shown that toxæemias, whether chemical or bacterial in origin, are best treated by flooding the tissues with water, on the principle that in this way we dilute the poison and enable the kidneys to eliminate it as quickly as may be. One has only to consider the wonderful results of treating general peritonitis and cholera on this principle to realize that we are only at the beginning of its use in dealing with toxic states.

The openers' theory of shock as essentially a toxæmia is a conception not compatible with surgical shock as it used to be seen in the operating theatre, since in such cases no cause for the toxæmia exists. I suggest, too, that they must produce direct experimental evidence that there is a toxæmia in experimental shock. This can be tested by a cross-circulation experiment, but so far the evidence from such experiments is all the other way and opposed to any change in the chemical constitution of the blood. The openers of the discussion apparently accept the theory that the arterioles are contracted in shock, but they have not brought forward any satisfactory evidence to prove it and so far all experimental and clinical evidence is quite opposed to such a supposition. But, to my mind, the weakest link in their theory is the fact that it has not suggested any new means of treatment. The methods used at the Front, and those suggested in the papers, are such as were suggested by me in 1905 as compatible with the vasomotor theory of surgical shock—namely, rest, warmth, and means to increase the total amount of fluid in efficient circulation.

Judging from the slides exhibited by Dr. Mott, it is quite evident that one of the patients who died from supposed shock was suffering from fat embolism, while the patient whose brain sections show collapsed cerebral vessels, evidently died from having been made to sit up while he had a low blood-pressure. He certainly did not die from surgical shock, but from being placed in the wrong position while his blood-pressure was inefficient. In other words, he died from collapse, the result of incorrect treatment.
Mr. C. H. S. Frankau.

With regard to one point mentioned by General Wallace—namely, the effect of cold on the wounded man—in order to find out something about this, I got out a certain number of figures. They were all from one clearing station, which was in the same area for thirty-six months on end, and was drawing patients from the same part of the line. We took three periods of winter months, December, January and February, and three periods of summer months, June, July and August, and took out the percentages of deaths from wounds in those periods. The numbers we dealt with were large, so the conclusions probably have some accuracy; we dealt with about 10,000 wounded in these periods. We found that for the summer months the average death-rate from wounds was 3.3 per cent., whereas in the winter months the average death-rate from wounds was 8.1 per cent. In other words, in winter the death-rate was rather more than double the summer rate. There was a slight diminution in the death-rate during the last period, both in winter and summer, owing to improved methods of transport.


There has been much discussion as to whether post-operative shock seen at the base in civil surgery is the same as the traumatic shock seen in the War. In the latter the factors concerned are primary nervous inhibition, cold, haemorrhage, exhaustion, toxic absorption, circulatory stasis and the vicious circle produced by prolonged low blood-pressure, while if an operation is necessary the anaesthetic used becomes also of extreme importance. In the causation of traumatic shock, primary nervous inhibition, cold and toxic absorption seem to play the chief parts.

In post-operative shock all these same factors may obviously be concerned, but their relative importance seems very different. Exhaustion of the circulatory system as the result of the strain due to the excitement as well as to the reaction to nerve stimulation is here very important, especially in a toxic individual with little reserve strength. Haemorrhage is of at least equal importance, since I have found the amount of blood lost to be much larger than is usually imagined. It has to be remembered that any patient seriously ill cannot afford to
strain his heart with excitement while at the same time losing blood, especially if he is anaemic, and as this combination of bad conditions is further strengthened by the anaesthesia, it is small wonder if he sometimes feels the strain too great for him.

I entirely agree with Captain Walker that nitrous oxide and oxygen is the ideal anaesthetic for these cases at the base as for the patient suffering from traumatic shock. Whether a patient be given nitrous oxide or ether it is most important that he should have a free supply of oxygen, but the nitrous oxide is superior to ether. This may perhaps be due to the possibility of giving the patients food and drink by mouth both shortly before and after the operation. There is no disadvantage to the patient in a light anaesthesia. I kept one patient who was very ill with a septic anaemia of 28 per cent. haemoglobin so lightly anaesthetized with nitrous oxide and oxygen that he called out during most of the operation, "Really, doctor, I can't stand any more," and yet he remembered nothing of the operation, had a higher blood-pressure after operation than before, and maintained it, and made an uninterrupted recovery. And this in spite of the operation, which, including amputation of the thigh in the middle third, and the incision of two big abscesses extending up the muscle planes almost to the hip, was extremely severe for such an anaemic and septic patient. A preliminary injection of morphia to diminish the excitement before operation is useful in removing some of the extra strain thrown on the circulation.

Exposure to cold with fall of body temperature takes place, but is rarely of much importance, a fall of rectal temperature of 1° C. being rarely exceeded. If, however, the exposure to cold is severe, considerable stasis with blood concentration occurs in the peripheral vessels.

No doubt some toxic absorption from the muscles may play a part in post-operative shock, but it is not often of primary importance in my opinion. In this connexion it is interesting to note that in the few head cases that I have examined during operation I have found dilution of the blood commencing even during the operation, while operations on the limbs seem to be almost invariably followed by some slight temporary blood concentration. But the differences do not seem great enough to suggest that this factor is of first-rate importance in the average patient.

Some septic patients who have previously shown a slow progressive anaemia are found to get a slight increase in the haemoglobin percentage during the few days before death. So that a blood concentration
in septic cases is probably sometimes in being even before an operation is commenced.

One can therefore conclude that the chief factors in post-operative "shock" are exhaustion, hæmorrhage, and the resulting anæmia, while the work of others suggests cold and toxic absorption from muscles combined with hæmorrhage and the resulting anæmia as the chief causes of traumatic shock. It would appear, therefore, that in civil surgery the most important factors to be considered are the anaesthetic, careful hæmostasis, and the avoidance of the desiccation of the patient.

Professor W. M. Bayliss, F.R.S. (reply).

It seems to me that something more than arterial constriction, as suggested by Mr. Malcolm, is required to explain the phenomena of secondary shock. It is difficult to realize how the loss of fluid, caused presumably by the high blood-pressure, is sufficient to account for the large decrease in effective blood volume, especially in view of the fact referred to by General Wallace that the tissues are not particularly moist. Evidence has also been presented that the arterioles are still constricted even when the blood-pressure has fallen very low.

As regards the absence of marked cyanosis in shock, we must remember that a very small dilatation of the capillaries, if widely distributed, may soak up a large volume of blood, and there is always the possibility that the pale cases may have lost blood by external hæmorrhage. I understand from General Wallace that the majority of cases of wound shock, seen by him, had lost blood, and were pale. The blue cases were exceptional, but may have lost blood. Two particular cases, which were blue, had not lost blood.

Captain Kenneth Walker's experience with a combination of blood and saline is important. It seems to show that the essential thing is to keep up a good volume of fluid in the circulation. I am somewhat surprised that he did not find gum to serve this purpose. The advantage of intravenous injection of gum over forced fluid by the alimentary canal is the rapidity of its effect, sometimes a matter of importance. When the state of shock is due to capillary stasis, it has always seemed to me that the advantage of gum over saline is that it keeps up a good circulation until the capillary blood has been restored to the main body of the circulating fluid. Saline leaves the
vessels too quickly to ensure this; otherwise, there is no obvious reason why it should not serve the purpose when no blood has been lost by hæmorrhage.

With reference to the bad effects of deficient oxygen supply in anæsthesia, the question as to why they are so lasting is an interesting one. It was also the case in the experimental shock investigated by Cannon and myself.

There seems to be no doubt that the cases in Macedonia, referred to by Colonel Bond, were not treated sufficiently early. Transfusion of blood was not tried. It has frequently happened that the first dose of gum is insufficient in amount, and that a repetition effects the desired object. It is remarkable how large a quantity can be given without harm. It is also to be kept in mind that some samples of gum are ineffective, for some unknown reason. I have not found this to be the case with that sold as “Turkey elect,” which was used for the supply in France.

The cross-circulation experiment suggested by Mr. Mummery, would be valuable. But the fact that shock is produced by muscle injury in cats when the nervous supply of the limb is cut off shows that there is something in addition to nervous reflexes.
The Royal Society of Medicine.

President—Sir Humphry Rolleston, K.C.B., M.D.

Marcus Beck Laboratory Reports.—No. 8.

An Enumerative Study of Entamoeba coli Cysts in Stools.

By J. W. Cropper, M.B., M.Sc.

In a paper read before the Section of Pathology, the Royal Society of Medicine, I described certain new methods for the enumeration of protozoa and other organisms, and I referred to the fact that one of these methods had been in constant use for counting Entamoeba coli cysts in stools. In the present article I propose to give the details of this enumeration, in order to demonstrate that the method is a practically useful and reliable one for the purpose. Moreover, from subsequent experience, I feel satisfied that the method could also be employed for the enumeration of almost any organism (or cell) which can be recognized, and differentiated from surrounding objects, with low powers of the microscope. The method may be briefly described as follows:

A microscope slide, 3 in. by 1½ in., is ruled in columns ½ mm. wide, parallel to its length almost across its entire width. The slide is placed on a turn-table, which is made to revolve, and a drop of molten paraffin-wax is applied by means of a small camel-hair brush to the ruled surface of the slide near one


2 A special counting chamber, known as a Protozoometer, has been designed for the enumeration of organisms and cells whose differentiation requires a high magnification (loc. cit.).
edge. The drop forms a large ring, of almost uniform thickness, and sets solid. In the centre of this ring a measured volume (usually 20 c.mm.) of a suspension of the organisms or cells to be counted is placed, and the "counting chamber" is completed by the application of a cover-glass (1½ in. circle). The liquid spreads out into an irregular area, and the objects (e.g., cysts) are counted with a low power in the whole of the volume, a process which occupies on an average about fifteen minutes. It is obviously unnecessary to know the exact depth of the chamber (it is usually about ½ mm.), and, indeed, it is rather an advantage that the thickness of the wax ring can be varied to suit different requirements. The improvised chamber can be used over and over again without material deterioration, and when the ring has become too thin it can be removed with xylol, and a fresh one supplied. The ruled slides can be obtained from Messrs. Angus and Co., Wigmore Street, London, and are comparatively inexpensive. A permanent chamber, constructed on the same principle, could doubtless be made, if required, by any manufacturer of optical apparatus.

The selection of Entamoeba coli cysts for enumeration was determined chiefly by the fact that they constituted a particularly suitable "test object" for the method, and the actual figures obtained are only intended to be regarded as of secondary importance. The cysts are of such a size that they can only just be recognized (and that often with uncertainty) with a low power, their presence is masked by the large amount of débris in the stool, and they are subject to marked variations in number from day to day. If the method were found to be reliable for enumeration under adverse conditions such as these, it could reasonably be assumed that it would be at least equally so under the more favourable circumstances which usually prevail, and it could therefore be regarded as a method of more or less general applicability. The occurrence of an Entamoeba coli infection in one of the staff provided an opportunity and the necessary material for carrying out such a test, and a daily enumeration of the cysts in the stools was therefore undertaken. At the same time it was thought that the available figures resulting from an enumeration lasting for a considerable time, say three months, might afford some indication as to whether a periodic rhythm was present in the case of Entamoeba coli, similar to that which has apparently been found in certain cases of Lamblia infection. The experiments were carried out in 1917, along with other investigations, on behalf of the Medical Research Committee, and their completion has been prevented and publication delayed by the war. For many useful suggestions and for practical assistance in the work I am greatly indebted to the late Mr. R. W. H. Row, B.Sc., and Miss M. Lovibond, V.A.D.
The Royal Society of Medicine

The procedure for the enumeration of *Entamaba coli* cysts in stools may be described in two stages, viz.:

1. Preparation of a uniform emulsion of the stool.
2. Counting the cysts in the emulsion.

1. *Preparation of a Uniform Emulsion of Stool.*—This is not always the simple matter it might appear. Many stools, even although in outward appearance soft and semi-formed, contain lumps of a clay-like consistency which are extremely difficult to disintegrate. Prolonged shaking on a mechanical shaker is insufficient to break them up, and in fact it is necessary practically to grind them up in a mortar. Such a proceeding is of course liable to damage many of the cysts, and the only remedy in such cases is to use a small amount of the stool, and as little force as possible. So far as I am aware, the uniformity of an emulsion can only be judged of by naked-eye examination, and I am not satisfied that the method, which has been used by others, of preparing an emulsion by mixing the stool with a glass rod in a small cylinder, can always be relied on. The method which I have found the most satisfactory is as follows: Weigh out 2 grm. of the stool on a watch-glass. Wash this with distilled water (or normal saline, if preferred) from a wash-bottle into a small mortar, and gently emulsify by stirring with a pestle. Decant the liquid through a small filter funnel into a 100 c.c. cylinder, fitted with a rubber cork and preferably without a lip. Add more water to the residue in the mortar, repeat the emulsification until the whole of the stool has been transferred to the cylinder, and finally make up the volume to 40 c.c. Shake thoroughly by hand (or on a mechanical shaker) for a few minutes. The 5 per cent. emulsion, which has thus been prepared, may be transferred to a small wide-mouthed bottle, in order to facilitate the subsequent removal of a small volume in a graduated capillary pipette for counting purposes.

I have found that a 5 per cent. emulsion has the maximum density which is generally permissible for counting by the present method, except in the case of liquid stools. Sometimes it is even necessary to dilute this to half the strength, with a consequent diminution in the number of cysts which can be counted in a given time. Since the practical value of a method of counting depends on the reduction of the labour and time involved in the process to a minimum, consistent with the enumeration of sufficient objects to maintain the errors due to random sampling within reasonable limits, the emulsion should be used as dense as possible.
In order to determine the number of cysts excreted per day, it is obviously necessary to know the weight of the whole stool evacuated. For this purpose the stool may be passed into a wide jar (such as an earthenware jam jar with half a Petri dish for a lid), which has been previously counterpoised or weighed. The necessary data are then readily obtained.

(2) Counting the Cysts.—A small quantity of the emulsion of stool (thoroughly shaken up immediately before use) is drawn up into a graduated capillary pipette provided with a rubber teat, 20 c.mm. (as a rule), is rapidly expelled on to the ruled slide already described, and the cover-glass is applied, resting on the wax ring. The low-power magnification is arranged so that the cysts can be seen fairly easily, and the columns on the slide occupy about half the diameter of the microscope field. It is most important to adjust the light so that the cysts stand out, on account of their high refractility, in sharp contrast with other objects. Indeed, it is owing to their glistening white appearance under proper conditions that a rapid and accurate count under a low power is at all possible. The addition to the emulsion of iodine, or of any of the various substances which have been used to form a contrast background, is a decided handicap to the speed of counting cysts with a low power. The cysts are counted in the whole of the drop, with the aid of the columns, and the necessary calculations are readily made. It is always advisable to make at least two counts of each stool, and several may be required when the cyst-content of the stool is unusually low. The following table is abstracted from the complete record of the counts, which is illustrated graphically in Charts I and II, and is too lengthy for reproduction in full:—

An examination of the above table and charts shows the marked variability of the cyst-content of the stools from day to day, and the enormous number of cysts which may be present in a given sample. The lowest recorded count (January 19) was one of 3,250 cysts per grammé of stool (moist), while the highest was 323,690 (February 27). The number of cysts excreted in a single day varied from 290,000 to 64 millions (March 5). The average cyst-content of the stools examined from January 10 to April 1 (nearly three months) was 122,700 per grammé of moist stool, and the average number of cysts evacuated daily for the same period was approximately 16 millions.

It can readily be estimated from the figures given in the table, that during the whole period of three months, if a film had been prepared for examination in the usual way between a slide and cover-slip, but
from a uniform emulsion, and the whole of the film had been searched through, on no occasion should cysts have been found absent. The amœbic infection has now persisted for three years (entirely unattended by symptoms), and only once during an occasional examination of a uniform emulsion of the stool have cysts been absent, while several times no cysts could be found in films prepared by the usual method of emulsifying one or two platinum loopfuls. I am convinced that the most reliable diagnosis of the presence or absence of cysts in stools (at any rate of Entamoeba coli cysts) can only be made by the examination of a uniform emulsion, prepared from a comparatively large bulk of the

<table>
<thead>
<tr>
<th>Date</th>
<th>Number</th>
<th>Consistency</th>
<th>Weight (grams)</th>
<th>Per cent. H₂O</th>
<th>Number of cysts counted</th>
<th>Number of cysts exercised per day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan. 10</td>
<td>6</td>
<td>M</td>
<td>163</td>
<td>74.0</td>
<td>440</td>
<td>78,640</td>
</tr>
<tr>
<td>,, 11</td>
<td>7</td>
<td>S</td>
<td>213</td>
<td>76.0</td>
<td>108</td>
<td>19,480</td>
</tr>
<tr>
<td>,, 12</td>
<td>8</td>
<td>F</td>
<td>21</td>
<td>67.6</td>
<td>204</td>
<td>40,470</td>
</tr>
<tr>
<td>,, 13</td>
<td>9</td>
<td>S</td>
<td>203</td>
<td>74.2</td>
<td>36</td>
<td>34,460</td>
</tr>
<tr>
<td>,, 14</td>
<td>No stool</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>,, 15</td>
<td>10</td>
<td>F</td>
<td>61</td>
<td>66.0</td>
<td>358</td>
<td>107,100</td>
</tr>
<tr>
<td>,, 16</td>
<td>11</td>
<td>S</td>
<td>133</td>
<td>76.4</td>
<td>74</td>
<td>49,500</td>
</tr>
<tr>
<td>,, 17</td>
<td>12</td>
<td>S</td>
<td>151</td>
<td>75.6</td>
<td>22</td>
<td>11,300</td>
</tr>
<tr>
<td>,, 18</td>
<td>13</td>
<td>S</td>
<td>76</td>
<td>71.8</td>
<td>31</td>
<td>10,430</td>
</tr>
<tr>
<td>,, 19</td>
<td>14</td>
<td>S</td>
<td>89</td>
<td>74.0</td>
<td>31</td>
<td>9,350</td>
</tr>
<tr>
<td>,, 20</td>
<td>15</td>
<td>S</td>
<td>193</td>
<td>78.8</td>
<td>10</td>
<td>5,880</td>
</tr>
<tr>
<td>,, 21</td>
<td>No stool</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>,, 22</td>
<td>16</td>
<td>S</td>
<td>184</td>
<td>70.4</td>
<td>33</td>
<td>18,770</td>
</tr>
<tr>
<td>,, 23</td>
<td>17</td>
<td>F</td>
<td>19</td>
<td>76.0</td>
<td>144</td>
<td>77,840</td>
</tr>
<tr>
<td>,, 24</td>
<td>18</td>
<td>F</td>
<td>95</td>
<td>67.4</td>
<td>147</td>
<td>75,580</td>
</tr>
<tr>
<td>,, 25</td>
<td>19</td>
<td>M</td>
<td>142</td>
<td>74.4</td>
<td>80</td>
<td>45,820</td>
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<tr>
<td>,, 26</td>
<td>No stool</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>,, 27</td>
<td>No stool</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>,, 28</td>
<td>No stool</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>,, 29</td>
<td>20</td>
<td>M</td>
<td>202</td>
<td>73.4</td>
<td>99</td>
<td>46,400</td>
</tr>
<tr>
<td>,, 30</td>
<td>21</td>
<td>F</td>
<td>68</td>
<td>70.0</td>
<td>710</td>
<td>214,500</td>
</tr>
<tr>
<td>,, 31</td>
<td>No stool</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feb. 1</td>
<td>22</td>
<td>M</td>
<td>246</td>
<td>78.0</td>
<td>504</td>
<td>78,370</td>
</tr>
<tr>
<td>,, 2</td>
<td>No stool</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>,, 3</td>
<td>23</td>
<td>S</td>
<td>189</td>
<td>73.6</td>
<td>38</td>
<td>20,630</td>
</tr>
<tr>
<td>,, 4</td>
<td>No stool</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>,, 5</td>
<td>24</td>
<td>F</td>
<td>107</td>
<td>70.4</td>
<td>1,061</td>
<td>160,270</td>
</tr>
<tr>
<td>,, 6</td>
<td>25</td>
<td>S</td>
<td>62</td>
<td>74.2</td>
<td>2,713</td>
<td>228,350</td>
</tr>
<tr>
<td>,, 7</td>
<td>26</td>
<td>M</td>
<td>102</td>
<td>73.0</td>
<td>1,146</td>
<td>210,120</td>
</tr>
<tr>
<td>,, 8</td>
<td>27</td>
<td>S</td>
<td>174</td>
<td>—</td>
<td>306</td>
<td>107,940</td>
</tr>
<tr>
<td>,, 9</td>
<td>28</td>
<td>F</td>
<td>31</td>
<td>—</td>
<td>639</td>
<td>127,290</td>
</tr>
</tbody>
</table>

* F = firm; M = medium; S = soft.
stool and preferably also concentrated by centrifuging. In the event of a negative finding which still leaves room for doubt, the usual examination should always be supplemented by this proceeding.

The charts give little, if any, indication of a periodic rhythm, either in the number of cysts per gramme of stool or in the number excreted per day. In both cases the maxima appear to recur about once every four or five weeks, but it is obvious that a number of stools would have to be counted over a long period before any definite deductions could be made. In order, however, to complete the investigations as far as possible, and to eliminate the possibility of a variable water-content of the stools leading to fallacious conclusions, it was decided to determine the moisture in a number of the samples, and calculate the number of cysts per gramme of dried stool. For this purpose 5 grm. of each stool were accurately weighed out in a small (2 in.) Petri dish, the water was expelled at about 90° C., and the weight was taken

![Chart I.](image_url)

To show the daily variation in the number of cysts of *Entamoeba coli* per gramme of stool. The figures representing the ordinates are multiples of one thousand.
hourly (after the fourth hour) until constant. In a consecutive series of twenty stools the amount of water varied from 66 per cent. to 78.8 per cent., with an average of 73.1 per cent., and a reference to Table I and the subjoined chart shows respectively that the amount of moisture is no criterion of the number of cysts which might be expected in a given stool, and does not appear to have any influence on a possible periodicity (cf. Chart I). The average number of cysts per gramme of dried stool during this period was 230,000, while for the full period of

![](chart.jpg)

**Chart II.**

To show the variation in the number of cysts of Entamoeba coli excreted per day. The figures representing the ordinates are multiples of one million.

three months it was 456,000 (assuming a mean water-content of 73.1 per cent.). In spite of the apparent magnitude of these figures, it can readily be calculated that the whole of this half-million cysts would occupy a volume of only 2.5 c.mm. (approximately) and would constitute not more than 0.3 per cent. of the total solid matter of the stool.
Experimental Errors in the New Method.

Experimental errors are an unavoidable accompaniment of all methods of enumeration, and may be due to a large and indefinite variety of causes. In advancing the claims of a new method it is necessary to show that such errors are limited to a reasonable amount, and the following will require consideration:

1. Errors due to the use of the pipette.
2. Errors due to the "personal factor."
3. Errors due to random sampling.

To show the daily variation in the number of cysts of Entamoeba coli per gramme of dried stool. The figures representing the ordinates are multiples of one thousand.
(1) Errors due to the Pipette.—It is of course well known that uniformity in the delivery of a definite volume of liquid from a capillary pipette requires close attention to certain details of technique. Various forms of pipette have been designed, and elaborate methods for using them have been employed, which enable a considerable degree of accuracy to be attained. Such refinements would, however, be quite unnecessary for the enumeration of cysts in stools, where the experimental errors due to other factors are overwhelmingly greater.

In order to determine roughly the extent of the error which might occur in the delivery of 20 c.mm. of an emulsion of stool from an ordinary capillary pipette, twenty such volumes were expelled in succession and weighed on a delicate balance. The average weight of the supposed 20 c.mm. was found to be 19.75 mg., and the maximum errors were 4 per cent. below, and 6 per cent. above, this figure. Even, however, if it be assumed on these data that the error in the delivery is not likely to exceed about 5 per cent., a special danger suggested itself in the enumeration of cysts (or other objects) in stools. Not only have the cysts themselves a high specific gravity, but they may adhere to or become entangled in heavy foreign matter, and be rapidly carried to the bottom of the emulsion. It was therefore considered absolutely necessary to determine whether the cyst-content of successive 20 c.mm. volumes of the same emulsion, delivered each time in the same manner and with precautions to minimize these risks, was reasonably constant. The following table shows that the method is sufficiently reliable in this respect, provided that at least two counts are made. All the preparations were made and counted by the same person, and various strengths of emulsion (made by diluting the original 10 per cent.) were used in the test. On comparing the results given under the same number it will be seen that the influence of the density of the emulsion is negligible. Occasionally a considerable difference in the counts may occur (vide Table II*) and in such cases a further two (or more) counts should be made.

(2) Errors due to the Personal Factor.—The recognition of a cyst of Entamoeba coli in an emulsion of stool is by no means always possible under a low magnification, however prolonged the scrutiny: and, moreover, since the time occupied in the process of counting must be reasonably limited, it is highly probable that a number of cysts will be missed, and a number of other objects, especially starch granules, will be mistaken for cysts. Under such circumstances it was obviously most important to ascertain the extent of such errors, and it was
decided to compare the figures obtained by two observers in the enumeration of the cysts in the same preparation, and to verify the results by examining the whole of the specimen with a high power. The errors arising from this cause could of course be considerably reduced by using a dilute emulsion, and by spending a long time over the enumeration, but the main purpose of the method would thereby be defeated. The following tests were therefore made with emulsions whose density was the maximum permissible, and the counting was carried out as rapidly as possible. It should be mentioned that one of the observers (A) was comparatively inexperienced in the use of a microscope, and the method can be used by a beginner after a few days' practice. With the exception of one large error of omission (12.3 per cent.), probably due to too rapid counting, the results in the subjoined table indicate that an allowance of 5 per cent. would cover the error due to the personal factor in the case of the two observers. I would suggest that anyone who uses the method should certainly control his own accuracy by a few preliminary counts made both with a low and with a high power.

(3) Errors due to Random Sampling.—One of the fundamental principles which determine the practical value of a method of enumeration is the facility which it affords of counting a comparatively large number of objects in a reasonable length of time, and with a minimum expenditure of labour. The present method was specially devised to meet these requirements as far as possible, and it is only necessary to refer to the tables to show that a large number of Entamoeba coli cysts can be readily and accurately counted by its means. I am not acquainted with any other method which is so suitable for the purpose of counting, with a low power, organisms which are liable to occur in scanty numbers. On the one hand, the usual forms of counting chamber designed for blood-counts are much too small, while the Sedgwick-Rafter counting chamber, which has been used for the enumeration of protozoa in water, is too large. The dimensions of the chamber in the present method are so limited, and the width of the columns on the ruled slide is so arranged, that a sufficient number of cysts can, in an average sample, be counted in from two to four preparations. In very scanty infections it is of course practically impossible to count as many cysts as is desirable, unless some method of concentration is employed. The method is not intended to be used for organisms or cells whose differentiation with a low power is a slow and uncertain process, nor for those whose
numbers are sufficiently large to enable an adequate count to be made in a small chamber. For example, the cysts of *Entamoeba histolytica* and *Lamblia* are best counted with a high power in the protozoometer.

**Table II.—Number of *Entamoeba coli* Cysts in Successive 20 Cubic Millimetre Volumes of the Same Emulsion of Stool.**

<table>
<thead>
<tr>
<th>Number of emulsion—</th>
<th>10 per cent. emulsion</th>
<th>25 per cent. emulsion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Successive counts (1)</td>
<td>284</td>
<td>246</td>
</tr>
<tr>
<td>&quot; &quot; (2)</td>
<td>287</td>
<td>244</td>
</tr>
<tr>
<td><strong>5 per cent. emulsion.</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of emulsion—</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Successive counts (1)</td>
<td>281</td>
<td>302</td>
</tr>
<tr>
<td>&quot; &quot; (2)</td>
<td>251</td>
<td>266</td>
</tr>
</tbody>
</table>

**Table III.—Comparison of the Counts of *Entamoeba coli* Cysts in the Same Preparation Made by Two Different Observers.**

<table>
<thead>
<tr>
<th>Number of emulsion 5 per cent.</th>
<th>Observer A counted</th>
<th>Observer B counted</th>
<th>Actual number of cysts</th>
<th>Estimated (—)</th>
<th>Estimated (—/)</th>
<th>Percentage error</th>
<th>Observer A</th>
<th>Observer B</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>147</td>
<td>142</td>
<td>144</td>
<td>8</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>149</td>
<td>129</td>
<td>147</td>
<td>19</td>
<td>17</td>
<td>-2</td>
<td>+1-4</td>
<td>7</td>
</tr>
<tr>
<td>3</td>
<td>325</td>
<td>320</td>
<td>320</td>
<td>13</td>
<td>8</td>
<td>+5</td>
<td>+1-9</td>
<td>18</td>
</tr>
<tr>
<td>4</td>
<td>407</td>
<td>382</td>
<td>390</td>
<td>33</td>
<td>16</td>
<td>+17</td>
<td>+3-4</td>
<td>17</td>
</tr>
<tr>
<td>5</td>
<td>126</td>
<td>133</td>
<td>130</td>
<td>5</td>
<td>9</td>
<td>-4</td>
<td>-3-0</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>133</td>
<td>132</td>
<td>137</td>
<td>4</td>
<td>8</td>
<td>-4</td>
<td>-3-0</td>
<td>2</td>
</tr>
<tr>
<td><strong>Total...</strong></td>
<td><strong>1,287</strong></td>
<td><strong>1,238</strong></td>
<td><strong>1,268</strong></td>
<td><strong>82</strong></td>
<td><strong>63</strong></td>
<td><strong>+19</strong></td>
<td><strong>+1-5</strong></td>
<td><strong>54</strong></td>
</tr>
</tbody>
</table>
STATISTICAL EVIDENCE IN SUPPORT OF "CONCENTRATION METHODS" FOR THE DIAGNOSIS OF PROTOZOAL CYSTS IN STOOLS.

Some time ago I suggested that the search for protozoal cysts in stools would be facilitated if an emulsion of the stool were concentrated by repeated centrifuging, with or without the addition of ether, before examination. An opportunity now presented itself of finding out whether concentration could be effected with sufficient quantitative accuracy to allow the enumeration of cysts in a concentrate to replace a count of the original emulsion, the advantage being that a larger number of cysts could then be counted in a given time. The concentration process has already been used for various enumerative purposes, such as that of leucocytes in milk, Filaria embryos in blood and urine, &c., but in the case of stools it is necessary in the first place to determine whether the loss of cysts on centrifuging can be accurately controlled. The few definite data which I have been able to collect indicate that the concentration can be carried out with an inappreciable loss of cysts, and at the same time lend support to the value of the centrifuging for diagnosis. The concentration must always be carried out in precisely the same way, while still maintaining a degree of maximum efficiency, and a large number of counts and experiments was necessary in testing the influence of such factors as variations in the rate and time of centrifuging, strength of emulsion, &c., before the following was adopted as the best method:—

Preparation of Concentrates for Enumerative Purposes.—Ten c.c. of a 10 per cent. aqueous emulsion of the stool are placed in a graduated centrifuge tube, of about 15 c.c. capacity, and centrifuged at 1,200 revolutions per minute for two minutes. The upper 7·5 c.c. (approximately) of the liquid is poured off (not pipetted off), the volume is made up to 10 c.c. again with water, and the mixture well shaken. This process is repeated twice (i.e., three times in all), and the deposit is finally made up to 2·5 c.c. The cysts in such a concentrate, which is the equivalent of a 40 per cent. emulsion, can usually be readily counted, and since the maximum density permissible in the case of an original emulsion is 5 per cent., eight times as many cysts can now be counted in the same time. As a matter of fact the cysts can be easily recognized for diagnostic purposes (but not accurately counted) in a concentrate which is from twelve to twenty times the strength of the original emulsion.
The available data (vide Table IV) are admittedly too scanty to justify a claim that the concentrates could be used for enumerative purposes. There is, however, a noticeable uniformity in the case of concentrates prepared from the same emulsion on the same day, and further counts would doubtless show the cause of the discrepancies in those prepared on different days. The most interesting point is that the average loss of cysts in thirteen concentrates is only 2.8 per cent.

Table IV.—To show the Effect of Concentration on the Entamoeba coli Cyst-content Count in Stools.

<table>
<thead>
<tr>
<th>Number of emulsion</th>
<th>Original Emulsion</th>
<th>Concentrates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cysts per gramme of stool</td>
<td>Number of cysts counted</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>1</td>
<td>271,300</td>
<td>351</td>
</tr>
<tr>
<td>2</td>
<td>197,000</td>
<td>319</td>
</tr>
<tr>
<td>3</td>
<td>53,343</td>
<td>152</td>
</tr>
<tr>
<td>4</td>
<td>82,663</td>
<td>293</td>
</tr>
<tr>
<td>Total...</td>
<td>501,703</td>
<td>...</td>
</tr>
</tbody>
</table>

Rate of Degeneration of Entamoeba coli Cysts in Stools.

It was occasionally found impossible to make a sufficient number of consecutive counts on the same day as that on which the emulsion was prepared, and it was desirable to know whether the counting could be completed on the following day. Four preparations, each of 20 c.mm., were therefore made (from different emulsions), the specimens sealed with vaseline and kept at room temperature, and the cysts in them counted on the first day, and at subsequent intervals, with the results shown in Table V (see p. 14).

The rate of degeneration thus varies considerably in different cases, and it was noticed to be most rapid in the case of emulsions made from soft stools, in which the presence of a large number of early, thin-walled, and less resistant cysts, was observed. The results clearly show that the counting must be carried out without undue delay, and au—16.
they also indicate that it is advisable to examine stools for diagnosis as fresh as possible.

<table>
<thead>
<tr>
<th>Specimen No.</th>
<th>Number of cysts</th>
<th>Percentage loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial count</td>
<td>50</td>
<td>—</td>
</tr>
<tr>
<td>After twenty-four hours</td>
<td>42</td>
<td>16</td>
</tr>
<tr>
<td>After two days</td>
<td>37</td>
<td>26</td>
</tr>
<tr>
<td>After five days</td>
<td>20</td>
<td>42</td>
</tr>
<tr>
<td>After ten days</td>
<td>22</td>
<td>56</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Specimen No. 2</th>
<th>Number of cysts</th>
<th>Percentage loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>43</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>38</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>37</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>54</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Specimen No. 3</th>
<th>Number of cysts</th>
<th>Percentage loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>68</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>31</td>
<td>54</td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>62</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>70</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>76</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Specimen No. 4</th>
<th>Number of cysts</th>
<th>Percentage loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>103</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>56</td>
<td>46</td>
<td></td>
</tr>
<tr>
<td>45</td>
<td>56</td>
<td></td>
</tr>
<tr>
<td>39</td>
<td>62</td>
<td></td>
</tr>
<tr>
<td>35</td>
<td>66</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Total</th>
<th>Number of cysts</th>
<th>Percentage loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>264</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>167</td>
<td>37</td>
<td></td>
</tr>
<tr>
<td>145</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>119</td>
<td>55</td>
<td></td>
</tr>
<tr>
<td>93</td>
<td>65</td>
<td></td>
</tr>
</tbody>
</table>

**CONCLUSIONS.**

The data presented in the foregoing pages are considered to furnish sufficient evidence that the method which I have described, for counting *Entamoeba coli* cysts in stools with a low power, is eminently suitable for the purpose. As is so frequently the case in enumerative studies, the actual results of the counting scarcely appear to repay the labour involved in the experiments, but it is at least desirable that one should be able to express mathematically the intensity of an infection and the variations to which it is subject. It has been shown that the method can be used for the enumeration of an object which frequently occurs in scanty numbers and under difficult conditions of observation, and it can reasonably be assumed that it could also be used for other organisms or cells under similar circumstances.
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DISCUSSION ON THE PRESENT POSITION OF SPINAL ANÆSTHESIA.

Dr. Felix Rood.

Spinal anaesthesia has now been used in this country with increasing frequency for the last ten or twelve years. In spite of considerable opposition it has attained a position as one of the recognized agents for producing anaesthesia. A large number of cases have been collected by various observers and I think we may hope that the time has now been reached when a comparison of experiences may be of considerable use to us all in deciding the exact type of case in which this form of anaesthesia is most valuable.

It is not my intention in opening this discussion to enter into the technique of spinal anaesthesia, which is now very well known, but rather to speak of my own experiences of its use in a considerable number of cases.

I have always used stovaine, except in about 250 cases in which novocain was employed. After this trial I gave it up, because although novocain produces perfect anaesthesia it does not produce a muscular relaxation equal to that produced by stovaine.

A 5 per cent. solution of stovaine, the density of which was increased by the addition of 5 per cent. of dextrose, was used in most cases. As this solution is heavier than the cerebro-spinal fluid the position and extent of the anaesthesia obtained with it can be regulated by the

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1 At a meeting of the Section, held December 6, 1918.
position of the patient during the injection. There is no doubt that although the stovaine-dextrose solution is diffusible its movements are controlled by gravity for a few minutes after injection. For instance, if a patient is placed upon his right side with his pelvis slightly raised so that there is a good steep slope from the third lumbar vertebra down to the mid-dorsal region, and the injection is made between the second and third lumbar vertebrae, this fluid will sink downwards in the spinal canal to the mid-dorsal region, producing an anaesthesia of the right half of the body before producing any effect upon the left side, and moreover if the patient is kept in this position for some few minutes before being turned on to his back, the anaesthesia will be more complete on the right side, that is to say, it will extend higher on the right side than on the left and it will last longer on the right side; muscular power will return first on the left side and the patient will be able to move his left leg before he moves his right leg.

If immediately the stovaine has flowed to the mid-dorsal level the patient is turned up his back it will flow across the mid-line and there will be practically no difference between its effects upon both sides of the body. Or again if the injection is given with the patient in the sitting posture the stovaine-dextrose solution will sink downwards in the theca and produce an anaesthesia which is more or less limited to the sacral plexus.

For young children I found that a dextrin-stovaine solution is less diffusible and consequently the upper limit of the anaesthesia and muscular paralysis was more sharply defined, which is naturally a great advantage, as in the short spinal cords of young children the vital centres are not far removed from those parts of the cord in which it is necessary to produce anaesthesia.

In a few hundred cases a solution of stovaine in saline was employed. It was found that irrespective of the position of the patient the stovaine diffused about 10 in. upwards from the point of injection and equally on both sides of the body. With this solution it was impossible to limit its action or to increase it beyond this point except by increasing the dose and then only very slightly. The anaesthesia produced by the saline-stovaine solution was found to be more transient than in those cases in which the denser solution was used and it was generally found necessary to employ almost double the dose of stovaine to produce equally long anaesthesia.

It might at this point be appropriate to say a few words in regard to the position of the patient after the introduction of the stovaine.
I have already remarked that the dextrose and dextrin-stovaine solutions are mobile in the cerebro-spinal fluid for a few minutes after injection, but only for a few minutes, and we have never found it possible after about five minutes to increase the height of the anaesthesia even by very considerable elevation of the pelvis; that is to say, I think that the stovaine becomes fixed in from three to five minutes. It follows from this that the patient’s head and the cervical region of the cord must be kept raised during the injection and for the first few minutes afterwards.

In the early days of the use of spinal anaesthesia we were very careful to keep the head and shoulders of the patient raised not only during the injection but throughout the operation and even in bed afterwards. This posture increased that tendency to syncope which is not at all uncommon. Latterly after a few minutes the patients have been allowed to lie quite flat and we have found that this fall of blood-pressure has been much less common and there have been no cases in which the stovaine has risen to a dangerous level.

Similarly in regard to the use of the Trendelenburg position it does not appear to be material whether the light or heavy solution is used. Generally I have employed the heavy solution as I found that the analgesia lasted longer with a smaller dose of stovaine.

When spinal anaesthesia was first employed the great merit claimed for it was that it did away with the necessity for general anaesthesia. But I think that as time has passed and experience been gained that opinion has been very much modified, so much so that all who have had any experience with this form of anaesthesia are agreed that the one great disadvantage of stovaine is that the conscious patient—the patient present at his own operation—outweighs many of the advantages of spinal anaesthesia, and nowadays it is rarely employed without either some modification of the far-famed "twilight sleep" or a little general anaesthetic. There is no doubt that a long operation in the Trendelenburg position or an operation on the rectum such as a combined abdominal perineal, or a Kraske’s excision are ordeals which few patients can face even if it were to their advantage to do so.

Speaking generally, for severe operations the method which has been employed has been to produce anaesthesia with ether, then to inject the stovaine, discontinue the ether for a time and then just to give a whiff to keep the patient unconscious.

For operations of a less severe type such as hernia or appendectomy, scopolamine and morphine are administered in the ward approximately.
an hour before the operation. The spinal injection is then given in the anaesthetic room before the patient reaches the operating table.

In this connexion I should like to mention a method for increasing the effect of scopolamine and morphine which was shown to me recently by Mr. Cole and which I have used since with very great success. After the administration of the scopolamine and morphine the patient's ears are plugged with cotton wool and the eyes covered with a bandage so as to more or less shut out the stimuli of light and sound. The increased effect of the scopolamine and morphine is most surprising and many patients who have been treated in this way although they have been lifted from the bed and carried upstairs, given an injection of stovaine and then been operated upon, have never known that they have left their room.

One of the results which we may hope to attain by a discussion of this sort is some idea of the safety of spinal as compared with other methods of producing anaesthesia, which is a very important point as, after all has been said, many of the indications for its use are relative and not absolute. Speaking from my own personal experience of about 8,000 cases I have had two deaths; one was a case of obstruction of the small intestine; the patient was very collapsed, there was profuse vomiting, and after the injection a flood of stercoraceous material escaped from the mouth. The patient was apparently asphyxiated. The respiratory passages were found to be full of vomited matter at the post-mortem.

The second case was that of a child of four years, more or less moribund, suffering from a gangrenous intussusception. Death occurred during the operation from circulatory failure and not apparently from any interference with the respiration, which continued for a short time after any sign of cardiac activity could be observed. Apparently the fall of blood-pressure caused by the stovaine, added to the shock already present, was sufficient to cause death. I think that in the light of further experience these were both instances of a mistaken choice of anaesthetic.

The above records include patients of all ages, from a few hours up to 80 years. The results obtained with young children were very satisfactory. Once the injection was given they generally passed into a somnolent condition and appeared to be in no way disturbed by the subsequent proceedings. The youngest infants were new-born babies, suffering from imperforate anus and hernia into the umbilical cord. It is interesting to note that a relatively larger dose of stovaine is
Section of Anaesthetics

required in infants than in adults to produce satisfactory anaesthesia. A dose of 2·5 cgr. of stovaine is required for the smallest babies and more for children of one or two years. Advanced age does not appear to be a contra-indication, and many of the patients included in this series were between 70 and 80 years. Elderly people are perhaps a little more liable to syncope if the anaesthesia reaches a high level. Although these were the only deaths I do not, of course, mean to say that there have been no complications. Those met with during the course of the anaesthesia have been three in number:

(1) Interference with the respiration, owing to the stovaine reaching too high a level.

(2) Complications due to fall of general blood-pressure, syncope, &c.

(3) Vomiting.

Difficulties due to the stovaine reaching too high a level have been very rare, generally occurring in children where the margin of safety is so much less, or the patients have been fixed in some form of splint, or were in such pain that it was difficult to get a proper position of the spine before injection. Usually this complication was quickly relieved by a little oxygen. Two patients did definitely and progressively stop breathing as the stovaine ascended. One most instructive incident occurred. The patient was a poor frail little boy who looked as if all his vitality had been sapped by the long strain of a suppurating tubercular hip. In spite of his condition, amputation through the hip-joint was decided upon. Great difficulty was experienced in getting the child into a proper position for the injection. The pain in his hip at the slightest movement as well as the rigid way in which he held himself, both combined to make the operation extremely difficult. The injection was complicated by something in the nature of a struggle, and the stovaine undoubtedly reached too high a level. The immediate relief of pain following the injection was most striking. Shortly after the commencement of the operation the intercostal muscles became paralysed; then very shortly afterwards the diaphragm also—the child became intensely pale, lost consciousness and ceased to breathe. Very gentle artificial respiration, by pressure with the hand on the front of the chest, was performed, oxygen administered, and the operation hurriedly completed. These efforts at resuscitation had been continued for about five minutes, when suddenly there was a slight movement of some of the muscles attached to the lower jaw, followed immediately by efforts at respiration first by the diaphragm, next by the intercostals. Then, with startling rapidity, the child completely recovered. Before
the last stitch had been put in, the little patient said that he had been to sleep. No shock followed the operation and the child made an uninterrupted recovery.

The majority of difficulties met with were due to a fall of blood-pressure, which varied from a slight pallor to a severe syncopal attack, with loss of consciousness and disappearance of the radial pulse. This complication was much more common in the earlier cases, before we realized that it was not necessary to keep the head and shoulders raised continuously. Only three patients stopped breathing from syncope. The sequence of events was the same in each case—sudden pallor, loss of consciousness, a few gasping breaths, then cessation of respiration. These cases also occurred in the earlier days, when the patients were propped up. As we did not like to lower the head in order to treat this condition we raised the legs and pressed upon the abdomen. Recovery in each case was as sudden as the onset, one patient again remarking that he had been to sleep. A certain amount of pallor and fall of the general blood-pressure occurred in about 30 per cent. of the earlier cases, but since the adoption of the recumbent position it has been much less frequent.

Vomiting occurring during the operation, seems to be more or less dependent upon the height of the anaesthesia. If the anaesthesia involved the dorsal cord, it was not uncommon, but very rare if the stovaine affected the lumbar and sacral plexuses only.

Without entering into the vexed question of the cause of the vomiting, whether it is subsequent to a fall of blood-pressure and more or less mechanical in origin, or is due to direct absorption of the drug, I think experience has shown that measures directed towards raising the general blood-pressure such as slight Trendelenburg position, elevation of the legs and pressure upon the abdomen much relieve this symptom.

Another question of perhaps hardly less importance than that of immediate safety is whether spinal is more prone to be followed by serious and unpleasant sequelae than other forms of anaesthesia. This, of course, could only be answered by time. I think that the length of our experience now justifies the expression of certain impressions and some definite statements being made upon this point.

Headache, vomiting and pulmonary complications have occasionally followed the administration of stovaine. Headache was not very common and then slight, but sometimes undoubtedly it was severe. My impression is that the headache was more common when the
patients were conscious during the operation, that is, before a general anaesthetic or scopolamine and morphia were used in combination with the stovaine. It also seems that if the patients were handled very gently after the operation and not jolted or shaken on the way back to bed, kept quiet afterwards and not allowed to talk, they were less liable to this symptom. One or two cases of severe headache were almost instantly relieved by lumbar puncture, and the withdrawal of about 20 c.c. of cerebro-spinal fluid, although this fluid did not appear to be under any abnormal tension. Post-anaesthetic vomiting, following stovaine, was very rare, and not prolonged in the few cases in which it did occur.

I think that there is evidence to show that spinal is much less frequently followed by pulmonary complications than any other method of anaesthesia. I have seen bronchitis and pneumonia both follow its use, as I have also seen these two complications follow after the use of local anaesthesia, and we must not forget that these are occasional complications in case of accidents, such as fractures, where no anaesthetic at all has ever been administered. It seems to me that respiratory complications under stovaine depended more upon the pathological conditions present, the condition of the patient and the type of operation performed.

Acute septic conditions, such as appendicitis and osteomyelitis, were generally present in those cases in which pneumonia followed the use of stovaine. Occasionally, there were pulmonary complications after operations upon the upper abdomen, which I presume were due to a reflex rigidity of the chest and insufficient expansion of the lungs, consequent upon the position of the abdominal incision.

One of the difficulties with which the pioneers of stovaine anaesthesia had to contend was the suggestion that permanent muscular paralysis might follow its use; and from time to time, cases in which there was some form of muscular paresis, loss of sphincter control, permanent anaesthesia and even complete paraplegia have been reported. I have been able to collect in all about 10,000 cases, of which I have actual personal knowledge. These include 400 cases which were done by the late Mr. A. E. Barker, which have not been published, but the records of which he gave to me, about 1,500 or 1,600 cases done at hospitals by the resident officers and about 8,000 done by myself. In not one of these cases has there been any permanent paralysis of muscles, or abolition of sensation, or any trophic lesions, with the exception of three cases of paralysis of the external rectus muscle of the eyeball, producing
Rood: The Present Position of Spinal Anaesthesia

diplopia, which lasted about three weeks. Many of these cases were done by people with no special skill, but the same technique was more or less followed in all cases, so that I cannot help feeling that those cases of permanent after-effects which are reported occasionally may be due to some error of technique. As far as I can gather, these permanent palsies have been more frequent when the puncture has been made very low down—viz., between the third and fourth lumbar vertebrae. Personally, I have generally made the injection between the eleventh and twelfth dorsal. It is generally easier, and if, directly the needle has passed through the supraspinous and interspinous ligaments, the stylet is removed and it is pushed on gently, it is difficult to see how the cord can be damaged, as directly the meninges are entered cerebro-spinal fluid appears, and, moreover, the peculiar sensation imparted to the fingers, as the meninges are punctured, is quite characteristic, much resembling the puncture of tense tissue paper.

It appears to me that the value of spinal anaesthesia is not, as I have previously remarked, that it abolishes the general anaesthetic. There are a few cases in which spinal anaesthesia presents great advantages over any form of general anaesthetic, such as in amputation for diabetic gangrene and for operations of emergency, which cannot be done under local anaesthesia in patients suffering from acute respiratory diseases. I have found spinal anaesthesia of special value for patients suffering from acute or chronic septic conditions with considerable toxæmia, such as acute appendicitis or osteomyelitis. Operations upon these cases are notoriously liable to be followed by disturbances of metabolism, leading to a general acid intoxication, and also to pulmonary complications. I think that the general opinion of those who have used spinal anaesthesia to any extent is that the results are on the whole better if general anaesthesia is not employed. Of course, as acidosis is already present in many of these patients before operation, and as the symptoms of acid intoxication following chloroform or ether (only very rarely after ether) are much the same as those produced by septic absorption, it is only possible to express a general impression of the value of stovaine in these conditions, after the experience of a considerable series. This is one of the points upon which we should much appreciate the opinion of our surgical friends.

But, undoubtedly, the great value of spinal anaesthesia is that it either abolishes or very much reduces the amount of shock associated with long surgical operations. This method of anaesthesia
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has been used nowadays extensively for many operations which are notoriously associated with shock, such as Wertheim's operation and various procedures for the removal of the rectum, and there seems to be a general consensus of opinion that the results in these cases have been much improved. Similarly, I consider that the more severe operations in young children, such as excisions and amputations, especially at the hip-joint, are much less formidable under spinal anaesthesia.

Lastly, stovaine produces absolute muscular relaxation. This, of course, much facilitates the performance of many operations, and renders long continued and forcible retraction unnecessary. It is hardly over stating the case to say that there are some operations, such as the radical cure of a large and irreducible hernia in a fat and muscular subject, which would be barely possible without its use. The slightest muscular rigidity makes the operation of prostatectomy very difficult. The complete muscular relaxation of spinal anaesthesia renders such great assistance that this operation is not often undertaken nowadays without its aid. Further, the amount of general anaesthetic necessary to produce muscular relaxation is very different to the amount necessary to produce loss of consciousness, so that by means of stovaine it is possible with a minimum of general anaesthetic to produce narcosis and complete muscular relaxation. Perhaps almost more important than the indications for the use of spinal anaesthesia are the contra-indications against its use.

These may be very shortly summed up if we say that spinal anaesthesia should never be administered to patients who are likely, from their condition, to be seriously affected by the fall of blood-pressure, which is so often associated with the use of stovaine. I believe that the experience of most operators in the military hospitals in France has been in accord with our experience in the civil hospitals at home; that spinal anaesthesia is absolutely dangerous for patients suffering from profound shock, and I believe that in most cases, after a short trial, it was given up. Of course, in the base hospitals, there has been more scope for its use, as the conditions more or less approximate to those of civil hospitals, and severe operations are undertaken upon patients who are in comparatively good condition at the time. Therefore, it seems to me that it should be clearly emphasized that spinal anaesthesia protects patients from the onset of shock due to severe and prolonged surgical procedures, but should never be administered to patients who are suffering from shock at the time.

The impression that spinal anaesthesia is a substitute where the
patient is supposed to be too ill to stand a general anaesthetic has been responsible for many of the reported fatalities. It is difficult to generalize as to the value of spinal anaesthesia in heart disease—in mitral disease with much pulmonary congestion it is sometimes very useful, but certainly never in aortic disease, or in any other cardiac or vascular condition in which the patients are prone to syncope. A problem which has often to be decided is whether the dangers associated with an immediate fall of blood-pressure outweigh the benefit to be obtained from stovaine. This is especially the case in acute abdominal surgery. Here the advantages of stovaine are well known; the muscular relaxation, the ease with which the whole abdomen can be explored and the consequent shortening of the operation, and the diminution of the shock, which is so often associated with manipulation of the intestines. But if the patient is much shocked and almost in extremis from long-continued obstruction, stovaine should be used only with the greatest caution. Each case of intestinal obstruction must be judged upon its own merits; some of the most brilliant results of spinal anaesthesia have been obtained in this field of surgery, but I think that a routine practice of using this method for all cases of intestinal obstruction, irrespective of the condition of the patient, is only courting disaster.

Finally, I should like to say a word in regard to the use of stovaine in hospitals, as one of the routine methods of producing anaesthesia—I mean, in those cases in which no definite indication for its use exists, but where anaesthesia merely is required for such operations as appendectomy, hernia, varicose veins and so on. It is obviously an advantage to gain experience of this method so that when those cases do occur in which the special indications for its use are apparent it is not in the nature of an experiment. It has been urged against this view that spinal anaesthesia is not so safe as other methods, but I do not think that, given the ordinary care and skill, the facts warrant this conclusion. I have never seen a death from spinal anaesthesia administered for any simple operation, and I believe that many lives have been saved by its use when occasion requires, and if it is one of the methods in daily use it is more likely to be selected and skilfully administered.

So that I think we may say that spinal anaesthesia is now long past the experimental stage. In summary, it may be said that—

(1) It is one of the recognized means of producing anaesthesia.

(2) It is not a universal anaesthetic to be applied to all cases.
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(3) It has its special dangers.
(4) It has its special merits, but used in its proper sphere it is a very valuable method of producing surgical anaesthesia.

Mr. J. P. Lockhart-Mummery.

If we consider an operation from the physiological aspect—as we always should—the object of those performing it, or assisting at it, should be so to arrange matters that the patient may lose the minimum amount of vital energy. Every patient may be considered as having a certain total amount of vital energy, part of which is immediately available, and part of which is stored up in reserve. A young man in good health will have a considerable amount of both, but a patient who is ill, especially if he is also old, may have very little of either. It frequently happens that it is necessary to operate upon a patient who has used up his reserve of energy, and who has nothing more to draw upon when that immediately available is used up (for instance a case of intestinal obstruction in an elderly person). An operation on such a patient will be a critical affair, and there is no margin to work with. The operation should therefore use up the very minimum of vital energy, and we must if possible begin at once to build up a reserve of energy for the patient by feeding. There are three factors to consider in choosing an anaesthetic:

1. The absence of vomiting. This is important (a) because vomiting prevents us from feeding the patient at the time when food is most required, that is, immediately after the operation; (b) because it often seriously interferes mechanically with the surgeon's handiwork, and (c) because in itself it causes loss of vital energy.

2. Complete protection of the patient against impulses from the field of operation. We know that no form of inhalation anaesthesia answers this requirement.

3. Protection of the patient against mental anxiety or fear. This is exceedingly important, and it is not possible under any form of local or regional anaesthesia at present known.

Clearly the ideal must be sought in a combination of local and general anaesthesia. My present belief is that the best combination is spinal or regional anaesthesia and morphia and scopolamine, or where the latter is contra-indicated, gas and oxygen, or all three. Spinal anaesthesia alone is not a complete anaesthetic as it leaves the brain
exposed to emotional disturbances which may often be very considerable. The objection which surgeons had to the administration of morphia in abdominal cases has been rendered invalid by the discovery of pituitary extract; the only disadvantage of morphia is that it may disturb digestion. (One of the great advantages of spinal anaesthesia is that there need be no interruption of the patient's normal meals.) The preliminary morphia treatment should be commenced an hour or two before operation, and should be sufficient to secure a condition of complete mental repose, which, it must be admitted, is at present often very difficult to gauge accurately. Careful stage management is also necessary, and on no account should the patient enter the theatre till the anaesthetist is quite ready to give the injection; there should be no fussing about, and no talking or noise.

I have seen very few cases of bad "after-effects" from spinal anaesthesia, the absence of which I attribute to great care in moving the patient after the operation. I have had two cases of neuralgia, one in the leg and one in the arm. So convinced am I of the value of spinal and regional anaesthesia that I believe inhalation anaesthesia with ether and chloroform will in a few years only be used in remote villages and for veterinary purposes.

Mr. Percival P. Cole.

I have been using spinal anaesthesia for six years in every variety of case. At first the glucose solution was employed, but this has been discarded in favour of the sodium chloride solution, the use of which allows the patient to be placed in the Trendelenburg position at once, and in cases in which this is not necessary the feet should always be kept higher than the head both during and after operation. This posture does much to abolish the effects of diminished blood-pressure and it was this observation that decided me to abandon the glucose solution. The case of a woman who was admitted to the casualty department of Queen Mary's Hospital for the East End is a tribute to its value in shock. She was apparently moribund, pulse 160, barely perceptible, abdomen distended and obviously suffering from diffuse peritonitis. Under spinal anesthesia the abdomen was opened as soon as she could be transferred to the operating theatre. Flaky lymph and pockets of pus were present everywhere from liver to pelvis, the result of a ruptured pyosalpinx. Subcutaneous saline was administered, the abdomen drained above the pubis and in both flanks. When she left
the table her condition was considerably improved and she slowly but completely recovered. The use of spinal anaesthesia is strongly indicated in the acute abdomen, for it is not only of great assistance to the operator but has a marked influence on peristalsis. The bowels have frequently been observed to act on the table in cases in which the intestines were enormously distended as a result of septic spreading peritonitis. Vomiting is very rare after spinal anaesthesia—a fact that has convinced me that it is the anaesthetic of choice in cases of umbilical hernia in fat women. In prostatic and urethral operations it is invaluable, and although not posing as an expert I have never encountered an impassable stricture since the routine adoption of spinal anaesthesia for such cases. My experience is that sodium chloride solution guarantees as lasting an anaesthesia as does glucose solution. To-day, for instance, I have resected a transverse colon and anastomosed the cut ends of the bowel without the assistance of any general anaesthetic. The operation lasted fifty-five minutes and perfect flaccidity was maintained till the end. During the War it has been particularly useful for it has enabled one to deal with acute abdomens single-handed—a great advantage in view of the difficulty of obtaining assistance. Unfounded enthusiasm is as much to be deprecated as prejudiced objection, but Dr. Rood has presented the case for spinal anaesthesia in a well-balanced and moderate manner.

Dr. F. F. Shipway.

I should like to ask Dr. Rood why he calls the stovaine-glucose solution the "heavy" solution and stovaine-saline the "light" solution. As a matter of fact the specific gravity of the former is 1.023, and of the latter 1.083. Owing to this mistake the saline solution is used as a "light" solution and the patient placed flat or in the head-down position very soon after injection. The result is that the fall of blood-pressure is largely compensated by gravity, and the stovaine-saline solution is looked upon as safer than stovaine-glucose, in which the head and shoulders are usually kept raised. I think that diffusion plays a very small part in producing serious symptoms. Faintness, pallor, vomiting, collapse are due to the large fall of blood-pressure, which results from the impairment of thoracic breathing and the paralysis of abdominal and skeletal muscles. The tone of the abdominal muscles, and of the muscles of the trunk (below the diaphragm), and of the muscles of the lower limbs, plays a very large part in maintaining
blood-pressure. It is known that blood-pressure may fall as much as 50 mm. in about ten minutes after injection. Such a large fall is often dangerous. My feeling is that blood-pressure readings should be taken much more frequently; they help one to select suitable cases and to form an accurate prognosis. I have been told that headache is more common in women. Is this Dr. Rood's experience, and does he think it is a nervous phenomenon?

Dr. Blomfield.

Looking back on the discussion of spinal analgesia which took place in its early days and comparing that discussion with the present, I am struck by two important features brought out by Dr. Rood which were scarcely perceptible in the earlier discussion. These are, first, the possibility of using spinal analgesia with patients in the Trendelenburg position, and secondly the advantage of combining spinal with general anesthesia. Concerning the latter I think that both Crile and Mr. Mummery under-rate the powers of inhalation anesthesia to prevent shock. Surely our everyday experience shows us that shock from operative procedures is effectively prevented by proper inhalation anesthesia and the physiologists themselves have shown how difficult it is to produce shock in an etherized animal unless the abdomen is open and viscera forcibly manipulated. Nevertheless the additional advantage of spinal analgesia is very great in selected cases and in such an instance as recently occurred, when a patient with active phthisis on one side of the chest, an artificially produced pneumothorax on the other, and a fulminating attack of appendicitis, had to undergo laparotomy. This could scarcely have been performed, and certainly not with the success that followed, except under the use of spinal analgesia reinforced with the minimum of inhalation anesthesia, in this case induced by chloroform and oxygen.

Dr. Rood (in reply).

I am very interested to hear from Dr. Shipway that the specific gravity of the saline-stovaine solution is greater than that of the glucose solution—I did not know it. The reason I used the term "heavy" is because I find the glucose solution is more easily and definitely localized by gravity than the saline. I have not noticed that headache is more common in women.
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President—Dr. Llewelyn Powell.

Successful Massage in a Case of Heart Failure due to Shock.

By Ashley Daly.

Successful cases of heart massage are now fairly numerous, and there is no doubt that, in certain cases of heart failure during anaesthesia, massage of the heart is the only treatment which will save the life of the patient. The following case seems worth recording as the heart failure was due to shock and did not appear to be caused by the anaesthetic. Although the patient eventually died, I think I am justified in calling it successful as far as the heart massage was concerned.

The patient was a private, aged about 25. He was in hospital at Étaples for some slight skin trouble, which was cured, and he was nearly due to return to his battalion when, during a night air-raid, he was struck in the abdomen and left thigh by portions of a bomb and sustained very severe injuries. At the end of the raid he was taken to the operating theatre, where I saw him. He had then been given morphia, $\frac{1}{4}$ gr., and atropine, 10,0 gr. His condition was extremely bad; he was cold and pale, the pulse was small, and of low tension.

He had a penetrating wound of the abdomen and a compound fracture high up in the left femur with much comminution of bone and extensive laceration of the soft parts on the outer side of the thigh. It was not thought that he would stand an operation, so it was decided to wait for a time to see if his condition improved. No

1 At a meeting of the Section, held April 4, 1919.
improvement took place, and after about three-quarters of an hour, as his thigh wound was bleeding somewhat freely, it was decided to operate. I chose as an anaesthetic warm ether vapour with oxygen through Shipway's inhaler. Induction was quiet and uneventful.

Lieutenant-Colonel Seymour Barling opened the abdomen and another surgeon dealt with the thigh wound. While the abdomen was being explored the respiration and pulse became more and more feeble and soon both ceased; pallor became extreme, the eyelids separated, and I informed Colonel Barling that the patient was dead. He was about to close the abdomen, but at my request he tried heart massage. This was performed through the diaphragm, and at first no contraction was felt; the heart was gently squeezed, and after about two minutes contraction was felt, slight at first but increasing in strength. Artificial respiration was at once started; oxygen being freely given. Automatic respiration soon started, and the operation was proceeded with. Four perforations of the intestine were sewn up, the thigh wound excised, the bleeding stopped, and a Thomas's splint applied; during this time anaesthesia was maintained by warm ether and oxygen.

Saline solution was given intravenously, and the patient returned to bed. The next morning his condition had improved slightly, but he collapsed again later in the day and died in the evening, about twenty-two hours after the operation. At that time blood transfusion was not being used, and the only apparatus available for nitrous oxide and oxygen was Hewitt's, with double bag.

From subsequent experience I think there is no doubt that the patient would have had a better chance of recovery if blood transfusion had been carried out, as he appeared to be suffering from both haemorrhage and shock, but as regards the question of anaesthetic I am not so sure.

Nitrous oxide and oxygen is of great value in military surgery in patients suffering from shock due to injuries other than abdominal, but I am inclined to think that warm ether and oxygen, with Shipway's inhaler, is to be preferred in abdominal cases. Even with the excellent apparatus invented by Mr. Boyle there is often difficulty in obtaining relaxation without giving ether fairly freely, and this being the case, I think oxygen given through ether and the vapour warmed is a better vehicle for ether than nitrous oxide and oxygen with re-breathing.
Case of Successful Heart Massage.

By Llewelyn Powell, M.B. (President).

During an exploratory laparotomy in a middle-aged man with very rigid abdominal muscles, ether failing to produce relaxation, C.E. mixture was pushed and cardiac failure ensued. Artificial respiration was carried on for two minutes, but it failed to resuscitate the patient owing to rigidity of the chest wall. The surgeon therefore enlarged the abdominal incision, and, passing his hand up under the diaphragm, massaged the heart, which was completely flaccid. It recommenced to beat immediately.

Case of Sudden Collapse during Laparotomy under Ether; Treatment by Heart Massage,

By F. E. Shipway, M.D.

I have had a case of sudden collapse during laparotomy under ether. The patient was a man aged 60, of poor physique, thin and feeble. His condition was entirely satisfactory during and up to the opening of the peritoneum, but when the stomach was pulled out of the wound he collapsed, respiration quickly failing and the pulse at the face going. Lowering the head, squeezing the chest, and artificial respiration were tried, but the pupil continued to dilate; the heart beat could not be felt. Massage of the heart through the diaphragm was quickly successful.

Notes of a Case of Heart Failure following Change of Position.

By R. E. Apperly.

Patient was a stout woman, aged 37, in whom laparotomy was being performed for pelvic inflammation. Anaesthesia was induced with gas and ether, followed by open ether. After the abdomen had
been opened, the table was tilted into the Trendelenburg position; the pupils immediately dilated widely, the face became pale, and pulse and breathing stopped. No pulsation could be felt in the abdominal aorta. The surgeon squeezed the flaccid heart through the diaphragm, and at the same time the table was put into the horizontal position; in about one and a half minutes the heart began to beat again. Towards the end of the operation the table was again tilted, and at once the pulse became irregular: the horizontal position was resumed and the pulse again became normal.

Case of Tonsillectomy in a Man weighing 23 st.

By F. E. Shipway, M.D.

In April, 1917, I was asked by Dr. Herbert French to examine a patient of his and to decide whether I would give him an anaesthetic for the enucleation of a septic tonsil. He was a man aged 52, and weighed a little over 23 st. His history was that he was very anxious to have the tonsil removed, but that he had seen several consultants who had either refused to do the operation or had told him that he was not a fit subject for an anaesthetic. I found him to be a highly intelligent and sensible man, but very restless and nervous, of huge size, with a very short thick neck, much fat round the chin and angles of the jaws, a perfect set of teeth, and very large abdomen. His movements were fairly brisk, and he told me that he was healthy, shot a good deal, was a careful liver and moderate smoker. He usually slept in a chair. The throat was irritable. The pulse was steady and full, arteries good, systolic blood-pressure 130; heart-sounds rather faint, but nothing abnormal detected, and lungs healthy. There was no albumen nor sugar in the urine.

Taking all these facts into consideration and realizing his keen desire to have the operation and his willingness to run the risk, I consented to give him an anaesthetic, as I felt that in spite of his great bulk and of the nature of the operation he was a healthy vigorous man with sound constitution and certainly a good cardiac muscle; moreover, he was likely to be a helpful patient. At the same time I was not unnaturally a little anxious as to possible complications; for example, jaw-spasm might be troublesome, bleeding might be free, and the likelihood of relieving a severe asphyxial condition by tracheotomy
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seemed to be remote. However, I assured him that he was a fit subject for the anaesthetic, and it was agreed that in order to avoid the risk of bronchitis the operation should be postponed three weeks—i.e., until May, when the weather would be warmer, and that in the meantime he should take regular exercise, knock off smoking altogether or reduce it to one cigar a day, and cut down alcohol to one whisky and soda a day. When he went into the nursing home he was in very good condition, much less nervous, and said he had never felt fitter.

Mr. Tilley and I had previously discussed the question of posture and anaesthetic and had decided that an attempt should be made to enucleate the tonsil under deep anaesthesia with a pillow under the shoulders, the head hyperextended, and the mouth widely open. The night before the operation the patient was given 15 gr. of bromide, and next morning, three-quarters of an hour before the anaesthetic, $\frac{1}{4}$ gr. morphine and $\frac{1}{10}$ gr. atropine. The effect of the morphine was good: the patient was calm, and with the aid of three pillows was made comfortable, in the semi-recumbent position. Induction was started with C.F. Consciousness was quickly lost, and as soon as the stage of excitement commenced open ether was given. This stage was not severe, and did not last long: it was characterized by a complete absence of struggling, slight raising of the head and shoulders, and some rigidity and spasm of the muscles of the jaw and neck. Respiration became noisy and the colour slightly cyanotic: this was quickly relieved by oxygen, and the administration was continued until the mouth could be opened by a wedge and a gag inserted. This was done with some difficulty, two of the pillows were removed, the tongue was pulled forward and a ligature passed through it. Whillis's gag was then substituted for the Mason gag. A sterile rubber catheter attached to the warm-ether apparatus was pushed down one nostril and a mixture of oxygen and chloroform, with a little ether, administered. Anaesthesia soon became moderately deep and of good quality; the last pillow was removed and the head was bent back as much as possible. The breathing at once became much embarrassed, and in spite of tongue-traction and of the jaw being held forward by a finger in the mouth it was obvious that the operation could not be done in this position. The head was therefore brought into line with the body and the breathing immediately became quiet and easy and slow. Deep anaesthesia was then established, chiefly by means of chloroform: the eyes were fixed, pupils small, corneal reflex moderately brisk, pulse slow, regular, and of good tension, and the colour excellent. Pulling on the tonsil evoked no reflexes, and enucleation was
successfully performed. Fortunately there was very little bleeding. Reflexes soon returned and the patient made a very good recovery.

I have reported this case in full because it was very unusual, and because it shows the importance of the opinion of the anaesthetist being taken in any case where there is any doubt as to the desirability or possibility of an anaesthetic being administered.

Mr. Herbert Tilley: Dr. Shipway has not in the least degree exaggerated the difficulties which my patient presented; nevertheless, I felt that if efficient general anaesthesia could be induced, there should be no particular difficulty in enucleating the tonsil by dissection. This proved to be the case, for the narcosis was so complete and satisfactory that the septic tonsil was removed in its capsule with no more than the usual amount of bleeding, and the operation did not last ten minutes. During the first two days after the operation the patient felt very "depressed" and "faint." His pulse was rapid, small, and very irregular. I ordered him a half bottle of champagne with his lunch and dinner, and in the intervals, hypodermic injections of strychnine and digitalin. On the third day all the symptoms diminished and the patient made an uninterrupted recovery. With regard to the choice of anaesthetic in the enucleation of tonsils, I am convinced, from a considerable experience of this operation, that the safest and most satisfactory method of induction is by open ether preceded by a hypodermic injection of \( \frac{1}{100} \) gr. of atropine given about forty minutes before the operation. When the patient is fully under ether and the breathing is regular and automatic, then the gag is inserted, the operation is commenced and anaesthesia maintained by chloroform from a Junker's apparatus. I was asked just now why I preferred "ether to chloroform." My reply is, that I have seen eleven deaths under anaesthesia induced by chloroform—three of these in my own practice, but I have never seen a death under ether when administered in the manner just described. In enucleation of the tonsil by dissection—the most surgical and scientific method—deep anaesthesia is necessary, and surely it is better to secure this by an anaesthetic which stimulates the heart rather than by one which acts as a cardiac or respiratory depressant.

**Case of Laryngofissure with Removal of Intralaryngeal Growth performed under Gas and Oxygen.**

By H. E. G. Boyle. M.R.C.S., L.R.C.P.

The patient was a man aged about 50. He was given morphia \( \frac{1}{2} \) gr. and atropine \( \frac{1}{100} \) gr. hypodermically half an hour before the operation. He was anaesthetized with gas and oxygen with regulated rebreathing.
The operation proceeded, and just before the larynx was split a tracheotomy was done, and into the tracheotomy tube I inserted a small catheter, through which I ran the gas and oxygen. As I was afraid that this would not be enough to keep him "under," I turned on the ether tap of my machine; the patient at once coughed, so I turned off the ether and continued with gas and oxygen alone. The operation proceeded with the patient perfectly quiet and breathing as though asleep.

At the end of the operation the patient was returned to bed with the tracheotomy tube in situ, and when seen ten minutes afterwards he signified by nods of his head that he was comfortable and in no pain.

This case is an illustration of what can be done with gas and oxygen, and it has led me to continue my efforts to perfect this method for other throat work. I find that my best results for nose and throat work are obtained by using gas and oxygen in combination with a C.E. mixture. How far I shall be able to develop the method and reduce the C.E. mixture it is impossible to say—time alone will show.

I agree with Mr. Daly that perfect relaxation of the abdominal wall is very difficult to obtain with gas and oxygen alone, but if the gas and oxygen is used in combination with a very little C.E. mixture then quite a satisfactory amount of relaxation can be obtained, at any rate for the lower abdomen. The upper abdomen is another matter; here operations can be done perfectly well with the gas-oxygen-C.E. combination, but I think that far better results are likely to be obtained if the surgeon will co-operate with the anaesthetist and inject some novocain into the muscles. This injection, or infiltration, is a perfectly simple proceeding, and only adds about three minutes to the length of the operation.

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Case of Caesarean Section under Spinal Anaesthesia.

By Llewelyn Powell, M.B. (President).

I was asked to go to Queen Charlotte's Hospital to see a patient of Mr. Clifford White's. She was a primipara who was ill with influenza and double pneumonia, and labour had just started. Mr. Clifford White thought she would not survive a normal labour,
and suggested Caesarean section under spinal anaesthesia. She had a "twilight sleep" injection, and was brought into the theatre in a drowsy condition. I gave her an intrathecal injection of stovaine. Her ears were plugged and her eyes covered, and she went to sleep. The operation lasted about twenty minutes. The patient slept throughout the operation, and only awoke on being moved from the operation table. She made an excellent recovery, and the child also did quite well. Seeing how exceedingly ill this patient was, I do not think any other procedure would have given her such a good chance.

Case of Abdominal Section in a Man with a Thoracic Aneurysm.

By F. E. Shipway, M.D.

Case showing the value of oil-ether anaesthesia. The patient was a man, aged 52, who had carcinoma of the pylorus, and had been the subject of aortic aneurysm for four years. He was a thin, nervous, excitable man, had had dyspepsia for ten months, and towards the latter part of this time had been vomiting large quantities of fluid about once a week. Wishing to have relief he consulted a physician, but was advised that nothing surgical could be done as he was not a fit subject for a general anaesthetic. Two months later his condition was so miserable, and he was going downhill so rapidly from starvation, that the question of operation again arose. It was decided to do a gastro-jejunostomy. It was felt by Mr. Burghard and by the patient's doctor, Dr. Harry Cooper, of Surbiton, that gastro-jejunostomy would give him relief for several months, and that as he was able, in spite of his aneurysm, to do his ordinary business with care, a palliative operation was not only justified but would be feasible. The selection of the anaesthetic and of the method of administration was not easy, especially as it was not possible for me to see the patient.

I learnt that the aneurysm formed a large swelling with some prominence over the first, second, and third ribs, with pulsation in the second right intercostal space. The apex beat was in the fifth space just internal to the upper line. There were loud systolic and diastolic aortic murmurs. The pulse was of the water-hammer type, and varied between 80 and 120; it was usually 80. The left recurrent laryngeal
nerve and the left cord were paralysed. The chest was emphysematous and there was some deficiency of respiratory murmur into the upper part of the left upper lobe. I came to the conclusion after consultation with Dr. Cooper, that with ordinary care the patient would take an anaesthetic if the induction stage, which was likely to be the most dangerous, could be passed successfully, and that the best method of tackling this problem was to give morphia and atropine and a small dose of ether in oil per rectum, the administration being continued with warm C.E. vapour and oxygen. This was explained to the patient, who agreed to the rectal injection: accordingly it was arranged that he should go to bed two days before the operation, and on both days the rectum should be washed out and half a pint of saline should be run in slowly, in order to establish tolerance.

On the morning of the operation, which took place in the afternoon, the stomach was washed out: three-quarters of an hour before operation a hypodermic injection of $\frac{1}{2}$ gr. of morphine and $\frac{1}{100}$ gr. of atropine was given, and twenty-five minutes later a rectal injection of 3 oz. ether and 1 oz. of olive oil. This was well borne, and consciousness was lost without any excitement or struggling. On arriving at the nursing home shortly afterwards it was found that the patient was fast asleep, breathing easily and quietly; he was completely unconscious, and the conjunctival reflex was absent, but anaesthesia was still light. He was carried into the theatre on a stretcher, but unfortunately the movements made him vomit slightly and return some of the oil-ether mixture. This did not interfere with the subsequent administration of warm C.E. vapour and oxygen, anaesthesia being straightforward and maintained at a moderate depth; corneal reflex was brisk and pupils were small. Respiration was quiet, colour good, and the pulse averaged 88 to 92, rising to 96 when the stomach was pulled down. Relaxation was complete. Very little anaesthetic was needed in addition to the rectal injection. At the end of the operation, which lasted sixty-two minutes, the bowel was washed out and half a pint of saline left in. There was very little shock.

Dr. Cooper wrote to me a week later saying: "Mr. —— is splendid. He was a little sick twice after being put back to bed, before he had regained consciousness. Since then he has not 'looked back,' and he is very grateful for such a merciful anaesthetic. His heart and aneurysm do not appear to have suffered at all; his pulse-rate has kept about 80, which is normal for him."

MY—1x
At the time of writing (April, 1919), patient was still alive—i.e., eighteen months after the operation—having kept well until quite recently, when he began to show signs of secondary deposits in the liver.

Mr. E. G. Slesinger, F.R.C.S., exhibited a pneumatic injector for local anaesthetics.
Section of Balneology and Climatology.

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Immunity and Mineral Water Treatment. (With an Introductory Note by R. Fortescue Fox, M.D.) ... ... ... ... ... 1
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GREAT TITCHFIELD STREET, OXFORD STREET, W. 1.
Immunity and Mineral Water Treatment.

By Dr. Paul Ferreyrolles.
(Médecin consultant à La Bourboule.)

(WITH AN INTRODUCTORY NOTE BY R. FORTESCUE FOX, M.D.)

I have much pleasure in presenting an experimental study by Dr. Paul Ferreyrolles on the power of certain medicinal waters to confer immunity against infective disease.

Many facts pointing in this direction have long been familiar to hydrologists, although unfamiliar to the profession at large. Dr. Ferreyrolles' thesis is welcome, as it is the most complete series of observations as yet made upon this subject, and its publication in England is opportune and cannot fail to stimulate other workers. More than one class of natural waters are now known to possess these antitoxic properties, and it will be a comparatively simple matter to institute experiments in order to ascertain whether muriated or sulphur waters such as those of Llandrindod or Strathpeffer, have the same power.

It is unnecessary to emphasize the extraordinary clinical interest that attaches to these investigations.

Dr. Ferreyrolles' demonstration a few years ago before the Royal Society of Medicine, upon the colloidal constituents of medicinal springs [4], followed by the present study on immunity, mark a new departure in hydrological medicine. They show incidentally that hydrology is not concerned only with the actions of water per se and with thermal actions

1 Presented at a meeting of the Section, held May 28, 1919, and taken as read.

AU—2
however important, but includes a study of the actions and uses of the particular constituents of the different waters, many of which, although present in infinitesimal quantities, we are now led to believe, may really be of great and even specific value.

Treatment by "mineral waters," as Tardieu said, "must be considered as the great school of natural medicine, the greatest clinic of those chronic diseases which settle, as it were, in the roots of the constitution, and can only be expelled by the mysterious and powerful action of mineral waters." Action, indeed powerful and mysterious, which for many years we have been trying to understand, in applying to the study of mineral waters the latest results of physical, chemical, biological, and physiological sciences; but how many undiscovered points remain which make it impossible at present to solve the problem of the specialization of each mineral spring! We shall arrive at it some day, no doubt. But since it is impossible at present to catalogue, so to speak, the diseases curable by such or such a mineral water, we must try to define more carefully the morbid type, whatever its manifestation, with which each mineral water agrees.

It is upon the morbid type only that certain mineral waters act, not only in modifying what everybody nowadays agrees to call "le terrain," but even in modifying the temperament of those persons having this morbid type. The resistance ought always to be the same, whatever may be the invading agent. It must be helped if it exists, created if it is lacking, so producing such a modification of "le terrain" and of the temperament as shall guard the patient against further morbid manifestations, and shall render him immune against his outer enemies.

These facts are known: they have been verified under the influence of the same mineral water on different patients, and on the same patient under the influence of different mineral waters without a satisfactory explanation. Sceptics have smiled, explaining the success of the thermal cure as being due to rest, diet, altitude and open-air life, whereas it really is a question of a deep-seated, extremely important action of the hydro-mineral medication—an action concerning which we propose to bring forward some striking points.

In a previous and more complete essay, I have shown, in collaboration with Professor Billard [1], when dealing with the waters of Choussy-Perrière at Bourboule, that they might be considered as a
true medicated arsenical serum, with a total mineralization of 6 grm. 4,997 per litre, containing 0·007 grm. of arsenic per litre. The density of this water is at 35° C., 1050, its cryoscopic point is $\Delta = 0\cdot33$, and its electric conductivity $67 \times 10^{-4}$. Laborde has stated that 10 litres of this water contained, four days after being taken from the spring, 1·78 mg. of radium emanation, and that the gases of the spring correspond to 11·02 mg. minutes, of emanation of radium.

When examined by the ultra-microscope [2] they show crystals, crystalloids, and electro-negative colloids, which we believe to be arsenic colloids, relying in this opinion upon the resemblance which exists between the colloidal arsenic obtained by the electrolytic method and in the Choussy-Perrière water, upon the same electric sign, the same toxicity (sfebler as per equal content of arsenic than hectine and 606), the same catalytic power, bactericidal action [3], &c.

We have studied its physiological and therapeutic action, both local and general, in hypodermic injections. It is their general action, especially in the administration by hypodermic injections, to which we propose to call attention—namely, the modification of the leucocytosis reaction and the anaphylactic phenomena under the influence of very small doses of this water, indeed of infinitesimal doses, and consequently of their different constituent agents.

We are perfectly aware of the insufficient number of our experiments, and how they are open to criticism. Professor Richet and M. Bellin have made similar experiments on immunity with all the necessary scientific controls. We have founded our experiments upon theirs. Our results have been found analogous. It is because of this fact that our experiments have remained limited. We publish them, then, because they seem to us very interesting, reserving for a future and more complete essay the indication of certain points in greater precision.

**Leucocytosis Reaction.**

The leucocytosis reaction may be defined as the variation of the number of white globules of the blood according to certain physiological or pathological influences. A toxin, a diet, a haemorrhage, a traumatism act on leucocytes and modify the number of those which can be seen in the unit of volume of circulating blood. We shall not dwell on the cause of this variation, contenting ourselves with seeing what happens under the influence of subcutaneous injections of a mineral water.
Influence of the External Cure.—Claisse was the first to notice this action of mineral waters. He has shown at Biarritz that the bath of strong chloride of soda lowered the number of the white cells, as well in the normal subject as in those with local tuberculosis.

Influence of the Administration of the Mineral Waters per os.— From observations made at Vichy, Salignat and Leger have shown the alternation of hypoleucocytosis and hyperleucocytosis under the influence of the thermal cure. They have seen polynucleosis disappear under the Vichy cure. The leucocytosis modification always leaned in the direction of mononucleosis, and eosinophils frequently appeared at the end of the cure. In the case of the water of Ahusquy, Dr. Feillé, too, has observed phases of hypo- and hyperleucocytosis. Thus following a thermal cure, internal or external, there is an irregular leucocytosis reaction, and last of all a hyperleucocytosis, but very slight in every case.

Influence of the Cure by Hypodermic Injections.—What happens under the influence of an injection of 1 c.c. of Choussy-Perrière water? The counting of leucocytes has been made by the very exact method of Lassablière. For simplicity's sake we shall refer to, as he does, in our figures, not cubic millimetres, but the one-hundredth of a cubic millimetre. Supposing, for example, that 80 is the number found for a normal animal. After leucocytosis reaction, we find say 150. We do not write 150, but the half of 150 plus the half of the average between 80 and 150, that is to say, \(115 \div 2 = 57\). The number is therefore 75 + 57 = 132, which represents one half of the absolute number 150, and half of the ratio between the absolute number and the number stated previously for the same animal. That will permit the comparison of the reaction of two animals having the same absolute figure, though not having the same initial number.

Intraperitoneal Injection and Leucocytosis Reaction.

If a very small dose of Choussy-Perrière water be injected into the peritoneum of a guinea-pig or a rabbit a clear leucocytosis reaction will be found to have taken place. Three guinea-pigs giving successive reactions of 75-80-79 white globules, five hours after an injection of 1 c.c. of Choussy-Perrière water, that is of 0'006 of its total mineralization, exhibited a blood count of 178-158-147. This reaction has been about the same in three guinea-pigs which received 5 c.c. of water, a dose five times stronger. Thus, for the water of La Bourboule, the leucocytosis reaction following an injection is very
pronounced, and seems in a large measure independent of the dose injected.

These results can absolutely be compared to those obtained by Professor Richet, who had studied the leucocytosis reaction of a great number of animals after the injection of different doses of NaCl or of a solution of sterilized peptone and has concluded: "Following these experiments, the very important conclusion, from the therapeutic point of view, is arrived at that too strong doses are used for injections. This efficiency of small doses of solution causes us to reflect. It is quite the same, whether one injects 1 c.c. of plasma or a liquid containing only 1 in 10,000 of muscular plasma."

**Leucocytosis Immunity after Peritoneal Injection.**—If after fifteen days we give those guinea-pigs a second injection of Choussy-Perrière, it is found that those animals which had reacted very strongly at the first injection do not react at the second, and pass from 178-158-147 to 145-114-135; in other words, they have obtained their leucocytosis immunity for the injection of this water. Thus, these guinea-pigs, after intraperitoneal injections of the water of La Bourboule, are immune against this water, and this immunity, as many observations show, requires a fortnight to become established. Neither at the first nor at the second injection has any serious symptom appeared, and had we not noticed the difference between the first counts, we should be tempted to say that the first injection had been without effect and that the second also had been without effect. If we refer to the experiments of Professor Richet, we arrive at the same conclusion: "To produce this immunity fifteen days afterwards, exceedingly minute doses were sufficient, doses so minute that it was long before I believed it, but after repeating my experiments again and again, I had to give in to the evidence. Perhaps physicians will here find material for reflection, and will try to obtain wide reaching effects with small means."

**Leucocytosis and Intravenous Reaction.**

We noticed in a normal series of rabbits that following intravenous injections of Choussy-Perrière water in the marginal vein of the ear, we obtained a leucocytosis reaction the same as that which followed the intraperitoneal injection. Fifteen days afterwards we give our animals a fresh injection of Choussy-Perrière: we obtain no reaction. We may then be allowed to say that as in the preceding case we have obtained a consecutive immunity.

Professor Richet has shown that the intravenous injection of
7 in 1,000 solution of chloride of sodium in a large dose produces a hyperleucocytosis which lasts sometimes six months, and if small doses are used the number of leucocytes becomes normal after about ten days.

**Injection of Mineral Water Followed by Injections of Toxins.**

Now, let us suppose that after fifteen days we inject into those animals which have received a first dose of Choussy-Perrière water, a dose of toxin of any kind. What is going to happen? We have injected into our guinea-pigs a dose of 1 c.c. of typhoid toxin prepared according to the method of Besredka and we notice that their leucocytosis number which, after the first injection was 178-158-147, and 145-114-135 after the second, was 137-92-95 after the injection of typhoid toxin. Ten days later they receive a dose of 1 c.c. of bouillon culture of coli bacillus; they show no reaction at all. The test guinea-pigs had extremely strong reactions; two of them died. We may therefore say that after injections of Choussy-Perrière water it is possible to confer upon animals an immunity sufficient to place them in a condition to resist various and repeated injections of deadly microbes; and also to furnish them with a sufficient resistance to combat the effect of those different injections with success.

**Injection of Toxins Followed by Injections of Mineral Water.**

Suppose now that we give to those animals, first an injection of toxin in a toxic dose; what will happen if we treat them immediately afterwards by an injection of mineral water? We have given first, typhoid toxin, afterwards a culture of coli bacillus twenty-four hours old, to a series of guinea-pigs and rabbits. We have injected a certain number of these animals every day for five days with an injection of La Bourboule water in the peritoneum; all the animals thus treated have resisted the infection while the control animals died in a period of time varying between three and five days. Four fresh rabbits received an injection of 1 c.c. of coli bacillus culture, twenty-four hours old, in the marginal vein of the ear. The two control rabbits died the third day; the two others which had received Choussy-Perrière water in the vein—5 c.c. every day for four days—recovered. We must, however, confess that they required nearly three weeks to recover their normal weight; six weeks later their blood count was practically normal.
Nevertheless, they slightly reacted to a new injection of water of La Bourboule: 99 to 112 (the test had passed from 95 to 157).

These recent experiments are only the verification of those made together with Professor Billard in 1906, when I noticed the fact without attaching much importance to it; my attention being then fixed on another point. I have taken them up again, because they again assume all their importance, showing the vital action of certain mineral waters on special pathological conditions. My experiments show results similar to the important and very curious research of M. Bellin, of Tours, which he allows me briefly to summarize.

Experiment I.—He injected into the muscles of three rabbits the virus of rabies. One received carbonate of soda in distilled water solution for twelve days, 2 c.c. per kilo weight of animal three times a day. A control animal and the animal treated had no symptom of hydrophobia; the third died. Twenty-one days after the last injection of saline solution, he injected into the peritoneum of each of them ½ c.c. of a streptococcus culture in glycerine bouillon, killing the normal rabbit in twenty-four hours. The control rabbit of the preceding experiment died in twenty-four hours. The subject previously treated by the solution of carbonate of soda had no untoward symptoms. Eighteen days after, it could tolerate without inconvenience the lethal dose of streptococcus. Finally, five months after the injection of the virus, it tolerated easily three-fourths of 1 c.c. of culture of Bacillus coli in the peritoneum, a dose which killed three control rabbits in nineteen, fourteen and twenty-three hours respectively.

Experiment II.—He injected three rabbits subcutaneously with ½ c.c. (a non-lethal dose) of a culture of streptococcus in pepton bouillon twenty-four hours old. One of these rabbits was treated by carbonate of soda, and another by calcium chloride, both given subcutaneously, 3 c.cg. per kilo weight of animal three times a day for three days, then twice a day for seven days. Ten days after the injection of streptococcus, he injected into the peritoneum of each of them a lethal dose of culture of hen cholera. The control rabbit died in eighteen hours. The rabbit treated with Na₂CO₃ showed no symptoms. The rabbit treated with CaCl₂ had diarrhoea and became thinner, but in ten days was quite well again.

Experiment III.—Three rabbits were injected subcutaneously with ½ c.c. of a culture of hen cholera twenty-four hours old (non-lethal dose). Injections of Na₂CO₃ and CaCl₂ were made for seven days, two injections every day, 3 c.cg. per kilo weight of animal. Five days after the last,
he injected a lethal dose of *Bacillus coli* into the peritoneum of each rabbit. The test animal died in forty-eight hours. The two animals treated had no symptoms. Three months from the beginning of the experiment, the *Bacillus coli* was injected under the same conditions, but in stronger doses, and killed both rabbits in nineteen and thirty-two hours respectively. The immunity conferred by the saline injections had disappeared.

**ANAPHYLAXIS AND THERMAL CURE.**

In final consideration of the matter, we may draw attention to the action of the water of Choussy-Perrière on the phenomena of anaphylaxis, demonstrated by the experiments of Dr. Daupeyroux [5] published last year. As an antigen, Dr. Daupeyroux used the haemostyle of Dr. Roussel, which gives very strong anaphylactic reactions, is not immediately fatal and allows of the series of phenomena which he wished to study being observed more easily. After having prepared his animals, he treated a series of them with water mixed with the bran with which they were fed. A second series was treated by injections of 1 c.c. of water every day; a third series by 2 c.c. every day. The water was taken from the spring and was used almost immediately. The water of La Bourboule was given to the rabbits twenty-four hours after treatment with the antigens. During eighteen consecutive days, the injections were made at the same rate and under the same conditions; at the end of this time, Dr. Daupeyroux made an injection "déchainante" and stated:

1. That the water of La Bourboule taken by the mouth does not seem to modify to any extent the anaphylactic conditions (perhaps by reason of the manner in which it was given).

2. The water of La Bourboule when injected has a certain anti-anaphylactic power, sometimes surprising, as it can make the animal absolutely refractory.

3. The question of doses seems to be of great importance, weak doses apparently being more active than stronger ones.

4. It seems that the longer the period of the action of the waters is protracted, the longer does the effect of small doses persist.

Thus a whole series of experiments made by different workers—Richet, Bellin, Daupeyroux, and myself—being compared, give analogous and confirmatory results.

From experiments dealing with the results obtained with the mineral water, certain facts become evident:
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(1) General immunity may be conferred upon the organism by the injections of mineral water, diminishing its susceptibility and protecting it from other infections.

(2) The infected organism may be endowed with sufficient resistance to allow it successfully to resist a previous infection which may be fatal to normal animals.

(3) In all these experiments, the importance of the amount of the dose of mineral water injected was clearly shown. Feeble and infrequent doses confer an immunity equal to that given by strong doses, and they appear to be of greater efficiency than the strong doses when it is necessary to detoxicate an organism or increase its resistance.

(4) Injections with strong doses lengthen the period of immunity.

For practical purposes it seems rational for two kinds of cases to be generally considered:—

(1) The case of an individual whose resistance is below normal, for instance, the case of a child who, when examined, appears to be in perfect health but is fragile, and his parents say: "He catches everything that comes along." Into such a child we would inject every eight or ten days, say 8 to 10 c.c. of water, according to his leucocytic reaction, ending the treatment with an injection of about 50 c.c., after which he will have acquired a leucocytic immunity, the final dose being given solely for its prolongation.

(2) The case of an organism seeking to rid itself of toxins, either arising within itself or from outside.

[Under (2) may be classed many patients at the thermal stations. If we consider—as may fairly be done—bearing in mind the works of Billard, Léry, Laisné, Galup and others, the manifestations of dietetic maladies as accidental forms of anaphylaxis, we should give these patients daily, or every other day, water injections, these being followed by a strong dose, given subcutaneously or intravenously, as soon as the organism has become, so to speak, disanaphylactized, so as to protect it from fresh infections.]

In practice this permanent immunity conferred by the thermal cure is often to be seen, and every year we observe patients whom we have treated for this or that trouble who have not had returning symptoms of it until the January or February of perhaps the following year, and every year we see children hitherto delicate who have passed the winter without the least sickness.

As a result, we believe that by this form of treatment we shall obtain results superior to those obtained by the various methods of
empiricism at present in use, which have, however, already given satisfactory results in many, perhaps the majority of cases. Nevertheless, whenever the condition of the patient troubles us, we do not hesitate to supplement the usual treatment by a few injections, or to substitute the injection treatment for the ingestion treatment.

But it may be objected that the experiments of Richet and Belin seem to prove that this is not alone a function of mineral waters. What advantage is there in employing them in preference to solutions of chloride, bicarbonate of soda, or calcium carbonate?

To this we answer, first, that the internal action of mineral waters can never be compared with that of the corresponding artificial preparations, because of the numerous elements present in the water and their chemical condition, which invests them with a decided superiority from a therapeutic point of view.

Secondly, this general property which is the attribute of many mineral waters is not common to all. All have not the same antitoxic power; this has been proved by the experiments published dealing with their anti-anaphylactic properties.

It is necessary that they should be studied and those which have common properties should be determined, as well as what these properties are, bearing in mind the special effect that each has on such and such organ in different affections. For instance, what is the local effect of sodium chloride, and where is this effect to be noted?

Prolonged observation has fixed respective regions of action for each mineral water—the skin, the mucous membranes, the kidneys, the circulating system, &c.—and experiments have confirmed these properties. Thus, in our special case, the experiments made at the laboratory of Professor Billard at Clermont-Ferrand, have shown that if we desire only to partially disanaphylactize an animal by an injection of mineral water, and if we consider the effects that follow this injection, we can see that after the use of La Bourboule water, for instance, the animal manifests principally lesions of the respiratory mucous membranes and of the skin, the signs of which are a definite dyspnœa and itching.

If, on the other hand, water of Châtel-Guyon be used, the effects noted will take the shape of intestinal symptoms, with severe, even blood-stained diarrhœa. If Vichy water be used, digestive disorders will be noticed; and it is this special study of the action of each thermal medication which will govern our choice, according to the location of
the special trouble to be treated, the general physical conditions being the same.

The poussée thermale, for instance, in my opinion, clearly determines the region of activity of each mineral water. It acts more, I believe, on the system of the patient than on the disease itself, but discussion as to the nature of the poussée thermale would take us too far afield; opinions about it differ so widely. We merely repeat what we said at the outset, that many experiments will be needed to determine exactly the limits of the fields of action of each mineral water on the different organs and the different parts played by these waters in various maladies.

How shall we explain the general action of hydro-mineral medication, which we have just discussed? We do not pretend to settle this question; although various works published up to now supply us with satisfactory hypotheses.

The action on leucocytosis, the defensive rôle of which is known, may be effected by the colloids in the Bourboule water.

As a result of the experiments of Victor Henri, Melle Cernavodéanu, Monnier, Vinard and others, we have become aware of the great potency of colloidal solutions. Foà and Agazotti, among others, have shown that if rabbits are injected with staphylococcus and streptococcus, the injection, one hour later, of electric colloidal silver will retard the death of the animal; while if injections of diplococcus and Eberth bacillus into rabbits are made, and these are followed by the injection of electric colloidal silver, one, twelve, or even twenty-four hours later, the animals' lives will be saved.

As to the toxins, Foà and Agazotti have observed that in vitro the tetanus, diphtheria, and dysentery toxins are not affected, while in vivo the injection of colloidal metal made immediately after the toxic injection, enables the animal to resist a ten times more than lethal dose of toxin.

With regard to the composition of mineral waters, and to the catalytic properties of some of their constituent elements, Bélin has shown the importance of their oxidizing properties in a series of recently published works. These data are confirmed by those of Lunière, Chevrotin, Delcourt, Bapin, &c. Does not, therefore, the chemistry of the organism, directly or indirectly, undergo some modification, so that the infecting agents are no longer able to find the necessary conditions for their growth? Is there not at the opportune moment an abundant production of oxidation? This question must be investigated.
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Numerous experiments are, however, still necessary to define exactly the roles of the different factors, the duration of the immunity obtained, and the immunizing power exercised by the different mineral waters on the various toxins.

Conclusion.

In any case the general immunity conferred by the injection of mineral water is not an hypothesis. It is greater than the immunity conferred by the usual means, so far as we may judge from experimental physiology and hydro-mineral clinics, considering the rapidity with which it is obtained, its strength, its relative non-specificity and the facility with which it may be determined. This method should be employed whenever it is necessary to immunize an individual from outside infections, either by diminishing his susceptibility or by strengthening his resisting power, allowing him to combat external or internal infections.

The administration of hydro-mineral injections whenever possible will always yield better results than when the waters are ingested; the action will be more rapid, more complete, and of longer duration.

A fuller knowledge of the action of each mineral water on the different organs will enable us to produce at the same time both general and local effects, and to determine with exactitude, for each particular case, its hydrological indications.

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The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.
Section for the Study of Disease in Children.

President—Dr. J. Porter Parkinson.

Case of Urticaria Pigmentosa.

By J. L. Bunch, M.D.

This patient, a boy aged 6 years, has a very well marked pigmented eruption over the whole of the trunk and limbs, spreading up also on to the neck, but leaving the face quite clear. His mother says he has had these spots ever since a short time after his birth. Each spot persists for a considerable time. He has a very well marked factitious urticaria: ten minutes ago I drew my nail lightly over his shoulder, and you can see the mark quite plainly now. The spots seem to irritate him to some extent; he scratches them from time to time, but they are said not to interfere with his sleep. He is said to be a fairly good patient, and does not lie awake scratching for long. He is a Russian dancer, it is said, and has been trained for the stage. Certainly he is a wonderful mimic, but here I cannot induce him to give an exhibition.

The question has arisen as to whether urticaria pigmentosa is more common among Russians and Poles than among English people. One does not see many of these cases at any time. Those I see at the Queen's Hospital for Children appear to be mostly of Polish or Russian nationality. The lesions tend to clear up rather late in life, sometimes they may start about puberty, but the usual happening is for them to start soon after birth, as in this boy.

Internal treatment seems to have no particular effect on the lesions, and local applications can only be directed to allaying the itching.

This boy appears to be fairly well nourished. He was admitted into Dr. Parkinson's ward, in the first place, because of vomiting and diarrhoea, and he had a definite rise of temperature. When the diarrhoea and vomiting were got rid of, the spots did not seem to have improved; in fact, there has been very little change in his condition during his stay in hospital.

1 At a meeting of the Section, held October 25, 1918.
Case of Aplastic Anaemia (previously shown).

By J. Porter Parkinson, M.D. (President).

I showed this case to you at the meeting held on May 24 last. Some of you may remember that he was a very anaemic boy. His blood count was a very unusual one, and was suggestive of aplastic anaemia. The symptoms he had were various hemorrhages, not only under the skin, but also from the nose and mouth, and occasionally there was blood in the faeces and urine. At the time he was last shown the report on the blood count was as follows: Red cells, 1,260,000; leucocytes, 800; haemoglobin, 20 per cent.; colour index, 0.9; polymorphonuclears, 27 per cent.; lymphocytes, 72.5 per cent.; eosinophils, 0.5 per cent. There were no nucleated cells, nor were any marrow cells seen. There seemed to be no doubt that it was a case of aplastic anaemia, in which the marrow of the bone ceases to work, so that it does not produce polymorphonuclear leucocytes. I presume that is the usually accepted explanation of these cases.

After the boy had been shown here on the last occasion, the anaemia had increased, and the hemorrhages in the gums continued. On June 11, that is, nearly three weeks after he was shown here, the red cells had gone down to 624,000, the white cells were 1,600, the haemoglobin was 17 per cent., the colour index 1.4, the polymorphonuclears 28 per cent., the lymphocytes 68 per cent., eosinophils 2 per cent. No nucleated reds were seen then or at any time. Transfusion was performed by Mr. Bankart, on June 18, from a healthy male aged 26 years. The blood was tested for haemolysis against that of the patient and was found to be satisfactory; 300 c.c. of blood mixed with 30 c.c. of citrate solution was injected intravenously. The patient's colour improved greatly during and after that procedure. About a week afterwards, the blood count showed a slight increase of red cells, in other respects it was about the same as before. The increase then was only up to 880,000. A second transfusion was carried out on June 28, the donor being a healthy female aged 25 years. Her blood also was tested for haemolysis against that of the patient and was satisfactory; 500 c.c. of blood mixed with 50 c.c. of citrate solution were injected, as before. After five minutes, the patient became collapsed, very cyanosed, and vomited, and the pulse became feeble and rapid. He recovered sufficiently from this, however, for the transfusion to be continued. But
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at the end of the operation there was some epistaxis, and several large pinkish spots appeared on the arms and body. On being returned to the ward he was still very collapsed. Pituitrin was given subcutaneously, and he passed bright blood in the urine and by the rectum; he vomited blood, and the epistaxis continued. Coagulose was given subcutaneously, and calcium lactate by the mouth. Next day he was still passing blood by the bowel and the urine, and was deeply jaundiced and comatose. On June 30 the haemorrhages had ceased, but he was still jaundiced. The following day he seemed rather better. Later his blood was examined, and was found to contain only 580,000 red cells. The rest of the blood count was similar to the others I have read out. One nucleated red cell was seen in counting 280 leucocytes.

On July 10 the jaundice had gone, but the child was weak and drowsy. The heart apex was 1½ in. outside the nipple line, and a systolic murmur and a presystolic murmur were heard over it. The temperature, which had been somewhat raised during the past month, went up to 103°8°F., but on the following day it fell to 100°F. The temperature variation was mostly between 99° and 100°F.

The child grew weaker and weaker and more drowsy, and had incontinence of urine and faeces. The mother refused to have another transfusion done, and on July 24 insisted on removing him home. He lay unconscious for a fortnight and then slowly improved. The donor of the second supply of blood developed acute influenza on the day following the transfusion.

At the present time the boy seems to have completely recovered. His colour is good, the heart has returned to its normal size, and the murmurs at the apex have disappeared. The blood count on June 22 showed 3,960,000 red cells per cubic millimetre of blood, whites 5,800, haemoglobin 65 per cent., colour index 0·8. Of the white cells, 65 per cent. were polymorphonuclears, 32 per cent. lymphocytes, 2 per cent. eosinophils, and 1 per cent. hyaline. There was no alteration in the shape of the red cells, there were no nucleated forms, and no marrow cells. There were many haemorrhages to be seen in both ocular fundi, and there was some optic neuritis. Those haemorrhages have now disappeared, and a little fibrous tissue remains along the vessels due to the old neuritis. That is yesterday's ophthalmological report. So we may say the boy has practically recovered from this grave condition, from which recoveries are almost unknown.

I think it is the only case of the kind which has been shown before the Society. It is a very rare type of anaemia, and it is a still rarer
event to have a case recovering from it. No immediate effect, anyhow, seemed to follow the treatment. The first treatment in the case which was tried was to apply X-rays to the bones, with the idea of stimulating them to perform their function—that is, the red marrow. As no immediate result ensued, it had been given up when transfusion was determined on.

DISCUSSION.

Dr. F. Parkes Weber: As far as I remember, we were inclined to give a hopeful prognosis in this case when it was shown last time: but the complete recovery which has ensued is a matter for congratulation. The diagnosis that it was a case of aplastic anaemia, but not of the acute type, still holds good.

The President (in reply): With regard to the possible causation of this case, it has sometimes been suggested that the condition may follow nephritis. This boy had kidney trouble six years previously, so the history says. But there was no sign of that when he was in the hospital with this anaemia: he had no albuminuria at any time. Hence I do not think we can attribute the causation in this case to kidney trouble. There is no history of "growing pains."

A New Counting Chamber for Cells, &c., in Fluids.

By K. S. Bhat.

The chamber is of the Bürker type, each ruled area being 5 mm. square, and the depth 0.1 mm. The volume over each ruled area is therefore 2.5 c.mm., or 5 c.mm. over the two. The rulings are so placed that, looking through the microscope, the two left-hand top corners are both seen to be occupied by doubly bisected squares, without having to turn the slide round. Each ruled area is practically a Türk square with the lines prolonged into a surrounding row of millimetre squares, a few lines being added to make the subdivisions more uniform. Four points are also marked on the slide where the corners of the coverslip ought to lie.

General usefulness is claimed for the chamber, but little originality. Bürker's principle of primary coverslip application ensures uniform cell distribution. Each half of the chamber serves as a control for the other. And the large size of the ruling, with the different degrees of subdivision in different portions of it, make the chamber equally useful for fluids with high cell contents like blood and low ones like some cerebro-spinal fluids.
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President—Dr. J. Porter Parkinson.

Case of Shortening of the Limbs on one side of the Body; ? Osteogenesis Imperfecta Unilateralis.¹

By Joseph E. Adams, F.R.C.S.

The diagnosis in this case is uncertain, as the condition is, in my experience, unique. The boy is now aged 6 years, and is the ninth child in a family of eleven. The parents and the other children are quite healthy, and there is no history of miscarriages. The abnormality of the right lower extremity was first noticed when, at the age of 18 months, the child began to walk. The abnormality in the upper extremity was not observed until he came to Shadwell Hospital, where he was under my care for a short time. When he was first seen, in 1915, there was a shortening of 2 in. in the leg, and temporarily a boot was supplied with a thickened sole. He walked about in it without pain. Apparently there has never been pain, and he uses both right upper and lower extremities perfectly well. The present condition was revealed in its entirety in March, 1918. Now, the right lower extremity is 3½ in. shorter than the left, and the right upper extremity is considerably shorter than its fellow. Wassermann’s reaction with the serum was negative, as also was von Pirquet’s reaction. The urine has been examined on two occasions for Bence Jones’ proteid, but with negative results. Except for measles at the age of 6 years, and a slight transient attack of jaundice in March last, the boy has been quite healthy: there was no rise of temperature in this last illness.

It is a remarkable fact that this condition is limited almost entirely to the right side of the body. There are bosses on the skull on both

¹ At a meeting of the Section, held November 22, 1918.
sides, but that is the only exception to its unilaterality. The bones affected are the upper end of the right humerus, the lower end of the radius, some metacarpal and phalangeal bones of the right hand, the upper and lower ends of the femur, the upper and lower ends of the right tibia, the upper end of the fibula, and, to some extent, the metatarsals and phalanges of the right foot.

With regard to pathological appearances, the majority of the skiagrams exhibit a lesion which is rather suggestive of inflammatory change than of any other obvious pathological lesion. I submitted the skiagrams to Mr. Shattock, who at once suggested that it was a tuberculous epiphysitis, or a tuberculous osteomyelitis, and that is chiefly why the von Pirquet test was done. But the bones do not appear to be typical of that, though certainly the lesion exhibits rarefaction. It is not limited to the epiphyses, and in all cases it extends well into the shaft of the bone. One might, therefore, call it a unilateral diffuse osteomyelitis. But it would be difficult to state the precise cause and nature of the lesion. Tubercle seems to be out of the question; there is nothing to support the idea of syphilis, and a pyæmic lesion is a rather improbable notion. If it had dated from birth, and not started when the child began to walk, perhaps it would have been reasonable to call it osteogenesis imperfecta, considering that it was limited to the right side. But the skiagrams hardly bear out that diagnosis.

With regard to multiple myelomata, these might reasonably be limited to the right side, and they are supposed to be associated with Bence Jones' proteid in the urine. But on two occasions the patient's urine has been examined for this substance, and it has not been found.

If we call the condition fragilitas ossium, that does not help to explain it. No doubt the bones are fragile, because the skiagrams show there has been a fracture at the upper end of the tibia, but apparently it has re-united, and no one knows when it took place.

A suggestion made to me this evening was that it might be due to multiple enchondromata. The skiagram of the lower end of the femur suggests the appearance of enchondroma, but most of the other skiagrams suggest an inflammatory lesion rather than a neoplasm.

DISCUSSION.

Dr. H. C. CAMERON: This case bears a resemblance to the one I showed at this Section at the meeting before the last, a boy of about the same age. The striking feature, at first sight, was that the right arm and leg were very much
shorter than those on the left side. The skiagram of the bones on the affected side showed changes, especially in the metacarpals and phalanges, which seemed to be of the same nature as the changes in the hand of this boy, although much further advanced. The condition in that case also was almost confined to one side: it was not absolutely unilateral because the little finger on the opposite side was involved. The general opinion at the meeting was that it was a case of multiple enchondromata. I think the same diagnosis applies here. The condition is likely to be progressive. The skiagrams do not resemble those from cases of osteogenesis imperfecta.

Dr. Leonard Guthrie: This is a most interesting case, and probably unique. Still, I cannot help feeling that the possibility of its being osteogenesis imperfecta has been a little hurriedly dismissed. There seem to be many points about it which bring it into line with those cases, though I admit that a unilateral condition of osteogenesis imperfecta has not been described. At the same time I do not see why such should not occur. We are told that it should have been congenital if it was that; but in many instances cases I have met have only been diagnosed when fractures have occurred. This child has had a fracture, and there is no history of when it occurred, nor any information about it. That is exactly what happens in many cases of osteogenesis imperfecta. The skiagrams seem to show rarefaction, which is what one would expect in such a case. I would not like to affirm or deny the possibility of its being a case of enchondromata, and I am not sure whether that finding was proved in Dr. Cameron's case. With regard to the bossing of the skull, over the ears, I thought Dr. Cameron would be attracted by that at once, as the bosses strongly resemble those he so aptly described as present in osteogenesis imperfecta. I regard this as a very anomalous condition of osteogenesis imperfecta.

Dr. F. Parkes Weber: This case may possibly be one of those conditions described as osteitis fibrosa, not the completely generalized form which is sometimes associated with von Recklinghausen's name, but one of the more local forms.

The President: There is a good deal of significance in the absence of pain in this case. It was at first suggested that the condition was inflammatory, but the absence of pain all through is very much against its being of inflammatory nature. The lesions apparently start in the neighbourhood of the epiphyses, and the skiagrams impressed upon me the same idea as upon Dr. Cameron. I thought they showed that there was a deposit of cartilage, a decided new growth of some kind there. There was considerable thickening of the parts of the bones near the epiphyses, and apparently some new tissue.

Mr. J. E. Adams (in reply): I am inclined to accept Dr. Leonard Guthrie's suggestion rather than the others. With regard to the suggestion of osteitis fibrosa, I thought that was more frequently limited to the region of the
epiphyses, and did not usually extend into the bone shafts, as this condition has done. I think the only skiagram which definitely supports the suggestion of hyperplasia, or the formation of anything like enchondromata, is that of the lower end of the femur. There does not seem any indication for surgical interference at the present time.

Case of Oxycephaly with Symmetrical Polysyndactyilia.

By H. C. Cameron, M.D.

This little baby is 7 weeks old. There is one other child in the family, and that is healthy. No other similar affection appears in the family history. The skull is of normal measurement for the age, and the weight of the child is 11 lb. 7 oz. The mother noted extreme prominence of the eyes at birth, and she thinks they are not so prominent since the first few weeks of life. But they are still decidedly prominent, the orbits no doubt being shallow. The skull shows the ordinary appearance of mild oxycephaly or tower skull. The optic disk is normal: there is no evidence of either optic atrophy or retinitis pigmentosa.

The interest of the case is, that it shows a deformity which is exactly like a deformity I have seen once before in a baby of about the same age with oxycephaly, a symmetrical deformity of the thumbs and great toes—a polysyndactylism—giant great toes and giant thumbs consisting of the union of two separate thumbs and two separate toes.

A French worker has written a paper (which I have not yet had time to read) on what he calls acrocephalo-polysyndactyilia, a term which sums up the chief features of the case. I think, from the fact that the two cases have come to me accidentally within a short time, that there is an occasional association between these unusual deformities.

Of course, oxycephaly has been described in association with many other deformities, and it is a most interesting speculation as to what causes these associations of deformity, such as cervical ribs with coloboma iris and retinitis pigmentosa, and so on, and what is the connecting cause. Here there seems to be an association between the faults in ossification of the skull, and this form of polysyndactyilia.
Dr. LEONARD GUTHRIE: The late Dr. George Carpenter showed, before the old Children's Society, two sisters,¹ whose heads were very much like this child's, only the deformity was much more gross to look at. Those children had a large quantity of Wormian bones, which made the head more or less of the shape of a turnip, and each of these cases had either syndactyly or six toes on each foot.

Case of Achondroplasia with Hydrocephalus.

By H. C. CAMERON, M.D.

I brought this case in order that I might ask the opinion of members as to whether the very large and curious head in this severe case of achondroplasia was, or was not, associated with distension of the ventricles of the brain—i.e., whether hydrocephalus was present. Of course, all these children have large rounded heads, with important-looking, dome-like foreheads. I saw this child when 4 or 5 months old, and have not seen it since until now, in its fourteenth month, and at the former date I made no notes as to the possibility of there being hydrocephalus. My feeling is, that it has hydrocephalus. The mother thinks the head is growing rapidly, and I wondered whether members had met with an association of hydrocephalus with achondroplasia.

A case of such severity as I have shown to-day does not, I think, often survive. When I saw it at 4 months old it was the oddest looking mortal imaginable: the limbs were much shorter than any I had seen before, and the head much larger; the pelvis was narrower, it tapered to a point more. It recalled those specimens of achondroplasiac foetuses which we see in the museums, more than the children, subjects of the condition, whom we see in life. I take it that achondroplasia is a disorder which, in its greater degrees, is incompatible with life, and that it is only the comparatively mild cases which survive and grow up. This present case is, I think, one of the most severe which is compatible with life. The spinal curvature described by Jansen is very well marked.

DISCUSSION.

Dr. Leonard Guthrie: With regard to the question of hydrocephalus in addition to achondroplasia, every case of achondroplasia is apt to be regarded as hydrocephalus, because the head is so much larger in proportion to the limbs. But the head of the present child is even larger than what one usually sees in achondroplasia: here there appears to be hydrocephalus. I have always regarded Jansen's brochure as an interesting and most remarkable example of special pleading: there is hardly any feature of achondroplasia which does not accord with his theory. But when one comes to actual facts, the theory is not so convincing. On the first page there is a picture of a perfectly round amniotic cavity, with a fetus doubled up within it, and a great deficiency of amniotic secretion. You are apt to say that is positive proof. But you find it is only a diagram, drawn to fit in with his theory, and that there was never anything like it seen at all.

Dr. Eric Pritchard: This head does not strike me as being a typical case of hydrocephalus, there is not the general roundness of the skull usual in such cases, and further the fontanelle is distinctly depressed.

The President: From the size of the head, it struck me there was some hydrocephalus here: I think this head would be 3 in. larger than the average for the age. The average is 18 in., whereas this would be about 21 or 22 in. There is no bulging of the fontanelle, but both fontanelles are very large, and the head is larger than that one expects to find when the condition is pure achondroplasia.

Notes on a Case of Chronic Priapism.

By Eric Pritchard, M.D.

This is a case, I believe, of vaso-motor habit. It is four years since the boy acquired it, and he has had it, I think, ever since without intermission. During the daytime the organ is collapsed, but as soon as the boy goes to sleep at night, the penis becomes engorged, and by the time the sleep is sound there is enormous priapism. At about the eighth day this boy was circumcised. The after-dressing was apparently conducted in an unsatisfactory manner, so that there was soreness. The second night after the operation there was considerable turgescence of the organ, and during the next three or four days the penis was enormously swollen, and apparently painful too, after which date the
wound began to heal and the pain to subside. The penis was erect during sleep, and subsided during the day: there had been nothing of that nature before the operation. I think it is a clear case of priapism induced by habit, which was started at the age of ten days.

I do not know whether anything can be done, but I think the mother's and father's anxiety tends, by suggestion, to keep up the condition, because, as soon as the child is asleep, they rush to pull down the bed-clothes to look at it, and they talk a good deal about it in the presence of the child. I think counter-suggestion may be efficacious in this case.

DISCUSSION.

The President: Spinal douching, and measures of that sort, might help, as well as the administration of bromide, which has perhaps already been given. There seems to be no focus of irritation now.

Dr. H. C. Cameron: Does the condition persist when the child is away from home? It would be interesting to see what would be the effect of a visit to somebody who does not make these unfortunate suggestions.

Dr. F. Parkes Weber: Is there any rectal irritation, such as constipation, which might produce the condition by reflex action, when the higher nervous control is removed, or partially removed, owing to sleep?

Dr. Eric Pritchard (in reply): The child has been treated with bromides. There was a history of some damage to the neck, but it seems to have been a haematoma of the sternomastoid. I do not think that throws any light on this condition.
Section for the Study of Disease in Children.

President—Dr. J. PORTER PARKINSON.

A New Case of Lipodystrophia Progressiva.¹

By F. PARKES WEBER, M.D., and T. H. GUNEWARDENE, M.R.C.S.

The patient, E. W., a girl, aged 12½ years, has been since the age of 7½ years progressively losing the subcutaneous fat over her face, neck, upper extremities and trunk, as far down as the pelvis. She now has an extremely emaciated appearance over those parts, but the buttocks and lower extremities are fairly plump (certainly not thin). Beyond her wasted appearance she has nothing to complain of, excepting an occasional catarrhal condition of her nose and pharynx. On the whole, she has been gaining in weight since she has been under observation during recent years; this gain in weight is doubtless due chiefly to growth in length of the body. There is no evidence of any disease of the thoracic or abdominal viscera, and the urine is free from albumin and sugar.

¹ At a meeting of the Section, held January 24, 1919.
At 7½ years of age she was rather full in the face, as a photograph of about that period shows. Then she suffered from measles, whooping-cough and pneumonia, close together, and the wasting of the subcutaneous fat commenced gradually after that. Fig. 1 shows the patient at the age of about 7½ years. Figs. 2 to 4 show her at the age of 12½ years, with all the appearances of lipodystrophia progressiva.

The case is a typical one of so-called "lipodystrophia progressiva," notably in regard to the distribution of the fat-atrophy, the patient's sex, and the patient's age when the change in her appearance was first noticed. The diagnosis was made by Mr. T. H. Gunewardene, who saw her at the East London Hospital for Children.

Dr. GUNEWARDENE: Her chest has been twice examined at Victoria Park Hospital, without any evidence of disease being found.

Osteogenesis Imperfecta.

By E. Bronson, M.D.

* Case I.—A male, full term infant aged 6 months. He had a fracture of the left humerus and the right tibia at birth, and of the right humerus when aged 6 months. No other fractures have been known to occur. He was first seen at the age of 1 month, when, as now, there was noticeable shortening of the proximal long bones. The shape of the head was entirely normal and symmetrical. Cranial ossification was deficient—a mosaic of bony islands over the occiput and the temporo-parietal region. The temporo-parietal suture lay open the width of one's finger. The sagittal suture was also gaping from the anterior to the posterior fontanelles. The anterior fontanelle measured 3 in. transversely. During the fourth month the temporo-parietal region began to ossify rapidly, and now membrane can be felt only at one spot above the right ear. At the same time the present bilateral bulging above the ears has become noticeable. This bilateral enlargement has increased up to the present, when it is sufficient to turn the ears slightly outward, giving the shape of head described by Dr. Cameron. Ossification over the occiput has not progressed as rapidly as in the temporo-parietal region, and here, also, there will probably be abnormal

protuberance when ossification is completed. The frontal bones showed no defective ossification when I first saw the child, and there is none of the frontal fullness and downward tilted axis of the eye, which Case II shows. Skiagrams show fractures with abundant callus of both humeri, shortened and somewhat bowed femora, and tibiae bowed in the lower third. This bowing is due, obviously, to the crossed leg position which the infant assumes. The family history is completely negative; both parents and two other children are healthy. The Wassermann reaction is negative in both mother and the patient. The sclerotics are not grey-blue, as has been described in several isolated as well as family cases of bone fragility.

Case II.—A female child, aged 3 years 10 months, who had a broken clavicle and right humerus at birth. The mother says she has had fifteen fractures in all, the latest three weeks ago. Most of these have been untreated, with the result of extreme deformity of the long bones. There is considerable hypotonia of the joints, and an underhung jaw. The head shows bilateral but also frontal enlargement, and slight downward rotation of the antero-posterior axis of the eyes. There is no occipital prominence. The sclerotics are not grey-blue. The father of this child had thirty fractures, the first in infancy. He was unable to get about except in a wheeled chair. He died recently, at 29 years of age, of influenza. No history of either blue sclerotics or otosclerosis could be obtained. The grandfather was reported to be quite normal. A brother of the patient, aged 8 years, has had no fractures.

DISCUSSION.

Dr. H. C. Cameron: Professor Keith has shown me the adult skeleton at the College of Surgeons of a case which exhibits the bilateral bulging very well. He was a man of most passionate and uncontrolled temper, and it has been suggested that this peculiarity of temperament is general. I believe there is a specimen in the Edinburgh College of Surgeons, called “Bowed Jimmy,” a rickety dwarf, probably a case of osteogenesis imperfecta. In the history it is noted that he headed the “meal riots” in Edinburgh. Certainly some of the children with osteogenesis imperfecta who have come to me have been of very excitable nature. It is obvious that an infancy spent with the handicap which these children have must be an abnormal infancy, and such an abnormality of itself would lead no doubt to excitability of temper. I ask whether Dr. Bronson has noted any peculiarity of temperament in these children.

Dr. Bronson (in reply): I may almost say the opposite was the case in regard to the temperament of the osteogenesis imperfecta cases which I have
seen. The infant cast smiles at each visit, and the little girl, whom I saw for the first time yesterday, was at once very friendly. One of the cases which I reported in the Edinburgh Medical Journal, April, 1917, was for a time the pet of the ward at the Edinburgh Royal Infirmary. The family of fifty-five described in the same article were jovial people: the grandfather, a public-house keeper in the town, had been especially noted for his good humour.

**Myoclonus Multiplex in a Girl aged 2 years 11 months.**

**By F. J. Poynton, M.D.**

(Shown by Miss K. Cass.)

**History:** October 27, 1918.—F. C., Jewess, aged 2 years 11 months, suddenly started to tremble all over, threw her arms about, cried out, and seemed to be in pain. She became quieter when nursed, and settled off to sleep. On attempting to lay her down she became violent, threw herself about and screamed. The parents say she has been like this for the whole of the last month. She takes her food well, and the parents say she has no difficulty in swallowing. She has to be held while being fed, and seems terrified to be left without support. The parents say the child feels about with her hands for an object as if she does not see clearly, and makes several attempts before she can take it. The father reports that the child has not been feverish nor sick, and has a good appetite. She has made no attempt to walk since the onset of this illness, and has not talked.

**Past history:** She was able to walk and talk up to a month ago. She has had no illnesses except whooping-cough, measles, chicken-pox, but she was a delicate baby.

**Family history:** Eleven children in the family. All of them are healthy. The parents are healthy.

**Condition on admission (November 23, 1918):** Sturdy, plump child, with high colour. All her movements are wild and choreiform. Rapid nodding movements of head combined with lateral movements. Eyes in a state of rapid coarse tremor in every direction, with dilated pupils reacting to light. Eyelids and mouth twitched incessantly. Respiration jerky and rapid. When held in a standing position the child is unable to keep herself steady, and is in a state of generalized coarse tremor. She does not appear to sway in any one direction. The child quietens

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Case of Pseudohypertrophic Muscular Paralysis in a Girl, aged 10 years.

By F. J. Poynton, M.D.

(Shown by Miss K. Cass.)

History of present illness: January 1, 1919.—Nine months ago the mother noticed that the child (a girl, aged 10 years), dragged the right foot slightly. She had no pain and was otherwise well. The patient has been going to school up to the present. She has found a slight difficulty in walking, and is apt to stumble when running. She says she finds it hard to get upstairs. The left leg is said to have been affected for the last nine months; she drags with both her feet. No weakness of arms has been noticed. Her general health is fair. She has been a healthy child, and has had no other illnesses except measles.
Family history: Seven children. All strong and healthy except the eldest, aged 18 years, who has a deformed back with weakness of legs and left arm. No history of any similar complaint in parents' families.

Condition on admittance: She walks steadily, but tends to sway from side to side. There is slight lordosis. She can get up from a lying position without any evident difficulty. Muscular system: The calves are large and hard, especially the left. The vasti externi are much enlarged, giving a curved appearance to the thighs. The shoulders are loose. There is wasting of the lower fibres of the pectoralis major and the latissimus dorsi. The serratus magnus is wasted, and promotes a tendency to winging of scapulae. The deltoids are slightly hypertrophied, the left more than the right. The infraspinati are hypertrophied.

The interesting points in the case are: the sex, age, and absence of family history.

DISCUSSION.

Dr. E. Cautley: Has Dr. Poynton considered the alternative of this condition being secondary to encephalitis? The clonic movements are due to irritation of the cerebral cortex. That seems to be a more probable diagnosis; the case hardly consorts with one of myoclonus multiplex, and there is definite mental deficiency. The comparatively few cases of myoclonus I have seen have shown no sign of mental deficiency.

Dr. Lapage: I can support what Dr. Cautley has said. The onset was sudden, at 2½ years of age, and the symptoms are those following some sudden illness. There is mental deficiency: the head is 17 in. in circumference, smaller than that of a normal child of the same age. Though the child can walk and talk, evidently she is not very forward. It looks as if it is likely to be encephalitis.

Dr. F. Parkes Weber: I think this case is probably analogous to those of so-called "acute tremor" in children, and those of so-called "acute ataxy" in children, both of which are generally due to encephalitis, though not of the same localization.

The President: The acute onset with these symptoms in a child accords with Dr. Cautley's views. It is an unusual distribution for myoclonus multiplex. I should have thought there was very little doubt about its being encephalitis, even though there was no convulsion or definite evidence of such an onset.
Dr. Bronson: Was the percentage of sugar in the blood determined? Hypoglycaemia in the muscular dystrophies has been reported in a number of cases in the United States along with a cretinism. The normal sugar content of the blood of 0.09 per cent. to 0.12 per cent. is reduced to 0.07 per cent. or even lower. In muscle wasting of central origin, such as progressive muscular atrophy, or in muscle wasting due to disabling disease, such as chronic arthritis, there is a normal amount of blood sugar and no creatin in the urine. The question of some relationship between the insufficient utilization of the muscles of sugar and the deposition in them of fat suggests a defect in the control of the glycogen-sugar metabolism, and further study of the chemical physiology of these cases would be of interest.

Miss Cass (in reply): No, the blood was not tested for the sugar content.
Section for the Study of Disease in Children.

President—Dr. J. Porter Parkinson.

Multiple Neurofibromatosis (von Recklinghausen's Disease).

By E. Bronson, M.D.

D. T., aged 7 years 11 months, had the first pigmented spot about two years ago. Within six months a soft tumour had started below the right scapula and many more pigmented areas had appeared. During the past year the tumour has increased in size. It measures 4 cm. by 7 cm., and is sessile in type. The skin is dependent over it. Over her chest and abdomen, and to a less extent over her extremities, there are café au lait coloured patches, irregular in outline and varying from a few millimetres to 3 cm. in diameter. She is 45 in. in height and sluggish in mental development. Her father has had similar pigmented spots, and "tiny kernels under the skin" since his earliest recollection. He is 5 ft. in height.

Case for Diagnosis; ? Xanthelasmoidea (Urticaria Pigmentosa).

By E. Bronson, M.D.

D. C., aged 1½ years, had at birth two brown patches on his right shoulder. These were increasing in size, so an unsuccessful attempt was made to excise them. At his fifth week other similar patches appeared over the body. Since the sixth month no fresh spots have appeared, and recently all have faded somewhat, a few entirely.

1 At a meeting of the Section, held February 28, 1919.
No urticarial symptoms have ever been present, and his general health has been excellent.

When I saw him first at 9 months of age, he had twenty-eight dark brown patches, scattered asymmetrically over his body, varying in size from 5 mm. to 6 cm. They were irregularly circular and faded gradually into the surrounding skin. The larger were flatly papular, the smaller, macular. Conspicuous scars marked the unsuccessful attempt at removal.

The case is not typical of urticaria pigmentosa, if urticaria is a necessary characteristic of that condition. Dr. Norman Walker prefers the term "xanthelasmoidea," and no longer classifies the condition with the urticarias. Unfortunately the mother refused further cutting of the skin for diagnosis.

DISCUSSION.

Mr. H. C. Samuel: This case, if it is urticaria pigmentosa, is extremely atypical. In the cases which I have seen the eruption was more symmetrical and more widespread on the trunk. A diagnosis can be made by doing a biopsy and finding a large number of mast cells in the section. With regard to the condition being of a nævoid nature, dermatologists do not generally regard it as such, but rather of the nature of urticaria of chronic type, often associated with obvious urticarial dermatographia, &c.

The President: I agree with the view of Mr. Samuel. I have seen a certain number of such cases, and they had a great family resemblance one to another. The patches I saw were of the size of a threepenny or sixpenny piece, and were scattered more or less over the whole trunk. They were usually round or oval, and nothing like the size of the patches in this case. Still, I cannot confidently suggest an alternative diagnosis.

Hypertrichosis in a Mentally Defective Child.

By E. Bronson, M.D.

B. M., aged 3 years 8 months, came to Dr. Poynton's clinic at the Children's Hospital, Great Ormond Street, when 15 months old, for excess of hair on his forehead, neck, shoulders, arms, and sacrum. The hair then, as now, measured from 1 in. to 6 in. in length, and had been present from birth. He had a negroid nose and rather a dog-shaped face. He cut his teeth early, and normally, except that each of his first two molars has a fifth cusp. The fontanelle closed early. Several fits occurred
between the fifth and tenth month. He did not say his first word nor walk until 2½ years old. He was in mischief and very "clever" many months before he talked, in fact was always a "bright" baby. He is an example of a typical "naughty boy" in a mentally defective child at present. He does not take colds easily, and is generally in excellent health.

Dr. Poynton put him on thyroid treatment when he first saw him and he has been on it since until two months ago. He has continued to improve in talking, and has continued as active since its cessation. I have followed him for a year. Though he had had thyroid treatment for fourteen months, when I first saw him he was just beginning to walk, and saying only a few words. The large tongue had persisted, and, according to a photograph, a similar animal-like face. In fact the thyroid had had no specific effect.

DISCUSSION.

Dr. Burgess: Has the thyroid treatment had anything to do with the growth of the hair in the case of hypertrichosis? I had under my care a middle-aged lady who was bald, and I advised her to take thyroid. She did so, and I did not see her for some time, but when I did she said it had been very successful, but the hair had grown all over her.

Mr. H. C. Samuel: Was there anything wrong with the heart? I examined the heart, and found it slow for a child of that age, and somewhat irregular. Has that any bearing on the case?

Dr. Bronson (in reply): The thyroid treatment had had no apparent effect on the growth of the hair. The hypertrichosis has been present from birth very much as at present. I should regard the slow, irregular pulse as the respiratory irregularity or the phasic variation which Sir James Mackenzie describes, both of which are physiological.

Section from a Case of Tuberculous Meningitis of the Spinal Cord simulating Anterior Poliomyelitis.

By H. C. Cameron, M.D.

I shall first mention a previous experience of the condition which I have had. A year or so ago I saw, in consultation, the little daughter of a doctor, and I concluded from the symptoms that the child had an anterior poliomyelitis of the lumbar centre. The symptoms had
been present seven days. The child was in the drawing room, just able to walk a little, and the father pointed out the weakness of the right leg. The ankle-jerk was absent, the knee-jerk just obtainable on the right side, but very much less than on the left. Dr. James Taylor, who also saw the case a day later, agreed with this diagnosis. Three weeks later I was again summoned. The child was developing tuberculous meningitis. Ten days later she died. Before the development of the meningitis the paralysis of the right foot had got rapidly better. I was unable to explain the association of two conditions apparently distinct, but the recollection of this case came to me when, a few months ago, there was admitted into Guy's Hospital a child suffering apparently from anterior poliomyelitis involving the lower extremity. The child made a good recovery, and three weeks later was about to be discharged, seemingly in good health, when the temperature rose and tuberculous meningitis slowly developed. I met the case for the first time in the post-mortem room, and that was the history given to me. The cord, at the level of the lumbar enlargement, showed some traces of adherent lymph: there was obvious extensive tuberculous meningitis of the brain, and there were caseous glands at the bifurcation of the bronchi. The section I show to-night is one across the lumbar enlargement, and there are giant-cell systems and foci of caseation among the issuing nerve roots of the lumbar enlargement. This section seems to me to afford the explanation of the symptoms of the case which I saw eighteen months ago. I think both the children were suffering from tuberculous meningitis of the cord, which preceded by two or three weeks the meningitis of the brain.

DISCUSSION.

The President: Did the symptoms of paralysis appear with that dramatic suddenness which one often witnesses in anterior poliomyelitis, or was the onset more gradual? If these cases were common, it would be useful for lumbar puncture to be done on all cases of anterior poliomyelitis for the sake of diagnosis. But, doubtless, the opinion of most of us is that they are very unusual cases indeed.

Dr. H. C. Cameron (in reply): The onset was very much that which one finds in many mild cases of anterior poliomyelitis. In my first case there was a history of a feverish attack, and on the child being taken out of bed again and allowed to walk, the lameness and weakness was noticed.
Case of Primary Atrophic Muscular Dystrophy (Amyotonia Congenita of Oppenheim). (Previously shown.)

By H. C. Cameron, M.D.

I showed this little girl, aged 4 years, to the Section in February, 1917, then aged 2 years. Because of illness I was not able to be present, but I could see from the report of the discussion, that my view was not generally supported. I have been watching her for two years, and I take the opportunity of bringing the case again. I think we must now agree that it is a case of amyotonia congenita. It appears to throw some light on the somewhat different attitude which neurologists and children's physicians take towards the prognosis of the disorder. We are apt to lay some stress on the tendency toward improvement in amyotonia congenita, whereas most muscular dystrophies and muscular atrophies steadily deteriorate. I think this child's case supplies us with the explanation of the discrepancy. We have here the resultant of two forces—the force of the increased power of equilibrium and increased control over the nervous system, and the force of the steady development of the amyotonia, which is acting in the reverse direction. The resultant of those two forces, at present, is such that the child at the age of 4 years has, with some support, just learned to walk. My impression is that the child's condition has gone down-hill, that the muscles are more flabby, and that the amyotonia is more evident than when I showed the case two years ago.
DISCUSSION ON THE AETIOLOGY, PREVENTION, AND NON-OPERATIVE TREATMENT OF ADENOIDS.¹

Dr. HARRY CAMPBELL.

By "adenoids" I understand a hyperplasia of the pharyngeal tonsil. In many cases, at least, this local hyperplasia is part of a general hyperplasia of the adenoid tissue throughout the body—that condition which, when pronounced, we term the "status lymphaticus." The immediate cause of the hyperplasia is clearly some defect in the plasma bathing the individual cells of the affected adenoid tissue. Cells do not misbehave themselves unless some injurious influence operates upon them from without. What, we have to ask, is the cause of this plasmic defect?

The central factor in the causation of adenoids is intestinal indigestion, due mainly (I do not say entirely) to an excess of imperfectly insalivated starchy food—a condition of things which is traceable to the undue softness of our vegetable food. It is here that the hereditary factor comes in. We know that adenoids tend to run in families: this is chiefly because in these families the bowel has less than the average power of coping with an abundance of carbohydrates.

How does this intestinal indigestion give rise to the plasmic defect referred to? Chiefly, I think, by the absorption of intestinal poisons. The defect is a positive rather than a negative one. Adenoids are as common, or nearly so, in the overgorged, pampered children of the rich as in the comparatively underfed children of the poor. In the former the blood is, if anything, surcharged with foodstuffs. I therefore

¹ At a meeting of the Section, held January 24, 1919.
assume a toxic factor to be in operation—a toxæmia secondary to intestinal indigestion. As a result of this toxæmia the tissues are saturated with poisons and nutrition suffers. In consequence of this the resistance to microbial infection is lowered, especially noticeable in the case of those microbes which give rise to catarrh; there is a pronounced tendency to catarrh,—of the nasal passages, nasopharynx, bronchi, intestines, &c. We have, in fact, the condition of things which Dr. Cameron has termed the "status catarrhalis," the clinical equivalent in his view of what is known, post mortem, as the status lymphaticus. The catarrhs are due to invading microbes. These generate toxins, which, when conveyed to the related adenoid tissues, cause the latter to take on hyperplasia. Thus catarrh of the mucous membrane related to the pharyngeal tonsil tends to cause hypertrophy of the latter,—i.e., adenoids. While I think it probable that the adenoid hyperplasia is mainly caused in this way, we must not lose sight of the possibility that it may likewise be the direct result of the toxic, or otherwise abnormal, condition of the blood already referred to.

An important factor in the causation of adenoids is defective mastication. This may operate injuriously in three different ways:—

1. If the jaws are not adequately used in mastication the nasal passages and nasopharynx fail to develop properly, and it is generally acknowledged that adenoids occur more frequently in those in whom these parts are ill-developed than in others.

2. Vigorous mastication promotes the flow of blood and lymph in the nasopharynx and related parts, and thus tends to establish a healthy condition of the mucous membrane lining them; defective mastication has the opposite effect. I would remind you of the close relation of the external pterygoid muscles to the nasopharynx.

3. Defective mastication, implying as it does imperfect salivary digestion, promotes intestinal indigestion.

I will now briefly consider the subject of salivary digestion. Is salivary digestion a minor function, or is it an important one? Dr. Sim Wallace considers that salivary digestion is comparatively unimportant, that its essential function is to digest the starch which remains in the mouth after a meal. I suggest that salivary digestion constitutes an important part of the digestive process. Some animals, such as the horse, the ox, and the sheep, have no diastasic ferment in the saliva; but books on physiology tell us that in man all the three great salivary glands—the parotid, the submaxillary, and sublingual—are endowed with the power of converting starch into dextrins and maltose (i.e., the
penultimate stage of starch digestion), and it is said that starch digestion goes on for half an hour, or even longer, in the stomach. I find that the total weight of the salivary glands is much the same as that of the pancreas (as 15 to 16).

Let us now consider some of the changes which have taken place in man's diet since pre-cookery times. Before man learned to cook his food he was compelled to masticate all the coarser varieties of starchy foods thoroughly in order to break up the non-digestible cellulose framework and thus liberate the contained nutriment. This implied effective salivary digestion. Now in those times the supply of starchy food was comparatively limited; consequently only a limited quantity of starch entered the bowel, and this not until it had undergone efficient salivary digestion. We come next to the early cookery period, before the introduction of agriculture. By means of cookery man greatly increased his supply of starch, for the application of heat breaks up cellulose much more rapidly than can be done by mastication. Hence the mechanical function of mastication became less needful and the starch was, in consequence, less insalivated than in pre-cookery days; in short, with the advent of this period, more starch entered the bowel, and this in a less digested form. With the introduction of agriculture the amount of available starch came to be enormously increased, and at the present time starchy foods are so carefully prepared that many of them are swallowed after a mere pretence at masticating, and the bowel is, in consequence, apt to be flooded with starch which has undergone little, if any, salivary digestion; thus intestinal indigestion is apt to be set up. Further, the consumption of sugar has of late years increased enormously.

I now ask your attention to the relative frequency of adenoids among different peoples. I suppose most will agree that they are more common among the British than any other people. Sir William Osler thinks there is more mouth-breathing per acre in England than in any other country. Mr. Cheatle found adenoids in nearly half of a thousand school-children examined by him. And observe this remarkable fact: It is not only among the inhabitants of these islands that adenoids are so prevalent. You find a similar prevalency among British communities all over the world—in Australia, New Zealand, South Africa, India, Canada. One of the worst cases I have met with was in a child who was brought up on the African veldt, under exactly the same climatic condition as the native negro. This shows that dampness is not a primary factor in causation; though it may doubtless act as a pre-
disposing factor by favouring catarrhs. We can obviously rule out heredity in explanation of the prevalence of adenoids among the British at home and overseas.

It is clear that there must be some specific environmental factor operating in the case of the British all the world over. What is it? The one specific factor operating in the case of the British and not in the case of other peoples has reference to diet. Wherever the Britisher goes he carries with him the dietetic habits of the old country. Inasmuch as there is nothing peculiar about the animal food of the British, the peculiarity must pertain to the vegetable food. What is this peculiarity? It consists in this, that our vegetable food is for the most part consumed in a form which does not favour adequate mastication. That the vegetable food of the British is, on the whole, softer than that of other peoples can be proved beyond doubt. You have only to examine British jaws. Jaws do not develop of themselves: they require exercise in the shape of adequate mastication. Now animal food requires little mastication. The carnivora do not—cannot in fact—masticate their food: mastication is a lateral grinding action of the lower teeth against the upper, and the upper and lower teeth of the carnivora, when opposed, interlock and admit of but little lateral movement. The teeth of these animals are adapted for crushing bones and tearing flesh, not for grinding. We may, therefore, safely conclude that the defective development of the jaws among the British is due to the fact that the vegetable food of the British does not provide adequate mastication. It is true that the Esquimaux, who consume but little vegetable food, have strong jaws, but it has to be remembered that these people consume much of their animal food in a hard, frozen condition. The defective development of the jaws among the British is not always secondary to adenoids—it is common in those who have not suffered from this affection. Its great frequency is shown by the fact that it is very rare to meet with a modern British jaw in which there is sufficient room for a normal eruption of the teeth. On the other hand, the peoples of the European Continent, to go no farther, have, in the main, a well-developed maxillary apparatus.

These considerations would lead us to expect a priori that the vegetable food of the British fails to promote adequate mastication. This a priori conclusion is confirmed by an actual comparison of their vegetable food with that of other peoples. The chief fault lies with our cereal foods, which are consumed in a spongy, pappy, and pultaceous form. We eat too much spongy (non-crusty) bread and pultaceous
pudding, and too little raw vegetable food in the shape of salads and fruit. Of late years we have, moreover, consumed large quantities of sugar.

As a result of this kind of diet the maxillary apparatus (including the nasal passages, nasopharynx and salivary glands) does not develop properly; the local flow of blood and lymph is not adequately stimulated; starchy foods do not undergo adequate salivary digestion; and an excess of wholly non-digested starch (as well as of sugar) passes into the bowel, there to give rise to indigestion and putrefaction (as shown by the malodorous motions). Then follow toxæmia, malnutrition, diminished resistance to infection, catarrh, adenoids.

The central factor is, I repeat, intestinal indigestion. A sound intestinal digestion—no adenoids. I do not mean to say that a large amount of starchy food necessarily causes adenoids. This depends whether or not the bowel can cope with it. Some have a greater capacity in this respect than others. We have, moreover, to take the total dietary into consideration. The Hindus and South African natives consume an abundance of rice and mealies respectively, but their general dietetic customs are such as to promote normal intestinal digestion; and that they are good masticators is shown by their admirable maxillary development. Nor do I wish to contend that there is only one form of intestinal indigestion capable of generating the adenoid habit of body; this may doubtless at times be brought about by a diet in which there is no great excess of starch or sugar.

The most effective way to diminish the prevalence of adenoids in this country is to alter some of our dietetic customs. Crusty bread should be substituted for the soft spongy article—the class of food typified by the pernicious bun should be banished; puddings should be limited to one or two days in the week; and the quantity of sugar should be kept within reasonable limits. On the other hand, more raw vegetable food should be consumed in the shape of salads and fruit.

Dr. EDMUND CAUTLEY.

I feel in the awkward position of one from whom some new light is expected on the subject of adenoid hypertrophy in the nasopharynx, whereas I can only remind you of the effects generally attributed to it and indicate those of most importance and most open to discussion.

It is on these effects that the decision as to the appropriate
treatment must be based. Their gravity must be very carefully estimated, for the presence of even marked hypertrophy does not always necessitate operation. Consider, for instance, the frequent history of recurrent attacks of nasal catarrh. Here we have an excellent example of a vicious circle. Adenoid hypertrophy, or the constitutional dyscrasia associated therewith, increases the liability to nasal catarrh, and each attack of catarrh is apt to lead to an increase in the hypertrophy. But nasal catarrh is often due to other causes and quite independent of adenoids. In such cases it is unlikely that operation will cure, or much reduce, this liability to catarrh; more especially in those patients in whom there is no evidence of adenoids except a moderate enlargement of Luschka's gland, found by the barbarous method of exploring the nasopharynx with the finger. I protest against this method of examining children, for it is painful, alarming and unnecessary. Operation should not be recommended for mere hypertrophy, but only for the effects, local or constitutional, produced thereby. The past history of the patient and a careful clinical examination will reveal these effects, and render even posterior rhinoscopy a superfluity.

It is a remarkable fact that the obstructive effects of adenoids did not attract the attention of the physicians of olden days, many of whom were apparently superior in clinical acumen to those of us who are dependent, more or less, on a laboratory for a diagnosis. I can hardly believe it was due to the recognition of Luschka's gland that attention was drawn to the ill-effects of its hypertrophy. I put forward the suggestion that in olden days the affection was not common nor pronounced, and that its development coincides in point of time with the introduction and increasing prevalence of bottle-feeding and of rickets. Although artificial feeding was begun in the eighteenth century it was very limited in extent until the development of the feeding bottle. In 1783 a cow's horn, with parchment or leather on the tip, was used. Early in the nineteenth century this was displaced by a glass of similar shape, and gradually the modern bottle was evolved, as well as numerous artificial foods. Year by year there has been a steady increase in the number of hand-reared infants and of cases of rickets, though I do not think the severity of the disease is as great as it was twenty-five years ago.

If this view is correct it becomes probable that we must ascribe to rickets many of the ill-effects which are at present thought due to adenoids. This does not exclude the hereditary factor as a concomitant
cause. Undoubtedly there are families with a "lymphatic constitution," that is, an undue tendency to lymphatic hyperplasia. In these families "adenoids" may be well marked and the rachitic element slight or absent. Almost all children with adenoid hypertrophy are rachitic, but rickets is often present without such hypertrophy. In infancy and early childhood there is a relative excess of adenoid tissues throughout the body, compared with the amount present in later life. After the age of four or five years it gradually decreases. We are not justified in regarding this normal lymphatic hyperplasia as a condition of hypertrophy.

The main effect on the child is an increasing nasal obstruction, leading to mouth-breathing, imperfect respiration and oxygenation, and interference with the functions of the nose and nasopharynx.

Nasal obstruction depends on many causes besides adenoids. In the new-born infant the nasal passages and nasopharynx are very narrow; still more so, if the nasopharyngeal vault is low and the palate high, as in Mongolism, crétinism and dolichocephaly. In such cases a normal amount of adenoid tissue may be sufficient to cause nasal obstruction. Or it may be due to congenital nasal stenosis, deflected septum, cicatricial stenosis, new growths or foreign bodies. But the main cause is a post-nasal catarrh—perhaps limited to the nasopharynx and giving no external evidence of a "cold"; apt to be overlooked, unless the throat is examined in a good light; decreasing in dry and recurring in wet weather; and often associated with a small, narrow nasopharynx, crowded teeth and receding chin. It is common in the rachitic, because of their undue tendency to catarrh, and it is often difficult to decide whether the attacks depend on rickets or adenoids.

Let me refer to the "adenoid facies"; the open mouth and resulting vacant look, the long narrow face, receding chin, pinched nostrils and projecting upper lip often seen in patients with nasal obstruction due to adenoids. I think it is now recognized that this facies is also associated with nasal obstruction due to other causes, and I suggest for discussion that it is not dependent on nasal obstruction per se, but, more often, on the rachitic factor so often present. It may be present in patients with no adenoid hypertrophy, and absent sometimes even when adenoids are exceptionally abundant. I am inclined to take the view that the maldevelopment of the nose and nasopharynx is due to a congenital factor or to rickets, and that it is only secondarily influenced by nasal obstruction.
Various types of malformation of the jaw have been put down to nasal obstruction:

(1) The arch of the lower jaw is larger than, and surrounds, that of the upper jaw; the lower jaw is prognathous and the palate high.

(2) The upper jaw is small and atrophic, and the curve of the alveolar arch is small; the palate very high, and the turbinal bones approximated to the septum.

(3) The sides of the upper jaw are approximated and push forward the incisor teeth. The sides of the lower jaw anterior to the molar teeth may be similarly approximated.

(4) The lower jaw is normal but the mouth cannot be closed fully (open-bite) because the upper alveolar process, anterior to the molars, deviates upward at a considerable angle: the palate is V-shaped.

All these deformities may develop in cases of rickets, even in the absence of nasal obstruction. The “comforter” and the teat of similar shape used on some bottles are accessory factors in their production, acting partly by pressure and partly by the suction necessary to obtain food through such a teat. Is it going too far to assert that they are the consequence of rickets and not of nasal obstruction? Supposing them due to the latter cause, the explanation usually advanced is that there is excessive external atmospheric pressure. It is asserted that in mouth-breathing the current of inspired air abstracts some of the air from the nasal chambers, rarefies the remainder, and reduces the pressure within these cavities and on their walls. It is difficult to regard this as a sound hypothesis, unless there is present a serious amount of anterior nasal obstruction preventing air entering through the nostrils. It would be more justifiable to ascribe the deformity to maldevelopment from disuse. The same arguments may be applied to palatal deformity, for it often exists although there is neither mouth-breathing nor nasal obstruction.

Deformity of the chest, on the other hand, is partly due to impeded respiration. Seeing that adenoids, if severe, are associated with enlarged tonsils, it is obvious that both nasal and oral respiration are interfered with. Hence we find that on inspiration the lower ribs are indrawn, and there is retraction or depression of the manubrium sterni; or the ribs are indrawn along the costo-chondral junctures and the child becomes pigeon-breasted. I doubt this condition becoming very marked in degree unless there is associated rickets.

Mouth-breathing interferes with suckling, for the child has to drop the nipple in order to breathe. It also leads to dryness of the mouth.
and throat and difficulty in swallowing, and it is an accessory factor to early dental caries. Noisy breathing and snoring are common. The lack of oxygenation leads to general ill-health and imperfect development. It is extraordinary how some children improve after operation, although there may be little evidence of respiratory obstruction in the shape and expansion of the chest. Head sweating, “snatchy” sleep, night-terrors, nocturnal enuresis, morning lassitude, headache, deficient growth and other secondary effects are due to defective oxygenation and the deep sleep induced by it. Still it is just as rash to promise to cure enuresis by the removal of adenoids as it is to promise the cure of of recurrent “colds.” Paroxysmal cough and sneezing, stridulous respiration and laryngospasm are set up by local catarrh and irritation. Snuffling, a stuffy dead voice and defective articulation, need only be mentioned. Backwardness and the appearance of stupidity are due to deafness, deficient oxygenation and the open mouth. Gastric derangement may be due to the swallowing of mucus; hence, dyspepsia, anorexia, morning nausea and vomiting.

Perhaps the most important effect of all is the secondary occurrence of ear trouble. There is frequently a history of earache, slight at first, then recurrent and becoming more severe; increasing or recurrent deafness; otitis media and otorrhoea. These results follow on the spread of catarrh to the Eustachian tube and middle ear. The membrana tympani is retracted. It is a remarkable fact that masses of adenoid tissue have been described as present in the fossae of Rosenmüller, the areas around the orifices of the Eustachian tubes, as well as the circumscribed mass known as Luschka’s gland; and yet some surgeons deny their existence and state that this region may be safely left alone. Ear troubles, in the presence of adenoids, are an imperative indication for operative treatment. Many children become deaf on account of neglected adenoids. But it must not be forgotten that otitis media and otorrhoea are very common complications of catarrhal respiratory affections and in marasmic infants; that they are usually mild and get well under simple treatment; and that they do not always indicate adenoid hypertrophy.

Another important effect is adenitis, at first a simple inflammatory hyperplasia, but very liable to become infected with organisms such as the tubercle bacillus. I regard ear complications, adenitis, deformity of the chest, mouth-breathing, recurrent colds and interference with general nutrition and growth, as the effects which warrant surgical interference. If these are absent or slight, it is justifiable to rely on
simpler measures. But I am sceptical about the possibility of curing or preventing them by sneezing exercises or respiratory drill, advantageous as these methods are in clearing away mucus and teaching nasal breathing.

And in the case of respiratory exercises I would point out that they frequently do harm rather than good. I have often found them prescribed for children, although each deep inspiration distinctly increased the deformity of the chest.

From what I have said it will be clear that I regard the preventive treatment as a matter of great importance. In the first place, I insist on breast-feeding, for adenoids and rickets are much more prevalent and severe among bottle-fed children. Secondly, all measures for the prevention of rickets and nasal catarrh must be adopted—viz., a suitable diet, hygiene, exercise, fresh air, &c. Thirdly, guard the child as far as possible during the first five years of life from exposure to "infectious colds." The importance of nasal catarrh in infants is never sufficiently realized by parents and nurses. Each such cold increases adenoid hypertrophy and the susceptibility to fresh attacks. It should be regarded as a serious, not a trivial, ailment and treated efficaciously. The hardening process should be adopted with discretion. In view of the climatic conditions the tendency to catarrhal affections is unavoidably great, but the individual used to fresh air, frequent changes in temperature and cold bathing, is much less susceptible than one coddled up in centrally heated chambers. The encouragement of breast-feeding and the prevention of rickets will help very greatly in the prevention of adenoids and their immense accumulated damage to the individual and the nation.

Dr. SIM WALLACE.

I once wrote a paper on nasal obstruction, and proceeded to some extent on the lines upon which Dr. Harry Campbell has gone to-night. But to all intents and purposes, I have given up every idea that I had at that time, as having any particular importance with regard to the causation of adenoids. I would however still like to associate myself with Dr. Campbell's views in regard to the effect of mastication in broadening the nasal passage.

I think the causation of adenoids is an extremely simple thing to analyse. I believe, first of all, that there is an exciting cause, and this has been indicated by both the previous speakers to-day: it is believed in by them, and by most authorities. This exciting cause is repeated or
prolonged catarrhal infections or inflammations of the nasopharynx: when these persist over a long time, it generally ends by having a surgeon to operate for adenoids. The predisposing cause of such infection is no doubt a depressed vitality of the mucous membrane of the nasopharynx. This has long been recognized by laryngologists. De Schweinitz and Randall,\textsuperscript{1} referring to acute catarrhal pharyngitis, say: "Exposure to cold really means lessened resistance of the tissue to germ vitality and germ entrance into the substance of the membrane." And, referring to acute naso-pharyngitis, they say: "Exposure to cold and damp plays the chief exciting rôle. Here also should be borne in mind what has already been said, regarding such exposure as related to temporary impairment of the tissue vitality. As occurring in very young patients, we generally find acute naso-pharyngitis associated with more or less enlargement of the pharyngeal tonsil, which in turn may have resulted from repeated attacks of acute rhinitis." So far, then, I think I am in complete agreement with what is pretty generally accepted as true. If cold and damp is a cause of reducing the vitality and the germ resistance of the mucous membrane of the nasopharynx, we should expect cold and damp air would bring about a tendency to catarrhal infections. What do we find? When the days begin to get cold and damp, there are many catarrhal affections, and in a cold and damp climate like this they continue through the cold and damp period of the year. It is stated that adenoids are much more common in cold and damp climates such as our own, and in climates which are not altogether cold and damp adenoids are commoner in the cold and damp districts, as for example the Lake districts of America. On the Continent one of the places where adenoids are most prevalent is Sweden. And Dr. Harry Campbell once drew my attention to the fact that it was in the northern part of Italy where adenoids are to be found, being relatively rare in the southern parts of that country. I have read, however, that adenoids are very rare generally in Italy. As to the prevalence of adenoids wherever the British race may be, I do not think that contention is quite correct. Where the climate is continuously warm, without excessive variations in temperature, you do not find adenoids very prevalent. I do not think you find them nearly so often in Anglo-Indian children, though they are most frequently very delicate. The most certain way to predispose children to chronic catarrhal infections is to force them to sleep in a cold and

\textsuperscript{1} De Schweinitz and Randall, "American Textbook of Diseases of the Eye, Ear, Nose and Throat," 1899.
damp atmosphere and then send them to school, where they will pick up an infection. First ensure reduced vitality, then infection, and you are pretty certain to get a succession of catarrhs throughout the winter.

Considerations such as these led me to make a statistical examination in reference to children who were condemned to sleep in cold and damp atmospheres, that is to say, those who slept in cold air through their parents pulling the windows down and admitting the cold, damp, sometimes foggy, air throughout the whole winter. I took extreme cases, in which the parents were real "fresh-air fiends." Of forty-nine such children, twenty-two had to be operated upon for adenoids. I took statistics with regard to children who slept with the windows shut, and among them I could hardly find any adenoids at all. I have also put doctors on the track, and they can hardly find them either, at least they have not supplied me with statistics. Among sixty-nine children who slept with closed windows, I found only two had adenoids.

It seems to me obvious that the proper non-operative treatment is to bring up children in such a way that when the temperature drops quickly, as it so generally does at night, they will be safeguarded against that drop, not only because of the cold but also because of the increased humidity. Further, when children have contracted a catarrh it is frequently desirable to have an open fire lit in their bedrooms so that they may be properly ventilated, and the humidity reduced and the temperature not allowed to drop too much. You may think that cold and damp have not much to do with catarrhal inflammations, but if you look up the death-rate statistics with regard to bronchitis and pneumonia, you will see how they soar up when the weather becomes cold and damp.

I have abandoned the theory that a great deal of this trouble was due to imperfect mastication: I now think it is the unnecessary exposure of the child to cold and damp during the night, and then sending the child into germ-laden surroundings.

Mr. W. Stuart-Low.

Dr. Harry Campbell said sufficient attention had not been given to this subject, but among laryngologists a great deal of attention has been devoted to it. We are always studying it, but we have not yet arrived at the conclusion we would wish. We do not yet know the exact cause of the presence of adenoid growths, one reason being that we have ceased to consider it of importance since we have such a perfect cure for
adenoids by means of operation. Dr. Harry Campbell mentioned how adenoids constitute a horrible disease, as such distressing consequences may follow, but I do not consider this is so, as it is remediable by one skilful sweep of the curette—I say “skilful”—by which the whole trouble can be removed.

What is the condition in the adenoid state? It is an inherited tendency to a hyperplasia of adenoid tissue of the nasopharynx. What puzzles me is, why do we get so many cases of adenoids among children who have been fed on the breast—viz., Nature’s nourishment? Dr. Harry Campbell asserted that carbohydrates constitute one of the chief causes of adenoids, but I do not think this has any bearing on the causation, one reason being the large number of breast-fed children who have come through my hands for operation and in whom the removal of very large masses of adenoids has effected a complete cure. If a child fed on perfect food develops adenoids, carbohydrate food cannot be one of the chief causes. I have preserved the adenoid masses removed from such children (in some cases these have been enormous)—a strong argument in favour of hereditary cause before mentioned. He also says that toxaemia, a result of gastric disturbance from improper feeding, is another cause of the condition, but I cannot entertain this at all, as in my opinion toxaemia is the result of swallowing septic material and pus generated in the nasopharynx from the presence of adenoids. Quite a number of stomach and other disturbances arise from swallowing septic material, as man, being an erect animal, swallows the septic material, which passes direct from the nasopharynx into the stomach, the result of his erect position. I do not think defective mastication has much to do with the generation of adenoids.

With reference to Dr. Sim Wallace’s theory that closed windows at night prevent the generation of adenoids, I would only remark that this would mean choice between adenoids and tuberculosis, as the exclusion of fresh air has been proved statistically to increase tuberculosis in children.

With regard to Dr. Cautley’s remarks on the persistence of catarrhalism and non-effective operations for adenoids, I would only say that this is not the case in my experience if the operation be properly performed. Where catarrhalism recurs, in my opinion it can often be traced to the patient’s surroundings and to his coming in contact with nurses and others, who reinfect the child by kissing him indiscriminately, a practice which I consider reprehensible and which ought to be suppressed as much as possible instead of everyone thinking
there is a perfect right to kiss the baby. In one case where catarrhalism existed after operation I found the nurse who had attended the child had very profusely discharging ears and very marked pyorrhea alveolaris, conditions which would be quite certain to reinfect the patient. With regard to the subject of inducing sneezing in children, mentioned by Dr. Cautley, I condemn this method of trying to prevent the development and to bring about the cure of adenoids as reprehensible, and about this I wish to be as emphatic as possible, as I believe the practice has been encouraged by some who ought to know better. The inducement of sneezing in children brings about engorgement of the upper part of the nasal passages, which means enlargement of the middle turbinals, which is likely to be fraught with very serious consequences.

There is a tendency on the part of many physicians to defer the operative line of treatment in favour of temporary measures, one of the worst consequences of which is liability to acute inflammation of the middle ear, and the launching of the child on that sea of troubles, chronic suppuration of the middle ear.

Dr. Camac Wilkinson.

I can speak on the subject of adenoids, not only from the point of view of the physician, but also from that of the surgeon, for in Australia, where we have few opportunities of investigating rickets, we have abundant opportunities of dealing with adenoids. I had about seventeen years' experience in one of the largest throat hospitals in Australia, where I had to deal with thousands of cases of adenoids, and I am bound to say the remarks I am most in sympathy with are those of Mr. Stuart-Low, because, after all, the most important matter in regard to adenoids is treatment. In the somewhat speculative and not yet proven views to which we have listened, there is room for the belief that these conditions will disappear of themselves. I think that is a fallacy which we should resist as much as possible. The treatment of adenoids will prevent a large number of cases of ear disease. When suppurative ear disease runs a chronic course and adenoids have been overlooked in the patient, it is surprising how the ear condition improves when the adenoids have been dealt with. A common idea is that adenoids belong to one particular time of life, especially childhood. But one operates, sometimes, on children who are six months old. That is
incompatible with some of the ideas we have heard expressed to-night. On the other hand, I have operated on adenoids in people over 40 years of age. An unfortunate thing is that when the method of dealing with adenoids is so satisfactory, if carried out by an expert, the disease should ever be tampered with. Even physicians dealing with children who have adenoids allow them to grow into adult life with this disability to handicap them, whereas there need have been no danger at all if the operation had been properly done in earlier life.

I would only add that my experience in Australia is not that either deficient mastication, or intestinal indigestion, or rickets are the only factors, though they may favour the condition. There is often a constitutional or family tendency in causation. When a physician comes upon a case of adenoids, he should so have it dealt with that the whole outlook for that child is altered for the better; the child should not be allowed to carry with it a condition which will probably be a source of injury to the chest wall and other organs.

The President.

We are all aware of the advantage of operative treatment in many cases, but the point for discussion to-day is the non-operative treatment, among other matters.

Mr. J. H. Badcock.

In treating German prisoners at Richmond Military Hospital for broken jaws, I was surprised to see the exceedingly good development of the men's mouths. I have been looking at mouths for thirty years, and there I saw far more perfect dentures and better jaws than I had ever seen before. I was told that these prisoners had petitioned that they might be supplied with less meat and more breadstuffs and vegetables in their dietary, and that the greatest delicacy they could receive from home was black bread. At about the same time I was told by a general commanding a labour battalion in France, composed largely of Germans, that the Germans had made a similar petition to him; they had more meat than they could do with, and they wanted more vegetables. I do not draw any inference from those facts, but I think they are interesting. I do not know whether adenoids are less prevalent in Germany than in England, but it is my impression that
they are. Among the poorer classes in Germany black bread is one of the main articles of diet. Another point about the German is, that practically always he is breast-fed.

**Dr. Eric Pritchard.**

I was sorry to hear that Dr. Sim Wallace had abandoned the theory he formerly held, because I thought there was some truth in it. To hear, however, that fresh air is a cause of adenoids makes one tremble to think of the results of putting this theory into practice. I was also disappointed to hear Dr. Campbell suggest that adenoids were due to faulty digestion of starch, because my experience, so far as adenoids are concerned, is largely with young infants, to whom this explanation cannot possibly apply, since the majority of them have not then been fed on starch. Infants fed on excess of Mellin's food, which is practically maltose, are particularly liable to present hyperplasias of lymphatic tissue.

Many of the apparently incompatible views we have heard are reconcilable if we consider the fundamental significance of hyperplasia of lymphatic tissue. Why should there be hyperplasia or hypertrophy of this tissue? What is the function of lymphatic tissue? I take it its function is, chiefly, bactericidal in character. Wherever there is relatively excessive bacterial invasion, there is a hyperplasia of lymphatic tissue to meet it, to protect the body from a serious danger. Therefore it seems logical to suggest that there will be hyperplasia of lymphatic tissue under two conditions. First, where normal amounts of lymphatic tissue are unable to perform this bactericidal function, the sort of condition which we find in the "status lymphaticus," a condition which we so often meet with before the infant begins to consume starch at all.

Why are there such great differences as to the power of performing this bactericidal function I have attributed to adenoid tissue? Because the function of lymphatic tissue to kill bacteria depends on a protein metabolism, not on a carbohydrate metabolism. All toxins and antitoxins contain a nitrogen basis: their elaboration and destruction must depend on a nitrogenous metabolism, not on a carbohydrate one. But if the infant has been brought up chiefly on carbohydrates, soluble or insoluble, the lymphatic tissue cannot so efficiently perform this bactericidal function. For hat
reason I believe the fault is connected with an excessive carbohydrate, not necessarily with an excessive starchy diet.

The second condition on which hyperplasia of lymphatic tissue depends is not on a defective power to do its work, but rather because the amount of work it has to do is more than normal lymphatic tissue can be expected to perform. If the killing of bacteria is the chief function of lymphatic tissue, hyperplasia of this tissue in the nasopharynx will depend on the number of bacteria there are to kill.

A very important factor in connexion with the work which is thrown on the lymphatic tissue of the nasopharynx is the efficiency of the drainage of the posterior parts of the nose. Where drainage is bad, there will be collections of mucus and of bacteria, and the work for the lymphatic tissue will be heavy. The kind of drainage there will be depends, largely, on what Dr. Campbell drew attention to, namely, the wideness and other anatomical relationships of the nasopharynx, and, possibly, on the capacity of the child to get rid of its mucus by sneezing or blowing its nose, and to some extent also in the exercise of the function of mastication.

I was sorry Dr. Campbell thought the digestion of starch in the mouth is such an important function. I feel convinced that the view held by Dr. Sim Wallace that the function of the saliva is to clean the mouth, and not to digest starch, is the correct one.

The President.

There is a point in regard to the prevention of adenoids I should like to bring forward. In 1902 I read a paper before the Society for the Study of Disease in Children in which I suggested a plan of treatment for children suffering from threatened or commencing adenoids. I pointed out that the method was not intended to apply to well-marked cases or to those with any complications such as deafness, enlarged lymphatic glands, &c. For these of course removal is the only cure. Many infants are born with, or acquire early in life, an excess of sub-mucous tissue in the postnasal space, which renders them liable to attacks of catarrh on slight provocation, and each such attack increases the amount of such tissue, till at length the typical symptoms of adenoids are produced. If we can lessen the tendency to catarrh and

\[1 \text{Rep. Soc. Study Dis. in Children, 1901-2, ii, p. 134.}\]
also diminish the post-nasal swelling we improve nasal breathing and prevent the development of adenoids. Sir F. Semon pointed out that in many cases only a small, soft, and gelatinous mass is to be felt in the posterior nares, and in them there may be only a very moderate or hardly any organized hypertrophy of the lymphoid tissue, but on the slightest provocation so much congestion and engorgement occur that for a time all the symptoms of genuine adenoids may be simulated. In cases like these I have been in the habit of applying a paint composed of equal parts of glycerine and liq. ferri perchloridi by means of a brush on a metal handle, which can be bent to a suitable angle and passed through the mouth behind the soft palate. This may be done twice weekly at first, and later on once a week, as the symptoms abate. The treatment of course cannot be applied to children under 6 or 7 years old for obvious reasons, but it is by no means as unpleasant as it sounds; a little practice enables one to apply it rapidly and with very little discomfort. It is so long now since I first began this method of treatment that I have been able to follow up some of the cases to adult life, and it seems that they have been entirely free from any of the usual results of adenoids. I always supplement the treatment by breathing exercises to abolish mouth-breathing and develop the lungs; without which I am sure no treatment is of any avail, and I believe a course of sea air is of great value in after treatment.

One speaker mentioned the absolute safety of operation as a cure for adenoids. I, as one who has lost one of my children as a result of such an operation, cannot agree with him. I know that the percentage mortality from the operation is very low, but that is no consolation to one who has suffered from it as I have done. Knowing, as we do, that adenoids are merely, in some cases, an expression of status lymphaticus, or something akin to that, there are many cases in which the adenoids are quite unnecessarily treated. It has been stated at this meeting that adenoids do not disappear in later life. I have not made investigations on the point, but I would like to know whether that statement is correct. I believe that with adult life the swellings previously existing in the post-nasal space are very likely to lessen, and that, at all events, the increased size of the cavities at that period makes such swellings much less important and inconvenient. Mild cases, therefore, are likely to be of little importance, if untouched, by the time puberty is reached, and I feel sure that some unnecessary operations are performed on such mild cases.
Dr. C. P. Lapage.

I have watched many cases of adenoids for a number of years, and I have long wondered why the condition should be so common. When the nasopharynx is very small, the child has a marked adenoid facies and usually a marked degree of tonsillar enlargement. These cases occur in any climate, and in different parts of the country. But there are also many cases in which there is not so much mechanical obstruction or blocking of the nasopharynx, not such large adenoids, and no enlarged tonsils at all.

I think that in most instances the cause of adenoids is infection. There are exceptions, such as cases in the first few months of infancy, which one has to send to the surgeon for operation. I agree that anything which, like intestinal indigestion or rickets, lowers the resistance of the body, adds to the possibility of infection. Still, I do not think I have seen more cases of adenoids in subjects of chronic indigestion and rickets than among ordinary children. It is well to remember what Dr. Sim Wallace has said, but he must add infection. There are now many more opportunities for infection for children than formerly, such as schools and the increased facilities for transport. And though I do not agree that cold air is the sole cause, I agree that when combined with opportunity for infection from proximity to a number of other children it increases the danger. Therefore for the prevention of adenoids, our main aim should be to prevent infection, and keep the nasopharyngeal channels clear.

It is not right, I think, to say you should operate on all cases of adenoids. I am often asked for my advice on the question of operation. There is some danger and I also have had an unfortunate experience where I advised the operation in the case of a relative. In many instances, if you remove the child from infection, and put it in a good climate, you secure a good result. Recent methods seem to promise well if acted upon properly. I have not much experience of them because I have been away, but Dr. Hickling, who has been doing my work for me at my out-patients', has had a number of children coming down and having nasal exercises under proper supervision, and from personal observation I do not doubt that there has been improvement; it does not involve any such thing as blowing snuff into the nose, or sniffing it up; it is simply a stimulation of the periphery of the nose with a harmless powder. They are not severe cases with large tonsils and the adenoids
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facies, but cases of adenoids with chronic pharyngeal catarrh. If there is deafness, or such a succession of colds as to lower the health, I advise operation.

Dr. H. C. Cameron.

The important point in regard to adenoids is to raise the resistance of the individual against catarrhal infection. The problem is to devise the best possible hygiene for the infant. That crowding together in insanitary houses, with bad ventilation and shut windows, is a potent cause of infection we know: so, too, is over-clothing.

The point raised by Dr. Harry Campbell in reference to over-feeding, especially by carbohydrate food, interested me very much. I have come to the conclusion that the high proportion of bread in the diet of children of the poor between 2 and 5 years of age is often responsible for the hydramic condition of the body, and the ease with which catarrh is contracted. Some of these children were found to be eating 16, 18, even 20 oz. of bread a day. The unsatisfactory diet of the children is, largely, an economic question. It has been the practice in this country to legislate for a cheap loaf and cheap sugar, and it is difficult to make out where these children get the necessary fat. Excess even of suitable food is important too. I know nothing on which there is a greater divergence of view than as to the amount of food which is suitable for a child. If you go, as I have done, to two nurseries to investigate the point, you may find that one child is receiving three times as much as the other. The overfed child is prone to repeated pyrexial attacks, which in a sense are reactions in its own interest, if only because for the time food is refused.

The question of bottle-feeding, which was mentioned by Dr. Cautley, is also one on which I have thought a good deal. I, too, have associated the increase of adenoids with the growth of bottle-feeding, but I had concluded that this too was due to infection. The great damage which bottle-feeding does to infancy lies in the lowering of resistance to infection. And with want of hygiene and excessive and unsuitable diet, we have to couple variations in susceptibility due to heredity. I do not think there is any one invariable antecedent which we can label as the cause of adenoids.

I was also interested in Dr. Cautley's remarks as to the relation between rickets and adenoids. I should have said that rickets and adenoids were distinct reactions of children of different types of heredity.
to repeated infections, that in one the result of catarrh is hyperplasia of lymphatic tissue, and in the other interference with the activity of the osteoblastic tissue, of the enamel organ of the teeth and so forth. In both dietetic defect is a main contributory cause, leading to a lowering of immunity and repeated infection.

We shall continue to meet with large numbers of cases of adenoids so long as the hygiene of our children is so bad. The growth of adenoids indicates that the soil is faulty. No child in perfect health has adenoids. To remove the vegetations is easy and often necessary. To correct the nutritional disturbance which fostered their growth is very difficult. That is why it has been chosen as the subject for discussion this afternoon.

(Discussion adjourned to the next meeting, February 28.)
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President—Dr. J. Porter Parkinson.

DISCUSSION ON THE ÆTIOLOGY, PREVENTION, AND NON-OPERATIVE TREATMENT OF ADENOIDS.  

Dr. Irwin Moore.

My remarks will be from the standpoint of the laryngologist and rhinologist. By "adenoids" we understand a chronic hypertrophy or hyperplasia of the lymphoid tissue (Luschka's tonsil) in the nasopharynx. It is no new disease, for the writings of Meyer and others show that knowledge of the condition dates back to the early part of the sixteenth century, and a reference to it can even be found in the writings of Hippocrates. This tends to dispose of the idea that civilization with its present day methods of feeding is the chief causative factor in this disease.

With regard to heredity as a predisposing cause. I think we all agree that adenoid hypertrophy is not a congenital condition; the unknown factor of heredity appears to be the inheritance of a lowered vital resistance that predisposes to an abnormal catarrhal inflammation of the mucous membranes and increase in size of lymphoid follicles all over the body. This inheritance is probably the result of tuberculosis, syphilis, gout, alcoholism, impaired health or various other diseases in the parents. A lowered vital resistance and an exciting or local cause, such as an attack of catarrh or an infectious fever are therefore all that are necessary to produce this hypertrophy. We know that lymphoid tissue distinctly increases in size and vascularity under such influences, and that children are most susceptible to cold and infection, especially in damp and cold climates such as prevail in this country.

1 At a meeting of the Section, held February 28, 1919.

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Hypertrophy of the adenoid tonsil is believed to be a local manifestation, in a minor degree, of the "status lymphaticus," which we know to be a condition of general disturbance of the lymphatic system, in which an increase in size of all the solitary follicles in the body takes place. We find rickets associated with it in 90 per cent. of cases. That hypertrophy of the adenoid tonsil is not congenital is shown by the age-incidence. Though this hypertrophy may occur as early as the third month, the usual period is from the third to the fifteenth year, the greatest number of cases occurring between the third and fifth years. Post-mortem examinations of 100 young infants in a New York foundling hospital revealed no instance of hypertrophy of Luschka's tonsil under 1 year of age.

Food and surroundings can have but little influence as exciting causes, for "adenoids" occur in the children of both the rich and the poor.

There is one important point in the histology. The enlargement is a true hyperplasia of Luschka's tonsil, not only of the normal lymphoid tissue, but, in many cases, of the connective tissue, also the increase in amount of connective tissue varies and bears no relation to the age of the patient. It is important to note that the majority of cases of fibrosis occur in the first decade and not necessarily in older people and that such fibroser adenoids tend to persist. In seventy-five cases carefully examined microscopically by one authority, fibrosis was found to be well marked in thirty, that is to say, the simple glandular tissue had become fibroser, and in this condition had lost all its normal function, and could not resist the invasion of bacteria.

With regard to atrophy of the hypertrophied adenoid tonsil, which in some cases occurs at puberty, it is now known that this is not so constant as was formerly supposed, judging by the large number of cases which persist in later life. It is generally only an apparent shrinkage due to the increased growth in size of the child's nasopharynx, and is not due to atrophy of the hypertrophied tissue itself. Hence, if we await Nature's attempt to carry out this atrophy, and if this fails, the child will have passed through an important developmental period and may emerge with impared health, with all its accompanying risks. We all know what the results of neglected "adenoids" are: rickets, defective growth, obstructed respiration, neuroses, deafness. According to one authority 73 per cent. of children with adenoids have some physical or mental defect, and 30 per cent. to 40 per cent. have ear disease. It is said that it is
rare to find a child up to 10 years of age with neglected adenoids in whom there is no discoverable loss of hearing. From the aural point of view, the soft variety of adenoid enlargement is the most serious, as it is more liable to inflammation and suppurition, and so may cause otitis media.

As we are confined in this discussion to the non-operative treatment, all the specialist can suggest in these cases is to treat the accompanying catarrh; and in that respect it is advisable that there should be more frequent co-operation between the physician and the rhinologist. Breathing exercises are recommended by some people. I have no objection to them after the hypertrophied adenoids have been removed, for then they may be very helpful, but I protest against their use in those cases in which the adenoids have become fibrous, as it is then impossible for breathing exercises to disperse them; moreover, in these cases in which there is nasal obstruction, such exercises must exert a damaging effect on the chest wall. The majority of neglected hypertrophied adenoids have passed the soft stage, and have become fibrous, so that no treatment short of operation can effect a cure.

Mr. H. L. Whale.

Mere removal of adenoids does not appear to cure without breathing exercises: it is a common occurrence to see a child so little benefited by operation on adenoids that months or weeks afterwards one wonders whether they have been properly removed. They are found to have been cleanly removed, but the mother has been doing nothing to make the child a nose-breather. I always point out the importance of that to the mother. Still, those exercises are only a help. There are two facts to be dealt with. One is, that adenoids do, in a few cases, tend to shrink, and even to disappear at certain ages. Another fact is, that they do recur, even after complete removal, if the removal has been carried out at a very early age. So we might have two lines upon which to argue: the possibility of disappearance and occasional recurrence. These can be correlated with the age of the patient. The disappearance, when it does occur, is seen in persons of 20 years and upwards. You may have to operate in extreme infancy, for, needless to say, a young infant cannot be taught breathing exercises. In the case of a patient aged 18 years, you might try orthodontia. Operation may become
urgent at any moment. If the non-operative treatment is not successful, you should choose the most convenient time and circumstances for operating, and, I think, the time of year is very important. For instance, a period of foggy weather is not a good season to select. If sepsis is proceeding from adenoids they should be removed as quickly as possible. We are apt to regard adenoids without reference to the size or shape of the pad: it is a mass of variable shape, and, I think, the shape it assumes in a given case is important. For instance, quite a small pad may obstruct the Eustachian ostia; and the converse is also true. A pad with pitting, as in Thornwaldt's disease, is much worse than a smooth oval or round pad, for in the latter case there are no crevices for the collection of pus. Some men, when starting on this work, rely too much on the use of the finger. The finger cannot tell you as much as a view with the mirror. Students should be better trained in the use of what I regard as the most difficult of all mirrors to manipulate—namely, the post-nasal mirror.

Dr. William Hill.

In reference to the question as to whether so-called "adenoids" are congenital, I have seen infants who appeared to be born with a good-sized Luschka's tonsil, just as some are born with a big nose. I think that, in some very young subjects especially, there is superadded a condition of nasal catarrh in association with swelling of Luschka's tonsil. The common catarrh of the nose and accessory sinuses is shared by the adjoining area, the post-nasal pharynx and its tonsil. The condition of catarrh is supposed to be a microbic one, and to be influenced by climate; but, I think, there is a condition of mucous membrane, especially in children, which is influenced less by microbic than by climatic conditions. There is a tendency to tumefaction, and there is a hygiene for preventing this, which I take to be one of the important things you are discussing. If we are to have a good air-way, there must be evaporation; therefore, if a child is taught to make some effort to breathe through its nose, it will have a less water-logged condition of nose and nasopharynx, and that will be all to its benefit. But it is difficult to teach children to get rid of excessive secretion by effectively blowing the nose, and getting a good blast of air which will prevent the nasal mucosa getting water-logged. Tonsillar enlargement does not tend to clear up if the child is subject to nasal catarrh. There
are, however, many children with normal noses who suffer from enlargement of Luschka's tonsil. I do not regard that enlargement as often a congenital one, but some children, especially rickety children, have a very small nasopharynx, and therefore, relatively, Luschka's tonsil occupies more of that space than in a child with a well-developed nasopharynx. That will influence air pressure, and lead to changes in the ear. All to the good, therefore, if members of the Children's Section can devise means by which children learn to breathe well through the nose, and can be kept free from catarrh, and so forth.

But there is necessarily the microbic factor to be taken into account as an exciting cause of "adenoids." In this country the responsibility for the enlargement of a Luschka's tonsil, which remains permanently enlarged and usually becomes fibrosed, must be largely due to attacks of the exanthemata. To attempt by breathing exercises or by medical means to reduce an enlarged Luschka's tonsil which has undergone fibrosis is about as futile as to expect reduction in an enlarged and obviously fibrosed faucial tonsil by sending the patient to Margate. It has come to be regarded as axiomatic that if you meet with enlarged tonsils in children which do not go down with the administration of cod liver oil and iron and a visit to Margate, or similar change of air, then they are fibrotic and the only thing to do is to extirpate them. And in the case of enlarged Luschka's tonsil, I think, we often waste much valuable time, even in very sickly children in whom operation is sought to be avoided, by trying to induce this and the other tonsils to shrink, when it is probable that there is a large element of fibrosis present. I should be glad to know in cases of haemophilia and of marked anæmia and debility, how these children could be improved without operation, but, so far, I have been much disappointed. The rhinologist is not thirsting for blood, but I am inclined to throw cold water on the suggestion that much can be done in the general run of cases by means other than operative, and while waiting valuable time is being lost, because we have to consider the question of deafness and other sequela.

Dr. W. H. Kelson.

We can clear some of the ground by classifying these cases of adenoids. It cannot be denied that what is called a bad case of adenoids—viz., one in which there is a complete block, cannot be treated successfully by medical means: such a case does not get better unless
operated upon. In the remainder, much depends on the relative size of the nasopharynx. If the nasopharynx be large in relation to the adenoid growths, the growths may not do much harm. Those of us who are constantly using the post-nasal mirror will very frequently see adenoid growths present in a big post-nasal space, which, so far as can be ascertained, have done no harm, and they do not require treatment: there is no obstruction. But there is a third group of cases with small adenoids, in which there is some catarrh set up, and possibly other symptoms, in a modified way, and in these one does occasionally see benefit from medical treatment. It is not always easy to detect such cases. It is not good to put the exploring finger into the post-nasal space, as it upsets the child. It is necessary for someone who can use the post-nasal mirror to diagnose these cases, otherwise serious mistakes may be made. I think the rhinologist is the only man who can use the post-nasal mirror with any degree of certainty. In this third group, where the parents object to operation, I have seen the adenoids disappear after the use of a nasal wash, and residence at school at the seaside. Still, it must be remembered that this is a numerically small group.

**Dr. H. C. Cameron.**

Referring to the question of atrophy of the lymphoid tissue generally throughout the body, one cannot but be struck, in making post-mortem examinations on children, how constantly a child who is plump and well-nourished, whose body retains a high proportion of water, and whose mucous membranes are succulent, has also a large proportion of lymphoid tissue everywhere throughout the body. It is, for example, the plump baby which has a tendency to intussusception, the baby which has such large Peyer's patches that it is possible that their prominence provides the explanation of the tendency to intussusception. If in contrast we examine the dehydrated wasted body of a child dying from infantile diarrhea and vomiting, we see what the old pathologists called the "shaven beard" appearance of the Peyer's patch, a few black dots representing the fibrous stroma of the adenoid tissue. I have often observed what laryngologists perhaps have not the same opportunities of observing—children with obvious tonsillar and adenoid enlargement who are the subjects of long-continued wasting pyrexial disorders, whose intake of food therefore falls much below their physiological needs. Under such circumstances we can observe how, week by week, the
succulence of mucous membranes and the discharge from them diminishes, and how tonsils, and adenoids, and lymphatic glands tend to shrink up. This observation is in keeping with the common experience that an eczema, the analogous catarrh of the skin, usually disappears during coincident pyrexial infection. The mother says "the rash has struck inwards," but it is the dehydration of the body which has caused the disappearance of the rash. I feel sure that in these plump, over-fed plethoric children we find the maximum reaction in their mucous membranes to infection, and I do not think it is right to omit considering the part which is played by long-continued over-nutrition in the production of an increased vulnerability of the mucous membranes, which leads to a secondary hypertrophy of adenoid tissue. Certainly the removal of the adenoids does not restore health to the children straight away. One can say at once that any child with persistent adenoid enlargement falls short of complete health in many other ways. Removal of the adenoids certainly does a local service, and we are constantly obliged to have recourse to it. The adenoid overgrowth is in most cases the result of a general nutritional disturbance carrying with it a lowering of immunity against catarrhal infections of all sorts.

Dr. Eric Pritchard.

I cannot agree that it is impossible to teach a baby nasal breathing, which is all that breathing exercises profess to teach. It is one of the simplest things in the world, and the younger the child, the easier it is. It is when an older child has acquired the fixed habit of mouth-breathing that the chief difficulties are encountered. The assumption of our laryngologist friends present appears to be that this Section does not seem to think surgical treatment is necessary in a large number of these cases. That is not the point of this discussion. We know the great advantages of surgical interference, and, I imagine, we all refer our suitable cases to the laryngologist with the greatest willingness and confidence. This discussion was arranged to deal with the best means of preventing adenoids by means of a study of the aetiology of the condition. One would have thought, after listening to some speakers in this discussion, that in this country, owing to climatic conditions, adenoids must necessarily develop. This is not the view I take. Hygienic and feeding conditions generally play an enormous part in adenoid development or otherwise. Hygienically treated children may
have some hypertrophy of lymphoid tissue, but if they are badly fed
their lymphoid tissue breaks down and degenerates, and their Luschka’s
and faucial tonsils are unhealthy. As a Section, we do not disagree
with the advisability of surgical treatment of adenoids; we simply want
to arrive at a means of rendering surgical operations unnecessary.

Mr. A. R. Colyer.

In the practice of dentistry we are constantly confronted with oral
deformities, most of which are associated with osseous changes in the
nasal cavity and also with the growth of adenoids. Endeavours are
usually made to correct the mouth condition; but my experience is
that little or no prophylactic treatment is undertaken by the physician
for the incipient adenoid condition, though in a later stage it is the
general practice to scrape away the infected organs.
The cases under consideration enter so largely into dental practice
that any light thrown on the aetiology of adenoids would probably serve
not only to guide the physician to the adoption of suitable measures to
prevent or ameliorate the condition but also to solve the problems
which present themselves to the dental surgeon.

I gather that Dr. Campbell’s views as to the aetiology of the condition
are as follows: The alimentary tract is the original focus of infection
and this infection leads to a lowered resistance and to impaired
functional power in the pharyngeal lymphoid tissue due to an impure
blood supply which is the result of toxins absorbed from the intestinal
tract and primarily caused by improper feeding. Concurrently the
natural stimulus of fresh air is lacking in consequence of the blocking
of the nasal passages by catarrhal conditions. A productive fibrosis is
set up and stagnation areas are created which form a suitable nidus for
the growth of micro-organism. A vicious circle is thus established.
In addition, one should always bear in mind that all congenital defects
of tissues, especially those of syphilitic origin, act as predisposing causes.

My remarks on prevention will be based on the above conception of
the aetiology. It is true that after the removal of adenoids a distinct
improvement takes place in the general condition of the patient. But
the improvement cannot be such as to make good the injury caused
by the disease and some permanent damage to the system remains.
Prevention may be considered under two heads: (1) Individual; (2) national.
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(1) Individual.—Breast-feeding is the most important factor during the early months of life because of the immunization conferred by the milk, and because the milk, being a perfect food, tends to promote normal conditions in the alimentary tract. In addition, breast-feeding follows Nature's methods, and brings into play forces which control normal development, and so maintain the anatomical relationship of the surrounding parts, the formation of a high arch of the maxilla and a deflected septum thus being avoided. Later on a change of diet is indicated by the eruption of teeth. Other speakers have dealt with the question of slop diet, and the consequent feeble masticatory movements which bring in their train loss of muscle pull on, and circulatory stimulus to, the surrounding structures. A loss of stimulus to the stomach muscles arising from deficient mastication is also a point which should be borne in mind. We cannot too strongly emphasize the importance of the thorough mastication of hard foods with the resultant tendency to normal development of the animal, especially as regards its jaws and nasal cavities. I am one of those who believe that food is taken too frequently, and that the stomach is not given the physiological rest which is essential to its well-being. It is also my experience that with a full meal of solids children are often given a half meal of milk. If any considerable quantity of milk is given it seems obvious that the other ingredients of the meal should be reduced. As regards the question of keeping the nasal air passage free, I consider that as soon as the habit of mouth-breathing is observed in a baby, the nasal breathing should be restored by closing the mouth gently with the finger, and, later in life, by means of mouth shields, used at night when the nasal passages are free from catarrh. When mouth-breathing has become a regular habit, and there is contraction of the arch of the maxilla, the arch can be expanded. Whether expansion of the arch stimulates growth in the nasal passages I do not know. As pure air acts only beneficially nurseries, both night and day, should be flooded with fresh air. I recommend besides open windows, free air vents in the upper parts of the doors. Elderly nurses and those suffering from nose, mouth or chest troubles should not be permitted to sleep in the same room as children. With regard to the prophylaxis of nasal catarrh, I have found the use of a salt douche most efficacious. While the nasal cavities are full of fluid the nose should not be blown, lest the germs be driven into the nasal recesses. The fluid should be removed by sniffting or mopping.

(2) National.—The question of "prevention of adenoids" is one of
national importance, because a very large proportion of the population suffer either from the presence of adenoids or are handicapped by damage directly or indirectly caused by adenoids. In fact if there is a clear relation of "cause and effect" between adenoids and intestinal toxaemia, adenoids may be said to play a part in the development of many diseases. I will touch upon this point briefly:—

(1) Micro-organisms developing in the adenoid area and directly infecting the gastro-intestinal tract favour the development of conditions which result in intestinal toxaemia.

(2) Adenoids are associated with mouth-breathing, mouth-breathing with oral sepsis, oral sepsis with intestinal toxaemia.

(3) Adenoids are associated with "lethargy," lethargy with constipation, constipation with intestinal toxaemia.

I believe it has been stated that 95 per cent. of all diseases probably result from gastro-intestinal toxaemia. If this percentage is anything like correct, and if adenoids assist in any marked degree in developing this condition, the case for "prevention of adenoids" is clearly strengthened.

Dr. Harry Campbell (in reply):

I hope members of the Section will try to see the matter from my point of view. I have studied the question for many years, and sought to apply strictly logical methods in arriving at my conclusions. Seeing how prevalent adenoids are among British communities throughout the world, and that the British carry their dietetic customs with them wherever they go, it is difficult to exonerate the dietetic factor from an important share in causation. We should eat less spongy, pulvaceous and pappy food, in the shape of non-crusty bread, buns, scones, puddings, porridge, and the like, and rely more on crusty bread, salads, and fresh fruit, as they do in many parts of the Continent, where ill-developed jaws, adenoids, and bad teeth are much less common than they are with us.

Dr. Eric Pritchard seems to underestimate the importance of salivary digestion. The present tendency among physiologists is to attach considerable importance to the role of salivary digestion, and my own experiments point in the same direction.

I agree with Dr. Cameron that adenoids are not merely a local disease: children suffering from severe adenoids give evidence of a
faulty blood-state and of general malnutrition, and this I attribute to intestinal indigestion, the result of unsuitable food.

Dr. Cautley has pointed out the frequent conjunction of adenoids and rickets in this country. This is consonant with my thesis, seeing that rickets is in large measure due to improper food; that, however, there is no essential connexion between adenoids and rickets is proved by the remarkable fact (referred to by Dr. Camac Wilkinson) that while adenoids are rampant in Australia, rickets is almost unknown there.

The frequency of adenoids among the white population in a warm country, where nature has evolved a black man, who goes about stark naked, proves that a cold, damp climate is not, as Dr. Sim Wallace suggests, an essential factor in the causation of adenoids. Investigation shows that climate has little influence on the prevalence of adenoids, and that the ætiological influence of food is predominant.

Dr. E. CAUTLEY (in reply):

My views may be briefly summed up by saying that the prevention of adenoids is mainly concerned with the prevention of rickets and nasal catarrh: that the best method is to encourage breast-feeding, and proper diet following the breast-feeding, guarding the child against exposure to infection, and treating nasal catarrh seriously. It is a mistake to talk of "adenoids" as a disease, because every child has an excess of adenoid tissue relative to its total body weight, as compared with the adult. Hyperplasia is a relative factor, and is induced by infection and other causes of catarrh, though, no doubt, there are hereditary tendencies thereto. If you guard a child against infection, you will probably guard it against this hyperplasia.

I drew attention to the effects with the view of indicating the cases in which operative treatment was essential, as against those in which medical measures might be relied on. I do not suppose medical measures will cure "adenoids," but the decision as to operation always depends on the degree of hyperplasia and its effects. As every child has an excess of adenoid tissue which easily increases in size, it is absurd to say every child who has got this hyperplasia should be operated upon. But when it is producing definite injurious effects, no physician of any established reputation will be opposed to operative treatment.

A—3b
I also laid stress on the disadvantages of respiratory exercises, and I am sorry to have heard them advocated so strongly in the treatment of this disease. I have frequently had to stop parents persisting with these exercises in children who have been operated upon for adenoids, and who have been recommended by the laryngologist to go on with the course of respiratory exercises. In many of these cases, when the child takes a deep breath the deformity is increased.

There is another point I brought forward, and which I am sorry has not attracted more attention—namely, the question of deformities of the jaw in association with adenoids. I took the view—perhaps partly to stimulate discussion on it—that deformity of the jaw was not due to adenoid hypertrophy at all, but due to the associated rickets.
Section for the Study of Disease in Children.

President—Dr. J. Porter Parkinson.

Congenital Absence of the Lower Portion of the Left Pectoralis Major Muscle and Left Mammary Gland.1

By E. A. Cockayne, M.D.

A. C., aged 4½ years. The father and mother are unrelated and have two other children, both normal. There is no history of a similar condition in their family. The boy is small but well nourished. The left pectoralis major is absent with the exception of the clavicular, and a small part of the sternal portion. There also appears to be some deficiency of the subcutaneous tissue on the left side. The left areola is as large as the right, but the nipple is retracted and the mammary gland cannot be felt. The right mammary gland is palpable. There are no other defects of development. I take it that the absence of mammary gland can be explained by the absence of blood supply, the same vessels supplying the pectoralis major as serve the mammary gland, and so one would expect the mammary gland to suffer too. I shall be glad to hear views as to the underlying cause of this condition.

DISCUSSION.

Dr. Eric Pritchard: I do not know whether Dr. Cockayne suggests that the association of the muscular with the mammary defect makes this case different from the common abnormality which one finds in muscles. In the course of inspection of school children, I have met with many muscular defects, but the most common were connected with the pectoral muscles. Sometimes the whole of the pectoralis minor was absent and part of the

1 At a meeting of the Section, held March 28, 1919.
pectoralis major as well. I do not remember whether there was associated defect or absence of mammary gland on the same side, or defect of nipple. In many cases, however, there was a considerable distortion of the spine and sternum owing to the absence of the physiological "pull" of the normal muscles.

Dr. Cockayne (in reply): Dr. Sutherland said, just before the meeting, that in these cases there was often more than a simple muscle defect, and that sometimes one found absence of part of the ribs on the same side: there might be defects of fingers on the affected side. So it looks as if in these patients there is some cause of a different nature from that found in the ordinary muscular anomalies due to atavism, such as are so commonly seen in the dissecting room.

Case of Imperfectly Descended Left Testicle Four and a Half Years after Operation.

By Philip Turner, F.R.C.S.

P. F., now aged 9 years, was admitted to Guy's Hospital in 1914 when 5 years of age, for left inguinal hernia and imperfectly descended testicle. The testicle was situated in the inguinal canal and had never descended through the external abdominal ring. It was freely movable in the canal, but could not be made, by manipulation, to pass beyond this. At the operation the hernial sac was excised and the left testicle was transplanted to the right side of the scrotum, through the septum, according to the method described before the Section for the Study of Disease in Children in November, 1914.¹ The left testicle now (March, 1919), hangs normally in the scrotum and is freely movable. There is no pain, and the testicle is not adherent to any surrounding structures, neither is there any thickening nor induration along the course of the spermatic cord. The transplanted testicle is, if anything, larger than the normally placed right organ. Owing to the age of the patient it is difficult to estimate the testicular sensation, but this appears to be the same on each side.

There is no recurrence of the hernia, and there is no sign that the testicle has been transplanted, though close inspection shows the scar on the scrotum.

Case of Double Imperfectly Descended Testicle operated upon in 1914, when the Left Testicle was transplanted to the Right Side and the Right Testicle to the Left Side of the Scrotum.

By Philip Turner, F.R.C.S.

D. E., aged 14 years, was admitted to Guy’s Hospital in 1914 when 9 years of age for double imperfectly descended testicle and inguinal hernia. The testicles could be felt in the inguinal canals, and neither had ever descended beyond this position. The left side was operated upon in July, 1914, when the hernial sac was excised and the left testicle transplanted through the septum to the right side of the scrotum. The patient was discharged from hospital in August, but was re-admitted in December, 1914, when a similar operation was performed on the right side, the right testicle being transplanted through the septum to the left side of the scrotum. It was noted at this time that the scrotum was small and ill-developed.

When examined, over four years later, both testicles were seen to hang normally and to be freely movable in the scrotum, which is now quite well formed. They are normal in size and contour, but the testicular sensation in the organ in the right side of the scrotum (true left testicle) is deficient. There is no induration and no adhesion to surrounding structures, and there has been no pain since the operation. There is no recurrence of the herniae, and the scars on the scrotum are scarcely visible.

Cases of Operation for Undescended Testicles.

By Philip Turner, F.R.C.S.

In November, 1914, I showed two cases of imperfectly descended testicle which had been operated upon a few months previously by a method which I described before the Society, after showing the cases.¹ The essential points of the operation are (a) that the testicle is transplanted to the opposite side of the scrotum through

the septum, and (b) that no sutures are required to hold it in position. The septum prevents retraction to the old position and provides a slight but long-continued force which draws the testicle downwards. The absence of sutures obviates injury to the testicle and diminishes the liability to atrophy. The results a few months afterwards were very satisfactory, but owing to the war I was unable to keep the cases under prolonged observation. With a view to ascertaining the late results I have recently endeavoured to follow up a series of twelve consecutive cases operated upon in the summer of 1914. Only three of the cases could be traced, and of these two are shown this afternoon. The result in the third, an adult, aged 39 years, was equally good. Neither of the two cases shown before the Section in 1914 could be traced. The result in the two cases shown to-day is very satisfactory. Unless one knew the history it would be difficult to realize that any operation had been performed at all. In both, the scrotum appears to be normal and to contain two normal testes. In the lad in whom the deformity was double the right testis was transplanted through the septum to the left side, and, after an interval of about five months, the operation was repeated and the left testis transplanted to the right side. It is generally agreed that the great majority of undescended testicles are functionless, at any rate as regards spermatogenesis. In both these cases, however, the testes, besides keeping in position, have increased in size, and this leads one to hope that, if the organs are transplanted before puberty and if this is accomplished without fixation by sutures, which must lacerate and injure these delicate tissues, normal development, both anatomical and physiological, may be possible in a certain proportion of cases.

DISCUSSION.

Dr. H. C. Cameron: Mr. Philip Turner's results in these cases are excellent, because we must all be agreed that the results of most operations for undescended testicle in the past have been, on the whole, very unsatisfactory. In my experience the diagnosis of undescended testicle is made too readily. I am not suggesting that this remark applies at all to Mr. Turner's cases; I am certain his have been true cases of undescended testicle. In a number of the cases I have seen, however, the diagnosis of undescended testicle has been made when there has been nothing of the sort present; there has only been an exaggeration of that which is common to all boys before puberty—an extremely active cremaster muscle, with the consequent tendency for the testicle to run upwards into the inguinal canal, with often, a small rugose scrotum. These testicles, on examination, perhaps with cold hands, disappear
and are hard to find. I know of cases which have been operated on for undescended testicle in which I am certain the real trouble was this undue mobility I have mentioned. A point of possible interest to the Section is the rather confident statement which has been made that thyroid extract is capable of causing descent of the testicle. I take it that the vast majority of those unduly mobile testicles become fixed in the development of the parts which takes place at puberty. I have seen very few cases of undescended testis without hernia: and I would say that, at any rate, hernia as an accompaniment is the rule, rather than the exception. I have seen no benefit, either to the undescended testicle or to the accompanying hernia from thyroid extract. The operations which I have previously seen done for fixing the testicle in the scrotum seemed in many instances to have been successful in the cases of undue mobility, but in the cases of true ectopia testis, very often the results were not good. It seems that Mr. Turner has really achieved a great success in devising this operation. Certainly the testicles of at least one of these boys are enlarging. I take it the elder of the two boys has reached puberty, and that his testicle is as large as it would be in a normal boy of the same age.

Mr. Mortimer Woolf: The cases of abnormal mobility of the testicle, mentioned by Dr. Cameron, are those in which the testicle can be brought into the scrotum by traction. But many cases exist in which it cannot be brought down, even to the top of the scrotum, and these cases, which are almost invariably associated with a hernia, are those of true undescended testicle. Mr. Turner's operation has produced a really wonderful result in the cases he has shown. I have performed Bevan's operation, dividing the structures of the cord with the exception of the vas, and turning the testicle upside down, so that the upper pole becomes the lower. The result one year later was very satisfactory. No sutures had been used for retaining the testicle and it remained at the bottom of the scrotum without any tension. But after seeing these cases, I think it probable that Mr. Turner's operation will become the classical one.

The President: As Dr. Cameron has said Mr. Turner has had excellent results from this form of operation, especially as it causes no damage to the testicle itself from suturing or otherwise. As there are no sutures, there is much less chance of subsequent inflammation and of any militation against the physiological testicular activity in subsequent years. Obviously this is a great advance on previous methods, and the ingenious plan of passing it through the septum of the scrotum, the elasticity of that septum keeping it there, is one which must excite our admiration.

Mr. Philip Turner (in reply): Dr. Cameron spoke of the difficulty which occasionally arises in diagnosis. I once had sent to a bed in my ward a case from the out-patient department which had been there diagnosed as double undescended testicle. When I came to examine the patient there was nothing abnormal, and the only thing I could imagine that had happened
was, that the cremaster had been acting with great vigour, and that led to the wrong diagnosis. Dr. Cameron also pointed out that it is almost invariable for a hernia to be associated with undescended testis. Yet in the last case upon which I operated—only two or three days ago—the testicle was in the inguinal canal, and I could not find any hernial sac. Only once before have I, in my recollection, operated upon an undescended testis without finding a sac. Even if there is no hernia, there is almost certain to be, in these cases, a patent processus vaginalis. I suppose it is a coincidence that in the three cases I have been able to trace the results have been extremely good, for I would not like to say that one is bound to get such results in every case. In fact, in a considerable proportion one finds some anatomical condition which renders a good result unlikely or impossible: the testicle may be found to be so very atrophied that it is not worth attempting to save it at all. So I cannot make any statement as to the proportion of successes or failures, though of these twelve cases three are quite satisfactory over four years afterwards, while two others, shown a few months after the operation, promised also to be successful.
Section for the Study of Disease in Children.

President—Dr. J. PORTER PARKINSON.

Case of Multiple Epulides.

By W. WARWICK JAMES, F.R.C.S., L.D.S.

(WITH REPORT ON SECTIONS BY DAVID NABARRO, M.D.)

W. B., a girl, aged 11 years, has suffered from the growth of a fibrous epulis (to be more definite, it is a hypertrophy of the fibrous tissue of the gingival muco-periosteum), associated with each tooth of the temporary and permanent series which has erupted. Removal of the growth alone is followed by recurrence, which does not take place when the teeth are also removed. Up to the present all the temporary and eight of the permanent teeth have been extracted.

The history of the case is as follows: The patient was admitted into the Hospital for Sick Children, Great Ormond Street, in May, 1912. Mr. Oswald Addison removed a portion of the tissue for microscopical examination. There was some uncertainty with regard to the nature of the tissue, but a further section showed it to be purely fibromatous. (See report by Dr. Nabarro, p. 69.) On examination the gingival margins were seen to be considerably enlarged and nodular. The tissue particularly involved was apparently the gum margin and the neighbouring portion, but not including the greater part of that covering the roots. As the result of treatment it would seem that the periodontal membrane was involved as well as the gum margin, but this can only have been in the particular part, as there was only slight displacement of the teeth laterally. It has also been found that the bone was not involved; X-ray photographs were taken, which showed no

1 At a meeting of the Section, held April 25, 1919.
marked changes in the bone. The growth had extended round the
crowns of the teeth, in parts even reaching to the top of the teeth. In
the early stages it appeared red as if distinctly vascular, and had
a somewhat denuded appearance, but later it appeared to be less
vascular, and was mottled, with faintly yellowish-brown patches.
There was no tendency in the tissue to break down except where
injured by opposing teeth. The rate of growth was slow, the increase
being marked by months rather than weeks. The patient suffered no
pain, although her general health seemed to be affected, probably on
account of the difficulty she experienced in eating. The growth bears
a superficial resemblance to a simple fibrous epulis, but is sessile, while
the simple epulis is nearly always pedunculated. The feature of par-
ticular interest in the case is the effect of treatment, and this coincides
with the records of other cases, and of that of a simple fibrous
epulis, it being necessary to remove the teeth involved in order to
avoid recurrence.

Seven separate operations have been performed, and an attempt
has been made to remove the growth without removing the teeth,
but as recurrence followed in due course all the temporary and
eight of the permanent teeth have been extracted, the growth
completely excised, and the margins of the alveolus removed. The
stages of the operations were as follows: All the temporary incisors
and the first temporary molars were extracted and the growth
removed, whilst the abnormal tissue was cut away freely down to the
bone margin round the remaining eight teeth. Where the teeth
remained there was a recurrence of the growth, and in July, 1912, the
four molars were removed as being less accessible than the canines, and
the tissue again pared away round the latter. In October, 1912, with
further recurrence round the canine teeth, they were similarly treated.
The child was now nearly 5 years old. No change took place in the
mouth for about two years, nor was there any sign of a similar growth
until after the first permanent molars appeared, when the condition
recurred in about a year, and it became necessary to repeat the previous
operation. In October, 1917, the maxillary incisors had erupted with
the recurrence of the condition, and although there was some sug-
gestion of it upon the lower incisors, it was not marked. An operation
removing the tissue without the teeth was unsuccessful in checking
the growth, and the teeth were removed in November.

The present condition shows the lower canines involved, also the
left lower second premolar, which is erupting into the socket of the
first permanent molar. The same change is taking place with regard to the right upper premolar, which is erupting into the socket of the first permanent molar. The gum presents the reddish, somewhat denuded appearance already described, which seems to be the first indication of the presence of the growth, although in parts it is reaching the nodular stage. The patient has been wearing a vulcanite block to provide her with something to bite upon, and it is interesting to note that the pressure from this seems to have arrested the development of the condition in one part. The child was strong and healthy from birth, with the exception of whooping-cough, and an attack of what
the mother describes as "dry eczema," which occurred before the appearance of the mouth condition at the age of 3 years. The patient did not suffer from rickets. She was not nursed for more than a few weeks.

She is not a mouth-breather—a fact of particular interest, as considerable hypertrophy of the gingival tissues is frequently seen in the front part of the mouth in such patients. Such a condition is of an infective nature, and the changes are in marked contrast to those that are seen in the case now shown. In mouth-breathers the swelling of the tissues conforms accurately to the line of contact with the lips, so that when the lips are pulled back there is no hesitation in saying that mouth-breathing exists. In cases of hypertrophy the lips may be apart on account of the bulk of the growth, but it is an effect, not a cause. The condition obtaining in this patient would probably be described as hypertrophy, and although a certain number of cases have been recorded, they are undoubtedly rare. The hypertrophy seen in adults is due generally to chronic infection, and involves the bone as well as the gingival tissue. Perhaps the commonest type is a marked thickening of the gum in the region of the maxillary molars, although several cases have been recorded in which the whole of the alveolar margin has been involved, but the maxilla appears to be affected more often than the mandible in children. Christopher Heath described a similar case to the one now reported. Cases mentioned by him occurred at the ages of 8, 2½, 7, 4 and 2 years, the three latter occurring in one family. In all the cases recorded there was defective mental condition. In another case recorded which was very similar to the one now shown, the child was 4½ years old, and healthy. After describing this case, he says: "In conclusion I should say that nothing less than complete removal of the affected alveolus seems to offer any hope of alleviating these cases." As far as I can ascertain, the length of history of this particular case is greater than that of any recorded cases.

As it is difficult to classify these cases definitely, the term epulides is used in its widest sense, but if they were allotted to a definite class, it is probable that hypertrophy would be the correct description.

I am indebted to you for permitting me to bring the case before the Section. I do so in the hope that I may have some suggestions as to future treatment. It seems a very drastic procedure to remove every tooth, and so render the child edentulous. It is true we can provide an artificial substitute, and that will have to be done if nothing short of extraction can be accomplished.
ANATOMICAL AND HISTOLOGICAL REPORT ON THE SECTIONS BY DR. NABARRO.

From the anatomical and histological points of view the case is not so interesting as from the clinical. The description of the growth will perhaps be best given in chronological order of its removal.

(1) The first pieces were removed by Mr. Oswald Addison on May 9, 1912. Microscopically the tissue consisted mainly of spindle-shaped cells with some multi-nucleated cells which suggested the diagnosis of a mixed-celled sarcoma, probably a myeloid sarcoma.

(2) On May 22, 1912, several more pieces were removed. Macroscopically these consisted of a tough fibrous material with a smooth surface. On section they were tough and whitish in colour, like a fibrous epulis. Microscopically the sections looked more like a fibroma than those previously examined. No multinucleated cells were found. There was a considerable downgrowth of the epithelium with the formation of apparent cell-nests. These and the previous sections were examined by Professor Shattock, who diagnosed them as fibrous epulis, and said that the case recalled that of the late Mr. Christopher Heath, of very extensive epulis in both jaws. The tissue was examined for micro-organisms both in section and by culture. Short-chained streptococci were found in the superficial part of the tumour and these were also obtained in cultures. Doubtless these were due to a secondary infection from the mouth and could not be regarded as being etiologically connected with the growth.

(3) On July 10, 1912, several further small pieces were removed. These again were white, tough and fibrous. Minute fragments of bone were attached to them. Microscopically the sections showed a fibromatous structure as before, and very few multinucleated cells were present. There was calcification and bone formation at one part of the section, and cells were present which may have been osteoblasts. At one spot, where the epithelium was just growing over it, there was distinct infiltration with small round cells.

(4) In October, 1912, more tissue was removed. The section showed fibrillated connective tissue covered by stratified squamous epithelium. Here and there, there were small haeorrhages into the tissue. One section showed some striated muscle fibres. There were no multi-nucleated cells present. The tissue looked altogether more like the normal than did that removed on previous occasions.

(5) On February 8, 1917, some growth in the region of the left
upper molar tooth was removed by simple excision by Mr. Ovey. Microscopically this was typical of fibrous epulis with hypertrophy and downgrowth of the stratified epithelium. No multi-nucleated cells were seen. There was a little small round-celled infiltration at one spot.

(6) On April 3, 1918, some further tissue was removed. As before, this was tough, fibrous, smooth on the surface and whitish on section. Microscopically, the tissue was characteristic of fibrous epulis; no giant cells were seen. There was proliferation of the epithelium with a few cell nests. In one place some calcified cartilage (or ? immature bone) was present.

(7) Finally, in October, 1918, the tissue examined showed fibrous tissue covered by proliferating epithelium, and, in one place, some calcifying cartilage.

On April 16, 1919, the blood was tested for the Wassermann reaction with a negative result.

DISCUSSION.

The Chairman (Dr. Langmead): The case is of very considerable interest. I take it that if we call it fibroid epulis we are not very much further on, except that we can put it with other cases of the same kind. Where does it come in the nomenclature of tumours? We shall be glad to hear suggestions as to treatment, aetiology, and precise nomenclature.

Dr. Nabarro: I suppose it comes under the heading of fibroma, but obviously it is not a pure simple fibroma, because of the multi-nucleated cells which are present and the absence of a tendency to recur. If fibroma is completely removable, there is no recurrence. But in cases where it is attached to the periosteum and the alveolar border it is very difficult to get away the whole thing, and, short of complete eradication, it is certain to grow again, just as a wart will do if not completely removed. I might, perhaps, say I regard this as benignly malignant in the sense that it will continue to recur unless complete eradication is possible. Mr. James asks what should be done to prevent it recurring? I think the only thing seems to be to make a pretty free incision and excision of the alveolar border, and to sacrifice the teeth. Mr. James has tried hard to get rid of the condition short of that drastic operation.

Dr. F. Parkes Weber: This seems to be intimately allied to the cases which have been described as diffuse hypertrophy of gums: for instance, the family cases recorded by Heath, as already referred to. When some of these rare cases have been under discussion it has been suggested that the diffuse hypertrophy of the gums was secondary to septic conditions connected with
the teeth and dental alveoli. I failed to see any proof whatever that the remarkable condition of diffuse hypertrophy of the gums has ever been secondary to septic conditions. In Heath's cases various members of the same family were affected, and that fact tells very much against the causation having been chronic septic irritation. A septic condition of the gums must of course necessarily become superadded as a secondary complication. Moreover, there is a really septic condition, which might be called septic hypertrophic gingivitis. I suppose three forms of the primarily non-septic condition are met with: first, the single epulis; then the multiple form of epulis: and, thirdly, a diffuse form, which may be called "diffuse hypertrophy of gums." In addition there are malignant forms of epulis, in which one may suppose that the epulis has undergone malignant transformation, or was from its commencement potentially malignant. I would like to leave those malignant cases out of this discussion. The treatment suggested seems most reasonable. The whole subject was discussed on the last occasion that Mr. Stanley Boyd showed a case.

Mr. E. D. D. Davis: I have had three cases which were sent on from the Royal Dental Hospital, and I regarded them as localized hypertrophies of the gum. I take it that Dr. Nabarro does not negative hypertrophy. In my three cases I could find no cause. One was carefully investigated by Mr. Hopewell Smith, who went down to Aldershot and looked up the family history in regard to such a tendency. In those three cases I removed the gum very freely, with some of the underlying bone. There was no recurrence. But in one other case I saw, upon which the late Mr. Stanley Boyd operated, there was a more diffuse hypertrophy of the gum, extending along the whole of the upper alveolus, and that recurred to a slight extent. I do not regard these cases as malignant in any way, but I think that free removal gives the best result. There is some scarring and the sacrifice of certain teeth, but not more. I would like to know from Dr. Nabarro whether he can definitely say this is not a hypertrophy: I think he is hesitant on that point. There is a large amount of fibrous tissue in the gum, and I take it the fibrous tissue would be hypertrophied to a greater extent than the epithelium.

Dr. Nabarro: In reply to Mr. Davis, I do not know whether one can tell the difference: the pieces removed looked like pure fibroma covered with thickened epithelium, except that multinucleated cells were present. This fibrous tissue seemed to be very well developed.

Mr. E. D. D. Davis: That is the difficulty we had: the pathologist would not say definitely whether it was a hypertrophy, or a fibroma. The late Mr. Stanley Boyd was interested in all the sections cut, and considered them to be a hypertrophy and benign.

Mr. Warwick James (in reply): There are cases which, I think, certainly are infective in origin, those which Dr. Parkes Weber described as hypertrophic gingivitis. They are more than gingival, and nearly always affect the
bone. If the infective process is active enough, there is rarefaction. If it is chronic, the condition becomes sclerotic, and it can go on to such an extent that the alveolus is almost double the normal size, while there is hypertrophy of gingival tissue as well. I think "hypertrophy of the gum" is an excellent term for the present case; the condition is limited to the gums, and must be of the nature of hypertrophy. I do not think we need haggle over whether it is a hypertrophy or fibromatous. If it were localized and had a capsule, we should call it fibroma, but in this case there is a mass of fibrous tissue, but without a capsule. With regard to Christopher Heath's cases, there is one family of three children who had it, but there is also the case which is illustrated here in this book, very similar to the one I am now describing, and in a child of about the same age. Localized epulis is more easily dealt with than in this diffuse form.

Dr. Nabarro (in reply): While I agree with Dr. Parkes Weber, as does Mr. James, that there may be cases of hypertrophic gingivitis of infective origin, there is no evidence that the present is such a case. We may call it simple "hypertrophy of the gums" as Mr. Davis suggests, or the name "diffuse fibromatous hypertrophy of the gums" might be given to the condition. In either case we have no clue as to its causation. Under what stimulus do the gums hypertrophy? And why should the condition recur after the lapse of some years—with the eruption of the permanent teeth? I believe myself that when the aetiology of neoplasms has been discovered we shall find that a case such as the present will have a similar aetiology, whether we label it hypertrophy of the gums or fibrous epulis.

Aplastic Anæmia.

By J. Porter Parkinson, M.D.

I am showing this boy to illustrate the complete recovery from a grave form of aplastic anæmia. You may remember that last year shortly after being shown at the meeting of this Section in May, he was nearly moribund, his red corpuscles having gone down to 580,000 per cubic millimetre, and the leucocytes 2,600, 76 per cent. of which were lymphocytes. In November, last year, when he had recovered, a blood count showed: Red blood corpuscles, 4,250,000; white blood corpuscles, 5,600; haemoglobin, 68 per cent.; colour index, 0.8. The white blood corpuscles were: Polymorphs, 55 per cent.; lymphocytes, 43 per cent.; hyalines, 2; eosinophils, 0. By the middle of January there was a further improvement: red blood corpuscles, 4,420,000; white blood corpuscles, 5,400; haemoglobin, 75 per cent.; colour index, 0.8; polymorphs, 67 per cent.; lymphocytes, 32 per cent.; eosinophils,
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1 per cent. You see to-day that the boy is as sturdy and strong as could be desired.

The specimens shown come from a fatal case of the same disease which was under my care in hospital from March 21 to 25 this year. He was a boy, aged 12 years. He had had measles and whooping-cough seven years ago, and scarlet fever two years ago, and since then he was subject to attacks of nose-bleeding. He never had had any swelling of the face or limbs.

There are no bleeders in the family and no history of consumption. He had two brothers in good health and two sisters who had kidney-trouble after scarlet fever. He was languid since Christmas last, but attended school till February 23, when he fainted; since then he was in bed, and seemed to be getting paler each day.

On admission to hospital he was seen to be very anæmic, the skin and mucous membranes blanched. There was much factor oris. and the gums were spongy and bleeding; a small purpuric spot was seen on left thigh. There were a few rhonchi in the lungs, and a systolic pulmonary murmur over the base of the heart which was not appreciably dilated. The abdomen was normal, the liver and spleen normal in size. The urine contained neither albumin nor sugar. The temperature varied in hospital between 100° F. and 99° F. Blood-pressure was 85, and the pulse about 120, small but regular. A few more purpuric spots appeared on the trunk. The blood contained: Red blood corpuscles, 434,000; leucocytes, 2,700; polymorphs, 33 per cent.; lymphocytes, 67 per cent.; no poikilocytes, nor nucleated red cells, but a few megalocytes; hæmoglobin content was about 10 per cent.

When I saw the boy the case appeared hopeless, but I ordered transfusion of blood if possible. Before this was done however the boy died from syncope on March 25.

The necropsy was done within twelve hours of death, and owing to a mistake I was not informed till after it had taken place, and therefore I was unable to suggest any special examination. The notes record considerable oedema of the right lung, the left being normal. The heart muscle was flaccid and anæmic-looking, its whole surface was thickly spread with large petechiae, and similar petechiae were found under the mucous membrane of the stomach and colon but not in the small intestine. The mesenteric glands were enlarged and dark purple from hæmorrhages, except those draining the small intestine which appeared normal. The other abdominal organs such as the liver, spleen and kidneys, were normal to the naked eye.
I much regret the imperfections of the recorded morbid anatomy of this case, and I can draw no conclusions from the scanty details; but one fact from the clinical history is significant, that both patients had suffered from scarlet fever, one eighteen months previously and the other two years before; in one case this was said to have been followed by kidney disease and dropsy, and the other boy was said to have not been well since, so there may be some aetiological connexion between this acute specific and the following aplastic anaemia, though in neither case was there any evidence of kidney disease, and no albuminuria.

Through the courtesy of Dr. E. Pritchard I was able to see a case of aplastic anaemia under his care and was interested to find that his patient also had had scarlet fever eighteen months previously. In no case was there any albuminuria or evidence of kidney disease. It seems to me extremely probable that the poison of scarlet fever inhibited the function of the red bone marrow and so produced the disease. It may be this form of anaemia is not so uncommon as has been thought and that perhaps a more careful blood count would reveal further cases. Its essential feature besides the great loss of red corpuscles is that the polymorphs are very much reduced in number, while the lymphocytes, though being about 60 to 70 per cent. of the total white corpuscles, are really normal in number. There seem to be no symptoms special to the disease; all those present in my cases are seen in any case of severe anaemia.

DISCUSSION.

Dr. F. Parkes Weber: It is a pity that in Dr. Parkinson's fatal case the bone marrow, especially that in the long bones, was not examined in order to demonstrate the actual presence of an aplastic condition of the red blood-forming tissue in the body. That would have absolutely settled the diagnosis, but there can be practically no doubt that the case was an example of sub-acute aplastic anaemia in a child. In a fatal case in an adult, which I published some time ago before another Section of the Society,¹ the bone marrow was examined after death, and was found almost completely devoid of red coloration—i.e., there was hardly any erythrocyte-forming tissue in it. In ordinary fatal cases of pernicious anaemia the presence of an erythrocytopoietic reaction is evidenced after death by much of the white bone marrow being found transformed into red marrow. The question arises as to what is the cause of the aplastic anaemia in these cases. Is there a congenital deficiency

in the power of reaction of the bone marrow as a blood-forming organ? In certain cases aplastic anæmia may have been set up by stomatitis or some infectious disease. Does the disease, whatever it is, poison the bone marrow in such a way as to inhibit its function of producing erythrocytes and polymorphonuclear leucocytes? I am inclined to think, on the whole, that the data we possess point to there being some connexion between aplastic anæmia and the toxic effect of a microbic disease, without there being, necessarily, any congenital predisposing factor present. This toxic theory of the disease is supported by the fact that in certain poisonings, such as tri-nitrotoluene (T.N.T.) poisoning, the function of the bone marrow as a blood-forming organ may occasionally be severely damaged, with the result that a condition of aplastic anæmia is induced.

Dr. Cockayne: How does Dr. Pritchard propose to treat this child? Does he intend to carry out transfusion?

The Chairman: In considering the advisability of transfusion in these cases there arises the whole question whether transfusion acts merely by providing blood or whether it stimulates the red marrow. If the red marrow cannot react it is not much good to transfuse.

Dr. Parkes Weber: If Dr. Pritchard decides on carrying out transfusion of blood, will he employ any special safeguards against the occasional terrible effects? I am afraid it is too certain that transfusion of blood in some "medical" cases may be followed by grave symptoms, and even by death. Dr. Parkinson, before the first transfusion, tested the blood of the donor for haemolysis against the blood of the recipient. Just about that time (1918) I was consulted in the country about a case of aplastic anæmia in an adult, and it seemed as if death was hastened by intramuscular injections of a healthy person’s blood.

Case of Unusual Tumour of the Abdomen.

By J. E. Adams, F.R.C.S.

H. P., a boy, aged 6 years, the last of three children; has been a delicate child since birth; has been taken to several doctors but his abdomen was examined only once and that was two years ago. The child was brought to the Shadwell Hospital with the complaint of looseness of the bowels, poor appetite, and occasional severe abdominal pain, dating from last December. The boy was recommended for admission as suffering from (?) renal tumour or tuberculous glands.

On admission, March 31: He was found to be fairly well-nourished, but small for his age (1 st. 10 lb.). The abdomen was soft, and there
was an elongated tumour in the right mammary line extending to the level of the umbilicus and about 3 in. wide; this tumour moved with respiration, was smooth, firm, not tender, and was fairly mobile in all directions: the lower, inner and outer borders were fairly well defined, but it was not possible to define the upper limit. It was possible to pass one's hand under the tumour. The lower border of the liver was not palpable: no note has been made as to its upper limit. The spleen was in its normal position and was just palpable. There was nothing else abnormal in the abdomen or elsewhere.

The radiogram was of no help in arriving at any diagnosis. The urine was absolutely normal and there was no trouble with micturition. There was no frequency of stools while the patient was under observation, nor was there any complaint of pain.

Laparotomy, April 2: The liver was found to be all to the right of the middle line. There was no left lobe. The stomach was above the level of the liver, and the inferior surface of the liver was situated so that the gall-bladder lay on the top of the liver, and the foramen of Winslow was entered by passing over the upper surface of liver and to left.

Discharged April 18.

I selected this case to bring forward this afternoon as I was under the impression that there were to be a number of cases of abdominal tumour and disease shown. It is an interesting example of an anatomical abnormality rather than a pathological tumour formation in the abdomen. I am indebted to Mr. Gunewardine, resident medical officer, East London Hospital, Shadwell, for notes of the case. The child is exceptionally small for the age of 6 years, is the last in a family of three, and has been delicate since birth, though there has been no positive illness until recently, when he was brought to Shadwell with the complaint of looseness of bowels and poor appetite, with somewhat severe abdominal pain, which dated from last December. The symptoms were quite vague, but the co-existence of those symptoms and the unmistakable abdominal tumour made one feel that it was justifiable and desirable to explore the abdomen. The clinical diagnosis lay between a Riedel's lobe of the liver or a tumour connected with the right kidney, though the absence of urinary symptoms gave no support to this second diagnosis.

On opening the abdomen I found that the tumour represented the whole of the child's liver. There was no left lobe and all the hepatic tissue was to the right of the falciform ligament, which was present,
though of small size. The liver extended far below the level of the umbilicus and a careful examination revealed the gall-bladder attached to the superior instead of the inferior surface, and in order to enter the foramen of Winslow one's finger had to be passed over the upper surface of the organ, and the stomach lay on a higher plane than the liver which had the duodenum to the left of it. I did not palpate the pancreas. The rest of the abdominal organs, including the kidneys, were in their normal positions, the right kidney being entirely behind the liver. The condition can best be explained by assuming that the inferior surface of the liver, with the important structures normally attached to it, had been rotated around an antero-posterior axis and the left lobe obliterated in this process and incorporated in the liver tissue to the right of the falciform ligament.

It was clear that no surgical treatment was required, and the case is merely an example of anatomical abnormality; more interesting, perhaps, to the anatomist than to the surgeon or the pathologist. Up to the present time he has escaped the attentions of the anatomist, and I hope he may do so for many years to come.
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President—Dr. Porter Parkinson.

Lymphangioma of the Tongue.¹

By B. Whitchurch Howell, F.R.C.S.

The patient is a little boy aged 6 years, the only child. The birth was a natural one. There is no family or personal history of disease. He gives a negative Wassermann reaction. He has a congenital nævus of the lower lip, which also involves the mucous membrane of the gum. This gets fuller when the child cries, and bleeds occasionally. The swelling of the tongue was first noticed when the child was 3 years of age. It commenced on the posterior aspect and has grown slowly forwards. It is now stationary. Vesicles continually break down and ulcerate, causing pain, and on this account the boy bolts his food, which is always minced. He eats most things, and takes a rather large amount of salt, of which he is fond, but avoids sauces. Occasionally the tongue enlarges, without apparent cause, and protrudes from the mouth. It is then excessively painful and is a source of anxiety to the patient and his parents. Salivation is excessive, and articulation impaired. The tonsils are much hypertrophied, the submaxillary lymphatic glands are much enlarged, and the submental glands are palpable.

This condition is extremely like those described by the late Sir Henry Butlin in his book on diseases of the tongue, and in St. Bartholomew's Hospital Museum there are several drawings and paintings showing this condition. Apparently Sir Henry Butlin used to remove a V-shaped portion of the tongue in these cases, but as far as I can see, surgery would not be of use in this patient, especially as Sir Henry Butlin said these cases are progressive.

¹ At a meeting of the Section, held May 23, 1919.
DISCUSSION.

Dr. H. C. Cameron: Has Mr. Howell shown the case to anyone who has had experience of the effects of radium in such a condition? We know how remarkable is its effect on superficial naevi, and I should think the naevus of the lip in this child could be got rid of in that way. Much depends on the depth of the lymphatic enlargement of the tongue. Is it possible to produce contraction of the superficial parts which must determine the infection in the deeper parts?

The President: This does not seem to be a suitable case for excision of a portion of the tongue, as it is so extensive a condition. No object would be gained by an operation of that kind. I do not think we have present anyone who is an expert on radium.

Mr. Howell (in reply): I have not shown the case to anybody who might suggest treatment in the way mentioned, but I will certainly do so.

Postscript.—The child is now under treatment at the Radium Institute by courtesy of Dr. Pinch.

Case for Diagnosis.

By E. A. Cockayne, M.D.

G. S., aged 11 years. The boy had influenza in December, 1918. He has never been well enough to go to school since and has been languid and unwilling to play out of doors. A month ago he became very drowsy. His appetite is good and he has had no vomiting. There is no family history of tuberculosis. The boy is wasted and looks delicate. There is slight jaundice, which has remained unchanged during the fortnight he has been under observation. The abdomen is prominent, especially in the upper part, and difficult to palpate. Both the right and left lobes of the liver are greatly enlarged, the edge of the right lobe can be felt some distance below the level of the umbilicus. The surface is smooth and the organ feels firm but not extremely hard. The spleen is palpable, and the splenic dullness is increased. There is no ascites; no tenderness; no abdominal pain. No enlarged glands can be felt anywhere. There are no enlarged veins over the abdomen and no spider naevi anywhere. There have been no haemorrhages. The stools are normal in colour and consistency. Urine, dark in colour, contains a trace of bile, but no excess of urobilin. There is a trace of
albumin. Red blood cells, 6,100,000 per cubic millimetre: leucocytes, 6,400 per cubic millimetre; polymorphonuclear, 54 per cent.; mononuclear, 46 per cent.; Wassermann reaction, negative; evening pyrexia, 100° to 101° F. on most days.

I have brought the case for diagnosis. I think it is one of abdominal tuberculosis. The question of cirrhosis of the liver arises, but the facies of the boy is not characteristic: the liver is smooth, and does not feel as hard as is usual in that condition. The question of haemolytic jaundice occurred to me, but there is definite bile in the urine and only a trace of urobilin. The spleen is not as large as and the liver is larger than one would expect, and there is no anaemia, yet the child is ill. I do not think there would be this degree of illness in an exacerbation of haemolytic jaundice unless there was marked anaemia, and I do not think it is that condition.

DISCUSSION.

Sir Humphry Rolleston: I agree that the underlying process is probably tuberculosis affecting the liver more than the spleen and so different from the condition of primary massive tuberculosis of the spleen with secondary but less extensive infection of the liver. Possibly it was a case of tuberculous abscesses in the portal spaces involving the bile ducts, a condition which is rather inexplicably very rarely associated with jaundice, for the bile ducts are obviously obstructed. There are, however, two other possibilities: post influenza hepatitis occasionally caused hepatic enlargement sufficiently persistent to suggest cirrhosis or even early malignant disease. Amyloid disease is compatible with the hepatic and splenic enlargement and with the albuminuria; but there is no evidence of a cause for amyloid change and the presence of jaundice is rather against this diagnosis.

Dr. F. Parkes Weber: Dr. Cockayne has admirably summed up the points in favour of tuberculosis, and I think with Sir Humphry Rolleston, that that is the most likely explanation of the case. But I would suggest a further alternative—namely, that it may be an early case of acute lympho-granulomatosis maligna—that is to say, acute Hodgkin's disease—in which the retropertitoneal lymphatic glands and the liver are involved at the same time. The liver may be pushed and tilted forward by the enlargement of those glands, so that it may appear to be larger than it really is. The same disease would also account for the enlargement of the spleen.

Dr. H. C. Cameron: I hope that, if we have another meeting of the Section this session, Dr. Cockayne will be able to give us a further history. The diagnoses fall into two groups, of different import for the patient. This boy did not strike me as being very ill, and it may be we shall hear in a month's time that, even though the condition has lasted so long, it is diminishing.
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Sir Humphry Rolleston: I would like to ask Dr. Weber a question on his remarks. Does he not think, if it is lymphadenoma with so much liver involvement, there should be relapsing fever?

Dr. Parkes Weber (answering Sir Humphry Rolleston): No, I do not think it at all necessary that there should be the so-called "Pel-Ebstein" type of intermittent pyrexia. That type of fever is present only in relatively few cases of Hodgkin's disease.

Dr. Eric Pritchard: Dr. Weber suggests that the enlargement of the liver here may appear to be greater than it really is, owing to it being pushed forward: will that explain the great enlargement of the liver upward? It almost reaches the level of the nipple, besides showing downward enlargement. The one feature of this case, about which there is, in my opinion, no doubt, is that the liver is very greatly enlarged.

Dr. Parkes Weber (replying to Dr. Pritchard): The retroperitoneal glands, being enlarged, might push the liver up and rotate or tilt it forwards. One might have the region of the diaphragm examined thoroughly by means of the X-rays (with the fluorescent screen, to see the movements). In an early case I think there may be enlargement of the retroperitoneal glands without any obvious enlargement of superficial lymphatic glands. There need not be œdema of the legs in an early stage of the disease.

Dr. Cockayne (in reply): I was interested in Sir Humphry Rolleston's suggestion that this might be a genuine post-influenzal condition, because I did not come across any such cases in the epidemic. In it I saw very few cases with jaundice, and none of the cases showed persistent enlargement of the liver. With regard to the suggestion that it is lymphadenoma, I know that enlargement of the superficial glands is not found in all the cases: but it is against that diagnosis if you find no enlargement with such advanced visceral involvement; and if the retroperitoneal glands are so greatly enlarged, I think there would be œdema of the legs when he walked about. He has never had that, and has none now, of course, while he is in bed. I will have him examined with the fluorescent screen, and, if necessary, have a plate taken, as suggested. I think he is ill, but not acutely so. He was very tired and disinclined to move about when he first came to the hospital, and he seems content to be in bed. He is no worse than he was a fortnight ago, in fact he has gained somewhat in weight and in general condition. I will report further when something definite is known in addition.
The patient is a girl, aged 3½ years. There is a history of tuberculosis in the family. Symptoms of abdominal pain commenced six months ago, and have continued on and off ever since. The pain usually comes on immediately after food, and is sometimes coloured with green bile. There is much wasting, sweating at night, and occasionally blood in the stools. The belly is doughy; on palpation a tumour can be felt extending across the abdomen just above the level of the navel, and below in the position of the left kidney there is a smooth rounded tumour, dull to percussion and elastic to the touch.

I think this is clearly a case of abdominal tuberculosis. I brought the case because of the presence of this tumour in the position of the left kidney. It is so smooth in contour that when I first palpated it I thought it was hydronephrosis, or a cystic tumour of some kind in the kidney region. It does not seem to accord with what I have previously met with, cases of abdominal tuberculosis with large deposits in the omentum. I brought the case in order to elicit the opinions of members as to whether the swelling is simply a loop of intestine entangled in the omentum which gives an impression of being cystic, or is really a tumour. The two skiagrams I pass round show very large deposits, not only in that region, but also on the right side, which are so opaque that they may be calcified glands.

[Since showing this case the tumour has become resonant, confirming the view that it is an incarcerated loop of intestine.—E. P.]

The President: I hear that the swelling on the left side is dull on percussion, but I think it consists of matted intestine, with, possibly, some focus of tuberculous material enclosed in meshes of intestine. Most of these tumours are resonant, and this feels more like omentum than matted intestine. The other string one can feel is like matted omentum.

Case of Joint Trouble.

By Miss C. A. King, M.D.

This child is a boy, aged 7 years. I first saw the boy in June, 1918, when he had an attack of erythema nodosum, which quickly cleared up. He came back in the following December with a history of pain and
swelling in the right knee of three weeks’ duration. He also complained of pains in the hip and ankle-joints. There was considerable effusion into the right knee, but the other joints appeared normal. The heart was unaffected, and no other signs of disease were detected. He ran a moderate temperature for a week, but the condition cleared up under salicylates.

A week or two after discharge from hospital there was return of trouble in the knee-joint. X-ray examination showed effusion, but no other sign of disease. He had a Wassermann test done, and the reaction was strongly positive. He has been on antisyphilitic treatment since. The fluid has subsided, but the condition of the knee has not cleared up so quickly as I thought it would. The second attack has been practically a pyrexial. The father, who was killed in France, was healthy, the mother has had rheumatic fever once, and there are four full-term children. There is no history of miscarriage. We have not found anything amiss with the boy beyond what I have stated. I have not a report of his fundi.

**Case of Syphilitic Bone Disease.**

**By J. Porter Parkinson, M.D.**

The patient, a girl aged 12 years, with a healthy family and personal history, giving no suggestion of syphilis, two months ago had pain and swelling in the right under eyelid; a week later she had pain and swelling in right ankle, followed by similar symptoms in the left elbow. The pain became less, but the swelling persisted, and she was brought to hospital. She is a well nourished, healthy looking girl, with no signs of syphilis. The incisor teeth are normally shaped. The leg above the right ankle is swollen, this being due evidently to enlargement of the lower end of the tibia; the movements of the ankle are normal and painless, and there is no fluid in the joint. The left elbow is swollen, and the swelling extends down the forearm for 2 or 3 in. It feels like bony thickening of the ulna, but fluid can be felt distending the capsule of the joints posteriorly. There is no pain on movement, which is quite free. The heart, lungs, and abdomen are healthy. The Wassermann reaction was negative on the first examination.

An X-ray photograph of the joints shows the bony changes in the tibia and ulna, and evidence of periosteal thickening in the tibia. I believe the disease to be syphilitic in origin, and shall have another blood examination made.
DISCUSSION.

Dr. Cockayne: With regard to the first of these cases, three weeks ago I saw a boy with both knees affected very much in the way this case is. The knees had been very painful at first. There were no signs of syphilis, just as in this case, and I did not think it was syphilis. But the Wassermann reaction was strongly positive, and mercury inunction and the administration of mercury and iodide of potassium internally have effected an enormous improvement, so that he can now straighten his legs and walk, which he could not do previously.

Mr. B. Whitchurch Howell: I saw the patient referred to by Dr. Cockayne—a case of double synovitis of the knee. He then had slight flexion, and peri-articular thickening. I think Miss King's case is identical, but it has progressed further. There is some hypertrophic change in the bone. I would suggest local mercurial treatment in Miss King's case after first applying something to completely extend the knee-joint. I do not think it will be safe for that child to walk about on it for six to twelve months, because of the hypertrophic change round the epiphyseal line. A Thomas's calliper-splint should be put on. A further sketch should be taken in six months time, anti-syphilitic treatment being persisted in during the interval. The other case I regard as very similar, also syphilitic. I think Dr. Parkinson's patient will need no supports.

Miss King: In the case of a family I saw a year ago, a boy came with symmetrical synovitis of the knees in his sixth year. We found that two years before, a brother of his had been to the hospital with symmetrical synovitis. Both gave a strongly positive Wassermann reaction, and in both the condition cleared up under antisyphilitic treatment. We found no other sign or symptom of the disease about them. I got the mother to bring up another child, two years younger, for examination. No signs of disease were found, but he also gave a strongly positive Wassermann reaction. The mother refused a course of preventive treatment, and I am wondering whether that child also will come back two years later with synovitis.

Dr. H. C. Cameron: Is not the symmetrical character of a tertiary syphilitic synovitis very constant? Do we see a one-sided painless effusion into the knee-joint as a sign of tertiary syphilis in a child at about the same age as we find deafness and keratitis due to the disease? I have seen a considerable number of these cases of synovitis, without disease of bone, in congenital syphilis, but the synovitis has always been symmetrical, always painless, and has always yielded readily to mercurial treatment, and has never been accompanied by anything like the muscular wasting, the fixation, or the pain, as is the case here. I should have thought this boy had a tuberculous knee, although there is the positive Wassermann reaction. The other case lacks nothing of the character of a syphilitic affection of bone, with involvement of the joints secondarily, except that it gives a negative Wassermann reaction.
The thickening of periosteum, indicating long-continued changes in the bones, is very conclusive of syphilis. I think we have all been in the same boat before: we have come across, here and there, a case in which the Wassermann test does not coincide with the clinical conclusion, nor with the post-mortem finding. The Wassermann test should be done again, more than once, in order to see whether the discrepancy is not accidental.

Dr. F. Parkes Weber: With regard to the boy (the first case) with the swollen knee, I do not see anything against the diagnosis of unilateral tertiary syphilitic synovitis. He has very little pain or pyrexia now, though there is much swelling of the knee-joint, and periosteal conditions in congenital tertiary syphilis are not rarely unilateral. Bilateral cases are commoner. I see, almost daily, a young lad who has signs of congenital syphilis, with bilateral chronic synovitis of the knees. Yet he feels quite well, and maintains that he suffers no pain nor stiffness in the joints. The other (second) case, apart from the negative Wassermann reaction, seems to be an example of chronic syphilitic osteoperiostitis of the right lower extremity in a subject of congenital syphilis. The Wassermann reaction might be taken again in both these patients, and if possible it should be taken in their mothers also.

Dr. G. de Bec Turtle: These cases are similar to, and it is difficult to distinguish them from, Still's disease, or multiple arthritis. I remember a case, some years ago, in which a child's joint was definitely diagnosed by a well-known physician, also by a surgeon, as tubercular, and it was put up and treated accordingly. In a few months' time other joints became involved, and the case was recognized as one of multiple arthritis, of which we know little, except that there are rheumatoid changes in the bones, and that it is very intractable.

Dr. F. Langmead: In regard to the first of these cases, I am inclined to agree with Dr. Cameron. I think that the diagnosis of syphilitic arthritis raises the question whether one should regard a positive Wassermann reaction as always indicating that the lesion under observation is syphilitic, seeing that syphilis is very prevalent and a positive Wassermann is also very prevalent. If the lesion were bilateral, and if it had reacted to antisyphtilitic treatment, one would be inclined more favourably to a diagnosis of syphilis. I strongly suggest that the Wassermann test be taken again. And, precisely, what treatment has been employed? The onset and the early reaction to salicylates suggest a subacute rheumatism of one joint, a condition occasional though rare. With regard to the second case, on looking at the child I was at first inclined to agree with Dr. Turtle that it is an example of Still's disease. But one would expect to find glandular or other lymphatic enlargement. The spleen has not been found to be enlarged, and I could not feel axillary or other enlarged glands. The periostitis is suggestive of syphilis, but I do not think it is an ordinary case of syphilis. There is great destruction of bone, causing considerable excavation, as seen in the radiogram, which is in favour of tubercle, and against syphilis.
Miss King (in reply): The boy has had mercurial inunction, as well as mercury by the mouth until the gums became spongy, and then we dropped the inunction. He is also having quite large doses of iodide of potassium.

Dr. Langmead: The first case began like acute rheumatism, and subsided under salicylates. I thought it was probably tubercular, and Mr. Blundell Bankart, whom I asked to see the case, agreed that it should be treated as such, but as the boy allowed very free movement of the joint without expressing a feeling of pain he suggested a Wassermann reaction.

The President (in reply): I shall have another Wassermann test done on my case, and yet again if the next comes out negative. In suspicious cases I have it done three times. I only heard late to-day that the report was negative. I was showing the case as one of syphilitic joint disease in a child. I think still, with most of you, that there is strong evidence of it being syphilitic. It is curious how localized the syphilitic manifestations sometimes are. I remember one case in which the only evidence we could find of the cases being syphilitic was disseminated choroiditis: there was nothing else. Blood examination showed the child had congenital syphilis. A brother of that patient also had a single manifestation—namely, paroxysmal haematuria. Therefore, the absence of other manifestation of syphilis does not impress me much. But the skiagram shows periostitis of the tibia above the ankle. I have rarely seen a syphilitic joint without periostitis of the neighbouring bone, and when I saw this present case I thought it curious that there was no more evidence of it, though the X-rays show it to be present. The case shown by Miss King is, in my opinion, tubercular disease in a patient with congenital syphilis.
Section for the Study of Disease in Children.

President—Dr. J. Porter Parkinson.

Chorea, complicated by Gangrene of the Fingers.\(^1\)

By Hazel Chodak, M.D., M.R.C.P.

V. H., aged 12 years, was admitted into the Royal Free Hospital on December 7, 1918, suffering from chorea of a week’s duration. This was a first attack, and there was no previous history of rheumatism; no history of shock or overwork. Two years previously she had had diphtheria, with a bad attack of tonsillitis during convalescence. The mother had had rheumatism and one sister has had chorea. On admission the patient, a thin slip of a girl, was found to be suffering from a moderately-severe attack of chorea, all parts of the body being affected. There was very little loss of strength on the left side, but the right hand grip was poor and feebly sustained. All reflexes were exaggerated.

On examination of the heart, the apex beat was found in the fourth space, half an inch inside the nipple line; a soft blowing murmur accompanied the first sound at the apex, and was conducted a short way toward the axilla; the second sound was accentuated at the base.

Ten days after admission the right hand began to go white, the finger-nails blue, though the hand did not actually feel cold to the touch. The onset may be described as rapid rather than sudden, and it was fully a week before gangrene of the finger-tips and ball of the thumb had definitely set in. During this time the pallor spread up the forearm. There was no pulse at the wrist, but the brachial could be felt pulsating about half-way down the upper arm, and after a time there was distinct pulsation of the superior profunda artery. The

\(^1\) At a meeting of the Section, held June 20, 1919.
pain, which was also gradual in onset, became very severe after the first few days, and could only be relieved by morphia. Meanwhile the cardiac signs showed changes in degree rather than in kind. The systolic murmur became much louder and rougher, while the second sound at the aortic and pulmonanty areas was distinctly accentuated. At first the apex beat remained within the nipple line, but in a few days it was found to be displaced slightly outside the nipple line. The pulse, however, remained at about 80, and was never increased in frequency, except occasionally when the pain had been very severe. The temperature throughout never rose above 99°F., and was rarely as high as that. Later still, the brachial pulse slowly disappeared, and the brachial artery could be felt like a thick cord along the arm. The little finger recovered, and lines of demarcation gradually formed on the remaining fingers. The ball of the thumb appeared at first to have escaped as the discoloured skin peeled away from it, but there must have been considerable damage to the muscle, followed by contraction of the scar tissue, which has led to considerable deformity of the thumb. The choreic movements subsided rapidly soon after gangrene was established. The heart signs also disappeared, but much more gradually.

The question of interest in this case is the exact cause of the gangrene. The three possible causes of gangrene complicating chorea are:

1. Embolus.
2. Arteritis (leading to thrombosis).
3. Arterial spasm (resembling Raynaud's disease).

Raynaud's disease is mentioned as an infrequent complication of acute rheumatism, and may lead to local gangrene, but in this case there is extensive thrombosis which can hardly have been caused by mere spasm.

As between embolus and thrombosis, the diagnosis is not easy. I do not think that the absence of abrupt onset negatives the embolism theory, as conceivably the artery might be only partially blocked at first. It is difficult, however, to feel convinced that there was ever any gross organic lesion of the heart, as the pulse and temperature kept so steady. At the time, however, the slight dilatation, the character of the murmur, and the loud accentuation of the second sound, made one believe that this was the beginning of a rheumatic carditis. There remains the supposition that there was a primary thrombosis in the brachial artery.
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Arteritis is not an infrequent complication of acute rheumatism, and although this is generally located in the aorta, there seems no reason to suppose that the brachial artery may not be affected also. Such a condition might be speedily followed by thrombosis. In these cases, the circulation is usually kept up by anastomosing channels, so that gangrene does not often occur. Probably in this child the general condition of debility and anaemia was a predisposing cause.

Finally, I should like to point out that an interesting detail in the history of the case was the attack of tonsillitis occurring during convalescence after diphtheria. Was this attack perhaps the actual point of entry of the rheumatic organism into the circulation?

DISCUSSION.

The President: With regard to the diagnosis, I agree with the exhibitor that the condition is due to an embolus. We know that the onset, in such cases, is usually sudden, though not always so. That was brought to my notice only during the last fortnight in the rather remarkable case of a policeman who was brought into the hospital because he was coughing up a considerable quantity of blood: he had mitral disease. He said he never had pain in his chest, that the blood-spitting came on gradually, and in fact the whole onset of the condition appeared to have been slow. In two days he died, and we found a large infarct occupying practically all the lower lobe of one lung. It was a definite case of embolus in the lung, which had not caused any dyspnoea nor other urgent symptoms such as is generally the case. If that sort of thing can be found post-mortem, when embolus seems to be such a likely solution, one does not exclude it because the onset was not quite sudden.

Dr. J. D. Rolleston: I agree with the President and Dr. Chodak with regard to the pathogeny of this condition: I think it is probably embolic, as are most of the cases of gangrene following acute infections. In 1910 I showed, before the Section, a case of gangrene following diphtheria. In that patient the lower limb was affected, as in most of the cases following acute infection, and on that account this case is a very unusual one. As regards diphtheria, for instance, there has been only about one case in which an upper limb was affected as a result of an embolus. In reference to the last point raised in the notes, as to the occurrence of tonsillitis in convalescence from diphtheria, several years ago I wrote a paper on the subject of tonsillitis in diphtheria, in which I said that in 900 cases it occurred in sixty-four. In thirty of the sixty-four it occurred about ten days after the throat had become

clear in association with serum manifestations, what the French call "angine de retour." In thirty-four of my cases there was an attack of apparently ordinary follicular tonsillitis much later, about the fortieth day. Therefore this is not a very uncommon condition in those who are convalescing from diphtheria.

Dr. F. Parkes Weber: I think the cause of the gangrene was embolism, and it seems to me that the onset was really acute. At the onset there was pallor of the hand, with local pain in the upper part of the arm. It required some time for the blocking of the artery to become quite complete, but the gangrene developed fairly early, in spite of the blockage being incomplete at first.

Dr. Chodak (in reply): I think it is right to regard the onset as acute, but one was inclined to speak of it as subacute because the pain came on so gradually. Of course, an embolus may have lodged in the site and the gangrene commenced acutely, though the pain took three days to culminate: its onset was so gradual that only after twenty-four hours did the patient think it necessary to mention it at all.

**Chloroma or Adrenal Growth with Secondary Metastasis in Skull.**

By Edmund Cautley, M.D.

Male, aged 2 years, 10 months; admitted on March 11 for anæmia. He was in previously from February 4 to 18, with a history of having been ailing and fretful for three weeks; occasional vomiting, constipation, screaming at night and lying curled up in bed. He had right otorrhœa on and off for about a year. Examination showed right facial palsy, slight rigidity of the neck, one torticollis, normal reflexes and cerebro-spinal fluid, and no otorrhœa. He was feverish and drowsy, but his temperature fell next day and, on discharge, he was well except for slight facial paresis and anæmia.

When re-admitted for anæmia, nothing abnormal was found except slight bulging in the temporal regions and right facial paresis. Head, 19 in. Blood: Secondary anæmia and lymphocytosis. No evidence of enlarged glands in the chest, on X-ray examination. Left kidney palpable. At first he gained weight fairly rapidly. Then he began to lose weight, take food badly, get paler, and cried with pain. An X-ray plate of the hip-joints showed rarefaction of the heads of the femurs, which was said to be tuberculous. The fundus oculi showed nothing
abnormal, save a good deal of mottling on May 5. Nodules on the head were noted on May 24; Wassermann negative on May 29; enlargement of spleen, liver and glands in groin on 29th. Head, 22 in. on June 3. Progressive anæmia and enlargement of the tumours of the skull, and of cervical, submental, submaxillary and other glands. Slight yellowish-green tint of complexion. Blood: Red cells have decreased down to 1,690,000, Hb. to 25.5 per cent., and colour index to 0.79. In a count of 400 white cells there were seen 18 normoblasts, 17 polychromatophils, poikilocytes, megalocytes, microcytes and anisocytosis. Leucocytes, 8,000 (March 6), 9,600 (April 2), 14,000 (May 28). Polymorphs have increased from 1,940 to 5,845 (24.35 to 41.75 per cent.); lymphocytes from 5,660 to 7,140 (70.75 to 51 per cent.): large mononuclears, 8, 12, and 9; eosinophils, 7, 3 and 7; basophils, 1, 1 and 3; myelocytes, 4, 3 and 10 in the successive counts.

Multiple Glandular Swellings.

By EDMUND CAUTLEY, M.D.

Male, aged 1 year, 9 months, one of three children, all living: parents healthy. Measles April, 1918, followed by pertussis; diarrhoea and vomiting in September; no other illness. Ailing since Christmas, on and off. Chief symptoms: diarrhoeal attacks, marked anorexia, and general malaise. Enlargement of glands and abdomen noted about the same time. Admitted on June 6. Much wasted; large, freely movable glands in neck, groins and right axilla; abdomen prominent and liver large. Blood: Hb., 40 per cent.; white cells, 11,400; polymorphs, 41 per cent.; small lymphocytes, 32 per cent.; large mononuclears, 11 per cent.; hyaline, 5 per cent.; transitional, 10 per cent.; eosinophils, 1 per cent. Total red cells, 6,000,000; no nucleated reds. Wassermann negative from the laboratory standpoint, but might be regarded as doubtful (Dr. Sanguinetti).

The child has improved a little since admission.

DISCUSSION.

Dr. Cockayne: I have seen two cases of so-called suprarenal sarcoma: they were included in Dr. Frew's paper in the Quarterly Journal. In one of them, the first sign was hemorrhage into the tissues of the orbit, before any swelling due to new growth appeared. The other had very early swelling.
and proptosis of the eyes with hæmorrhage into the orbit. In neither of
them was there the peculiar yellowish colour seen in Dr. Cautley’s case. I
think this first case of Dr. Cautley’s is one of chloroma, not suprarenal
sarcoma, though the age and the location of the secondary growths are
similar in both conditions.

Dr. Eric Pritchard: This first case is probably not one of chloroma. I
had two cases of sarcoma of suprarenals, both of them commencing like Dr.
Cockayne’s case, with hæmorrhage into the eye and proptosis. One of them
had quite as chlorophilic a colour as the present patient, in fact, if anything,
it was a little more green. As chloroma in a child is exceedingly rare, and
sarcoma is common, I think this is more likely to be sarcoma. The left
kidney is certainly enlarged, and I should not be surprised if there were a
tumour there. If there is one, probably it will develop rapidly.

Dr. F. Parkes Weber: With regard to Dr. Cautley’s first case, I would
only point out, for what it is worth, that the Röntgen skiagram of the skull
shows no bony spicules projecting outwards from the outer table. In chloroma,
with such an extensive involvement of the cranium, I should have expected bony
spicules to be present, projecting outwards into the cushion formed by the
diffuse chloromatous periosteal infiltration. Such a condition was most
strikingly present in Firth and Ledingham’s case, brought before this Society
some time ago.¹ With regard to Dr. Cautley’s second case, I would suggest a
third possibility. Dr. Cautley mentioned some form of glandular tuberculosis,
and in that condition there might likewise be intestinal tuberculosis, which
would account for the diarrhœa. He also suggested that some form of lymph-
adenoma, that is, Hodgkin’s disease, or “lymphogranulomatosis maligna.”
The asymmetry in the glandular enlargement perhaps is in favour of Hodgkin’s
disease. But, as a third possibility, I suggest that it may be a leukæmic case,
even though at present the blood does not show typical changes. In the paper
I have contributed to the current number of the Quarterly Journal of Medicine
on “Acute Leukæmia and so-called Mediastinal Leucosarcomatosis,”² I give the
case of a boy, aged 7 years, who was admitted to hospital under the suggested
diagnosis of tuberculous peritonitis (with peritoneal effusion). There was
enlargement of the superficial lymph-glands. It turned out to be a case of
acute leukæmia, and the latter diagnosis was proved to be correct by the post-
mortem examination. Therefore I suggest the possibility of Dr. Cautley’s
second case being one of atypical leukæmia, and I would like to see a Röntgen
skiagram of the chest, because very decided enlargement of the glands in the
mediastinum would favour the diagnosis of leukæmia with so-called media-
stinal leucosarcomatosis. [Dr. Cautley now tells me, however, that Röntgen-
ray examination has been carried out, but without showing anything special
from the point of view of diagnosis.]

(Sect. Path.), p. 60.
Dr. F. Langmead: With regard to the first of these cases, I find myself in the same difficulty as does Dr. Cautley. The appearance seemed rather to favour chloroma, whereas the early swelling of the kidney and the blood changes would favour it being adrenal sarcoma. And, as Dr. Cautley said, since sarcoma is far commoner and there may be such colouring in it, I think that the probable diagnosis. The second case I regard as lymphadenoma. I think the glands are too hard for acute leukæmia. I have watched a case of lymphadenoma which began at the age of 3½ months, and went on for several years. It started with a mass of glands on one side. Those glands were excised, the scar being treated with X-rays. The child lived for five or six years afterwards, dying eventually with general glandular enlargement.

The President: I am inclined to agree with Dr. Pritchard and Dr. Langmead as to the first case being a primary tumour of the suprarenal, chiefly because I think the left kidney is much enlarged. I think there is enlargement of the right kidney too. The tumours in the bones of the skull are probably secondary, similar to what was seen in a case shown here some years ago. Absence of proptosis in this condition is uncommon. In this case there has been no proptosis at any time. I do not think the presence of proptosis is a necessity before you can diagnose the condition. The patient has a pallid look, but I cannot detect much green discoloration. It is of much the same colour as in the case I showed here some years ago.

Dr. Jewesbury: Does Dr. Cautley propose to have one of the glands excised in his second case? The diagnosis of these glandular cases is always a matter of considerable difficulty, and the only way to clear up the doubt is to submit a section to the microscope.

Dr. Cautley (in reply): I propose to have the whole mass of glands in the axilla excised. I do not regard the glands as tuberculous, but as lymphadenomatous. A microscopical examination will enable us to decide. I have nothing to add about the case shown as possibly chloroma, except that one is rather in a difficulty as to the causation of the anaemia. If this is a case of primary adrenal tumour, I do not see why a small adrenal tumour should cause anaemia in a child for months before secondary growths develop. Still, it is a possibility, and I think that in this instance we shall be able to verify the diagnosis later on. On the other hand, the differential blood counts are not in favour of chloroma.

Note.—The child died two days later during an attack of acute abdominal pain. At the autopsy a large tumour, apparently a subperiosteal sarcoma, was found attached to the anterior surface of the dorso-lumbar region of the spine. The kidneys were not enlarged. Extensive growth separated the dura mater from the calvarium, which was necrosed in several areas, and the brain was unaffected.

AU—5a
Sclerodermia with Calcification in a Mongol.

By F. S. Langmead, M.D.

The patient, a boy, aged 4½ years, is the last child of a family of three. The mother was aged 42 years at the child's birth. The other children are aged 17 and 15 years, ten years elapsing between the last gestation and the birth of the patient. The mother says she has always been healthy except that she has suffered from anaemia. About five years before the birth of the patient she began to suffer from severe menstrual pain and from a foul vaginal discharge. One year before the pregnancy she was admitted to St. Thomas's Hospital and had an operation for "a tumour." During pregnancy she felt quite well. The father and his family are described as highly strung and nervous. There appears to be no neuropathic tendency in the mother. No alcoholic history.

The boy was small and appeared healthy at birth, but the mother noticed a discoloration and rash similar to that which is now visible, on the knees, face and hands. She is sure that the rash has always been present, but has varied in intensity from time to time. He has always been backward, sitting up at 10 months, walking at 1 year and 10 months. Walking has never been properly acquired, but has been feeble with the legs apart. Until the last few weeks he had ceased walking altogether for two and a half years, and became unable to stand. In October or November, 1917, he was admitted to the Victoria Hospital for Sick Children for "blueness" over the fronts of the upper arms, which had begun to spread to the chest over the pectoral muscles. This apparently diminished considerably before his discharge. In December, 1917, he developed pneumonia, but made a good recovery. In June, 1918, lumps began to appear on the surface. At first they were quite soft and limited to the area over the biceps, but about two months later began to harden. More recently they have been noticed to spread inwards over the pectoral region. Flushing of the face varies greatly. It has been noticed for about two years, and is more marked on warm days. The legs have been noticed to be getting thinner for about six months, and about two months ago stiffness of knees and ankles supervened.

Present state: The child is a moderately marked example of the Mongolian variety of amentia, but with mental capacity above the
average in such cases. The cheeks have a dusky red, patchy appearance, the skin being somewhat atrophic and shiny. A similar blotchy bluish-red discoloration is seen on the fingers and hand as far as the wrists. The fingers are shiny and small, but no definite sclerodactyilia has developed. The skin on the knees, extending upwards on the outer aspects of the thighs, and on the buttocks in the neighbourhood of the

ischial tuberosities, is similarly affected. In the arms the discoloration has disappeared, and is replaced by white, firm, contracted skin, thrown into unevenness and puckers by subcutaneous nodules of sizes varying from minute seed-like bodies to plaques larger than a shilling. Some of these are confluent, others discrete. They are roughly linear in distribution and symmetrical, though rather more extensive on the
left side than on the right. This condition has spread inwards over
the pectorals, and backwards over the triceps on each side. On the
left it reaches somewhat farther down beyond the elbow, and over the
extensor carpi ulnaris muscle. A similar alteration of skin and sub-
cutaneous tissue appears in a roughly symmetrical manner in other
areas, notably on the thighs, legs, and in the popliteal spaces. Some of

the subcutaneous thickened areas are adherent to the skin, others are not.
The consistence of some of the nodules is very hard, so that they feel
almost like bone. Here and there a nodule has caused redness and
soreness of the overlying skin and slight scab formation. X-ray exami-
nation shows that the subcutaneous tissue in the nodular and thickened
areas is sprinkled with small pleomorphic calcareous deposits. The
muscles do not seem to be affected, but it is questionable whether they are not being gradually infiltrated from the subcutaneous layer. The movements are limited by the inelasticity and fixation of the skin. In addition there is some limitation of movement of the knees and ankles disproportionate to the subcutaneous hardening, probably due to accompanying synovial changes.
The points of interest are:

1. The discolouration of the skin dating from birth, and later being replaced by sclerodermia.
2. The calcification of the sclerodermatous subcutaneous tissue (calcinosis, petrifaction).¹
3. The associated arthritic changes.

And, with respect to the Mongolism,
4. The long period of sterility before the child was born.

DISCUSSION.

Dr. F. Parkes Weber: The calcification in Dr. Langmead's case resembles the condition in the case which I described before the Section of Dermatology, in the Seventeenth International Congress of Medicine (1913), in London.² I do not know of other published cases which exactly correspond to these two. I am not convinced that there is true scleroderma present in these two cases. There is, however, a group of cases of generalized scleroderma (sclerodactylyia) in which nodules of subcutaneous and cutaneous calcification occur, especially in the fingers.³ In my case one was able to see what happened in the course of a year or two: marked improvement took place, together with great diminution in the calcareous deposits, as judged by the Röntgen skiagrams. The child was treated chiefly by means of wholesome food and iodide of iron.

Mr. Mortimer Woolf: What is the prognosis in these cases? I have in mind a case which attracted a good deal of public attention years ago, as it was extensively reported in the Daily Mail, under the title of "The Brittle Man." He was under the care of Dr. Theodore Thompson, at the London Hospital, and I was his house-physician at the time. The X-ray appearances were similar to those in this case, and I was particularly interested in hearing Dr. Langmead say scleroderma affected the muscles. There was bone in the muscles of the case I speak of, because we cut out a piece from the pectoralis major and had it microscoped, showing bone cells. That patient was aged 15 years when first attacked. Are the two conditions really separate or is the underlying pathology similar?

Dr. Cockayne: The interesting feature in Dr. Langmead's case is the early age of onset. I think there are on record only three cases of congenital

generalized scleroderma. In Dr. Langmead's case the actual scleroderma was not present at birth, but changes were present at birth in the skin, which later became sclerodermatous. If scleroderma is due to an infection, it is interesting that it can start before birth.

The President: The question of the cause of mongolism has always interested me, and I noticed that in this case the mother had a long period of sterility before the birth of this child. I recently had a case of a Mongol brought to me whose parents were healthy, were between 20 and 30 years of age, and this child was born within two years of their marriage. And this was their first child. Another child, born to them not very long afterwards, was normal in every way. So that the mother's general health does not seem to be so important in this matter as has been generally thought. What is Dr. Langmead's view of the causation of mongolism?

Dr. Langmead (in reply): In answer to the President, I do not know what the cause of mongolism is. It is certain that in a good number of cases the parents appear to be quite healthy, but the patients seem to be more often first children of the marriage, or else the last in a long series—either one or the other. It is suggested that in some cases the age of the mother is the important factor, that is to say, they are born at a period when the mother is getting past the ordinary child-bearing period. But that is not always so, though a Mongol is sometimes the first child of a mother who is over 40 years of age. In answer to Dr. Weber, I would suggest that sclerodactylias is being produced in this child: the skin of the fingers seems to me to be somewhat atrophic and shiny, and the fingers are certainly small. We ought to make a clear distinction between myositis ossificans and such cases as this. When I said the condition sometimes affects the muscles, I did not mean calcification. I meant that scleroderma sometimes spreads to muscles, in other words, there is fibrosis in muscles as well. As far as I know, the calcification of scleroderma does not involve muscles, it begins in the subcutaneous tissue. The two conditions should be kept distinctly apart. With regard to prognosis, the condition has not been studied long enough for us to know what will happen. I thought, before Dr. Parkes Weber spoke, that one must expect an increased scleroderma and calcification, so that the child will become more hide-bound than it is now.

Apophysitis of Os Calcis.

By Paul Bernard Roth, F.R.C.S.

This condition has been described by J. Warren Seaver, M.D., and his account of it with radiograms of several cases appeared in the New York Medical Journal for May 18, 1912. According to him it is a
painful condition of the heel which always occurs in children,characterized by a slight persistent limp, with a marked disinclination to complete the full step in walking. Tenderness is complained of about the posterior aspect of the heel, low down, which has persisted for several weeks or months without change. There may also be a slight amount of pronation of the foot. Examination shows an area moderately tender on pressure, situated over the posterior portion of the os calcis, and localized in front of the tendo-Achillis on either side. The motions of the foot are all slightly limited, especially dorsiflexion, and any movement which tends to put a strain on the tendo-Achillis causes pain. Radiographically there is to be seen an enlargement of the epiphysis itself on the affected side both in thickness and in length from top to bottom. There is also considerable cloudiness along the epiphysial line suggesting a deposit of new bone. Often the condition suggests a slipping of the epiphysis with the customary inflammatory reaction. Similar conditions existing in the tibial tubercle have been spoken of as Schlatter's disease.

Normal Ossification of Os Calcis.—The bone is developed from two centres, one for the great mass of the bone which appears at the sixth month of fetal life, and one, an epiphysis, for the posterior end including the great tuberosity, about the date of appearance and fusion of which with the rest of the bone there is some difference of opinion. Thus Quain, Holden, and Gray state that it appears at the tenth year and fuses soon after puberty in the fifteenth or sixteenth year; but according to Cunningham, the centre appears from the seventh to the ninth year and unites between the ages of 16 and 20 years. Seaver puts the date for its appearance as the seventh year.

Case.—Dr. X. wrote to me on July 14, 1918, as follows: "My son, aged 14 years, has been suffering for some years with double flat foot. I have tried various methods of treatment but he gets no better, but seems to get worse." I examined the boy on July 29 in his father's presence and made the following note: For last ten years or longer has had trouble with his feet; last two years pains in his feet and ankles up to his calves when he runs; can't walk for quarter of an hour without sitting down; can't play any games: played football two years ago and overdid it, and since then has not been able to play; can only just walk from his house to the schoolhouse (ten minutes' walk). Has had various arch supports and appliances: thickenings of the soles and heels of his boots; electric baths and massage; present boots with heels prolonged and wedged soles and arch supports were ordered by a
Fig. 1.

Fig. 2.
specialist in February; but still the pain continues and the father is beginning to wonder. Examination: Has excellent feet; there is a slight tendency for his feet to be valgus when he walks, that is all; boots a good shape: all the pain is at the back of the heel and up the tendo-Achillis; there are tender spots on both sides of insertion of tendo-Achillis. I suggest this is a case, not of flat foot, but of inflammation from trauma of posterior epiphysis of os calcis."

Radiograms taken on August 6 by Dr. X. showed a cartilaginous gap with irregular ossification between the upper part of the epiphysis and the os calcis, especially marked in the left—the worse—foot, and some rarefaction of the bone at this point (see fig. 1). As these radiograms confirmed the diagnosis, I suggested treatment by prolonged rest in bed, followed by gradually increasing spells of walking in boots with a rubber disk fixed to each heel, a pad of sponge rubber beneath each heel inside the boot, and a ¼-in. valgus wedge of leather applied to the inner side of each sole and heel. This treatment could not be carried out at once, but the following letter from Dr. X., dated November 9, well describes the subsequent course of the case:—

"I think you will be interested and pleased to have a short account of my boy's progress. After his visit to you in July, he went with his family to the seaside in Devonshire, and it was not practicable to follow your advice immediately; but during the month he was away he undoubtedly got worse, although he used his feet very little for walking and got about almost entirely on his bicycle, and this only about two or three miles a day. By the end of the holiday he could not walk more than one or two hundred yards without resting and suffering very considerable pain. On September 1, on his return home, I put him to bed and kept him there without once putting his feet to the ground for six weeks. When he first got up and walked he had pain, but of a different character, which by the end of a fortnight was quite gone, and was evidently only due to stiffness and muscular weakness from his long rest. It is a month now since he got up, and he has increased gradually his walks, and can now walk a mile in the morning and the same in the afternoon without any pain. He has even tried running a few steps with the same good results. He is wearing boots as you advised. I enclose radiogram prints (see fig. 2) taken a few days ago, and if you compare them with the former ones, I think you will see a very marked difference. The wide cartilaginous band with irregular ossification has given place to sound bony tissue through its entire length, except a very small portion at the top (most marked in left foot) where the junction of the epiphysis is not quite complete."
Section for the Study of Disease in Children

According to Seaver this condition is not uncommon, but the case described is the first I myself have been fortunate enough to observe.

DISCUSSION.

Dr. Eric Pritchard: I take it from what Mr. Roth said that this was taken for a case of flat-foot. Was there any radiation of pain to parts other than the os calcis to lead to such a diagnosis?

Mr. P. Bernard Roth (in reply): No, there was no such radiation of pain as Dr. Pritchard refers to. As I say in the note, he had slight pronation of the foot. There was no flat-foot, and no pain in the front two-thirds of the foot at all. When people come complaining of pain in the foot it is very common for it to be attributed to flat-foot. Those who had seen him before, seeing the slight pronation, regarded it as flat-foot, and that diagnosis having been once made, was handed on without sufficient examination.

Case of Severe Rickets in a Child of Three Years.

By G. Graham, M.D.

F. M., the ninth child of a family of Polish Jews. The child was breast fed till the age of 3 months. She was then given glaxo for three weeks, but was admitted to St. George's Infirmary for wasting. She was discharged after four months and has had ordinary food with little milk ever since. She began to crawl at the age of 2 years, but never stood up, and for the last year has not crawled. Since Christmas, 1918, she has not moved her legs, and she ceased to move her arms about Easter, 1919. She can only say a few words.

Condition on admission to East London Hospital for Children, Shadwell: A fat flabby child; height, 27 in.; weight, 1 st. 7½ lb. The head is square in appearance but only slightly bossed. She has sixteen teeth and the gums are fairly healthy. The chest is small in comparison to the large abdomen. The clavicles are bent forwards at an acute angle about 1 in. from the sternum and are freely movable at the angle. She does not move the upper arms but can move the forearm and hands. She cannot feed herself. The lower end of the radius is large. The carpus and metacarpus appear normal, but the phalanges are spindle shaped so that the interphalangeal joints are smaller than the middle of the phalanges. The legs cannot be moved at all. The lower end of the tibia is large while the tarsus is small. The metatarsus is much
wider than the tarsus and gives a wedge-shaped appearance to the foot. The phalanges are normal.

The X-ray photographs were very difficult to take as the shadow of the bones was very little denser than that of the soft tissues. I have to thank Dr. George, of St. Bartholomew's Hospital, for taking the plates. The plates show numerous old fractures. The callus around the fracture of the left femur throws a denser shadow than the bone itself. The interval between the end of the shaft of the long bones and the epiphysis is very wide. The shadow of the teeth was quite dense. The peculiar swelling of the fingers and of the feet do not show on the plates. During the last three weeks the general condition has improved greatly. She can feed herself and move her legs. She cannot lift her head but if put in a sitting position she does not fall over.

DISCUSSION.

Mr. P. Bernard Roth: I think this condition should be regarded as fragilitas ossium accompanying rickets. I do not see how rickets could account for all those fractures. The child's head bulges at the sides, which is one of the characteristics of fragilitas ossium. And would not the presence of fractures account for the child not being able to use her legs and arms? After these fractures, it takes a long time for the union to become sound. I operated upon a case of fragilitas ossium, the patient being a girl, aged 16 years; it was for straightening the tibia, and it was eighteen months before the union was sound. And might not the inability to move the head be due to fracture of the clavicle? The clavicles are, to some considerable extent, concerned in holding up the head, through the attachment of the sternomastoid muscles, and in this case both clavicles are "wobbly," like rings on a jar.

The President: I did not hear whether there was pain in this case in the earlier stages. Scurvy might account for some of the features Dr. Graham mentioned, it would account for a considerable amount of pain which would prevent movement. I also ask whether there was blood in the urine, as there is in mild cases of scurvy. There may be both rickets and scurvy.

Dr. F. Parkes Weber: I suppose the President's suggestion might explain the slight changes in the phalanges of Dr. Graham's case, which are undoubtedly present according to the Röntgen skiagrams. According to the skiagrams these changes resemble somewhat those of periostitis, or of so-called "secondary osteo-arthropathy," but they may be merely a result of slight scorbutic subperiosteal haemorrhages.

Dr. Eric Pritchard: I do not see why this should not be regarded as an ordinary case of severe rickets. It has the typical symptoms, the shape of
head and flatness of the top, with beading of the ribs, and extreme calcification and marked paresis. In severe cases of rickets one of the marked symptoms is paresis of muscle—which according to the view I hold is cause and effect: where there is paresis of muscle there lies the chief cause of rickets. As soon as you can get a child on to its legs so that it can use its muscles, the symptoms of rickets begin to disappear. The explanation is, I believe, that it is the use of the muscles which causes the chief demand for food, for oxidation and increased metabolism. Seeing that this child has improved so much under better hygienic conditions, I think that it will be walking in a few weeks if it is encouraged to use its muscles and they are massaged. I do not see anything in this patient to make one think it is suffering from fragilitas ossium. The clavicles are much distorted: they have both been the seats of fractures, they may possibly have been submitted to unusual strain. I have at the present time, in hospital, a child with extreme rickets in which the legs are quite straight, but one arm is bent at an acute angle because, although the child could not stand it did sit up and rested the weight of its body on its arms. Distortions of bone occur where the strains happen to be imparted.

Dr. Graham (in reply): Pseudo-paresis was one of the conditions we thought of. But the child had very little pain, there was none of the fear of being touched which scorbutic cases exhibit; the gums were practically normal and bore no resemblance to spongy gums.

**Trophœdema of Leg.**

By E. A. Cockayne, M.D.

S. S., Male, aged 8½ years. No history of similar condition in family. The boy was normal at birth. At the age of 1½ years there was a swelling, rather soft and lobulated, in Scarpa's triangle on the right side. Three months later the leg below the knee began to enlarge, and has increased in size gradually ever since. At the present date there is enlargement of the whole leg below the gluteal fold, much more below the knee than above. The right thigh is about 1 in. greater in diameter than the left, and is soft. No swelling can be felt in Scarpa's triangle. The lower leg and dorsum of the foot is enormously swollen. Circumference of right calf, 13½ in.; left, 9½ in. Sometimes there is no œdema, at other times it pits on pressure. The texture of the skin is normal, and no naevus is present. There is no cyanosis. The X-rays show no enlargement of the bones. There is no lengthening of the limb. The right side of the face is a little larger than the left, and the right palpebral fissure is wider. The condition causes the child very little inconvenience.
DISCUSSION.

Dr. J. D. Rolleston: The case is very interesting to me because it is very like a typical case of Milroy's disease which I showed at the Clinical Section two years ago in an adult. Although it does not possess one of the essential features of Milroy's disease—the congenital feature—yet it shows the others which Milroy described: persistent œdema, the fact that the œdema is confined to the lower limbs, and the absence of constitutional disturbance. Dr. Cockayne told me this boy has no local trouble, that he can play football and other games. That was so in my case of Milroy's disease: the patient was a daily worker in a military hospital, and did her duties without inconvenience. I think lymphangioma is a little too definite a name: I would prefer the non-committal term of persistent œdema. I glean from the literature that none of these cases had come to autopsy, therefore a variety of unconfirmed theories have been advanced. One of these who have done most work at the subject—Meige, of Paris—called it trophædema.

Dr. F. ParkeS Weber: I think this is a non-familial example of the class which has been often described in France under the name "trophædema." It is generally supposed to be the same condition as Milroy's disease, except that Milroy's cases were so markedly familial; but several of the French cases were also definitely familial. I think the disease is commoner without than with any history of other members of the patient's family being similarly affected. Some of the cases are congenital, some develop later in life. Some involve only the portion of one lower extremity below the knee, others involve the whole of one lower extremity up to the gluteal fold, and others again involve both lower extremities. One lower extremity may be affected below the knee, whereas its fellow may be affected right up to the gluteal fold. Occasionally the upper extremity is affected. The œdema in these cases need not particularly involve the end of the affected limb.

Dr. Langmead: Is not the way in which the condition started in this case against the rule for trophædema? Apparently it began as a swelling in Scarpa's triangle. In trophædema is it not the fact that the swelling is at first transitory, and later persistent, and begins about the ankle and dorsum of the foot?

Dr. Graham: It might be worth while to examine the blood of this patient. There is, at Shadwell, a Roumanian child who has been in this country all its life, and its parents have been here twenty years. The child developed a swelling of the left leg, and Dr. Bhat, the resident medical officer, looked frequently at night and at last found filariæ in the blood film. That examination might be made in this case for the purpose of excluding that condition.

Dr. Cockayne (in reply) : The possibility of filaria did not occur to me. The child has never been out of England, and I was not aware that filaria was ever acquired in this country. The difficulty about the blood examination is, that it would have to be done at night, and the child lives in the country. But I will try to arrange it.

Specimens from Case of Purpura.

By Eric Pritchard, M.D.

Aged 1 year and 2 months. Child fed on milk; no fruit juice given. Family history: Father and mother and one child healthy. Past illness: Pneumonia last December; burn on neck three weeks ago. History of present illness: for last week child had ulcerated mouth; treated with borax. On May 11 a bruise on left leg was noticed: increased in size very rapidly so was admitted to hospital on May 12.

On admission: Temperature, 99.4° F. Well-nourished child; extremely pale. Large blue areas over left leg (6 in. by 8½ in.) and thigh (2¾ in. by 3½ in.) Also patch on right leg (3 in. by 3½ in.). Petechial haemorrhages over legs. Nil on body nor arms. Enlarged gland left side of neck. Discrete glands right side of neck. Chest: Lungs, nothing abnormal detected. Heart, normal size; no murmurs. Alimentary: Gums firm, no ulceration; teeth very discoloured; liver 1 in. below costal margin.

June 12, 1919: Scrotum very oedematous. Purple patch spreading over left foot. Child died same day.

Post-mortem Examination by Dr. W. P. Hillier.—There is an unhealed ulcer on the left side of the neck due to a burn. It consists of two areas of about ½ in. diameter each, joined together. There is subcutaneous haemorrhage about ¾ in. across on the chin, and another area of about the same size on the right side of the neck. Large subcutaneous dark purple haemorrhages are seen on both legs. On the right side one haemorrhage is about 2 in. in diameter over the patella, another large one is situated on the inner side and back of the middle of the leg, and all but joins a third one on the front and outer side just above the ankle. On the left side the lower part of the leg is encircled by a haemorrhage measuring about 4 in. from above downwards and the epidermis over it is raised as a bulla. Both kidneys with their perirenal fat are the seat of extensive haemorrhages which are almost entirely cortical. Both testes are dark red from haemorrhage. The only other haemorrhage
observed was in the middle portion of the thyroid gland. The thymus is a well-marked bilobed structure of ivory-white colour. Prepuce oedematous, liver pale, spleen slightly enlarged. Heart, lungs, pericardium, stomach, intestines, pancreas, adrenals, all healthy. Head not examined.

Specimen of Liver Abscess.

By Eric Pritchard, M.D.


On admission: Child looking very ill; temperature, 101° F.; pulse, 132; respiration, 42. Fairly well nourished. Alimentary: Vomiting bright green material; abdomen distended, soft; no visible peristalsis; no tumour visible; liver and spleen not palpable. Lungs normal; heart normal.

May 31, 1919: Vomiting after breast and after broth. One test meal, 6'9 dr.; another test meal, 6'97 dr. Stomach wash-out, two and three quarter hours after feed, contains 1 oz. of green, slimy fluid; acid ++; size of stomach, 2 oz. Rectal lavage almost clear, not offensive.

June 1, 1919: Turpentine enema resulted in slight yellow stool; another yellow stool, large amount and undigested, with very slight trace of mucus.

June 2, 1919: Temperature 102° F. Child vomiting its broth and water. Small green stool passed after turpentine enema, which did not lessen the distension. Subcutaneous saline. Child died.

Post-mortem Report by Dr. W. P. Hillier.—Abdomen distended. Peritoneal cavity contained 6 oz. or 8 oz. of yellow fluid, and the intestines were loosely matted together by plastic lymph. Umbilicus soundly healed. The umbilical vein (round ligament) runs through a tunnel in the liver substance. When the liver is cut into, the part of the vein within the liver is found to be greatly enlarged and to have thick walls so that it measures about 1/2 in. in diameter. The lumen, however,
is almost obliterated, but some greenish pus is exuded from it. Gram-bacillus obtained from pus. All the organs in the thorax were healthy, as were also the kidneys, stomach and intestines. The hepatic flexure of the large intestine was situated however beneath the neck of the gall-bladder, the ascending colon lay close along the right side of the spinal column, the cæcum was situated over the right ilio-sacral articulation, and the appendix lay across the fifth lumbar vertebra. The spleen was rather firmer in consistence than usual. The adrenals were rather large and the right one of reddish hue.

**DISCUSSION.**

Dr. Pritchard: Have other members of this Section noticed any difference in the forms which so-called infantile scurvy takes under different regimes of feeding? I have noticed that children who are fed on boiled milk are more liable to simple haemorrhage conditions, whereas those fed on patent foods, containing a large excess of starch with probable absence of antineuritic vitamins, develop more the pseudo-paretic type with œdema, such as one sees in beri-beri and pellagra. I cannot help thinking that, now these accessory factors of food are being studied, we shall find clinical distinctions between the different forms. We may find mixed forms, also forms in which one or other kind predominates. This child was fed on milk, and had no antiscorbutic vitamin, as far as I know. There was an extraordinary condition of haemorrhage, superficial and deep. The specimen was brought because the degree of haemorrhage into the suprarenals and kidneys seems to me remarkable.

Mr. W. T. Hillier: In the first case, that of purpura, the only blood film obtained, during life, was a very poor one. One can only say that there was a great excess of leucocytes—no less than 300,000 per cubic millimetre. With regard to the case of liver abscess, the organism which I have obtained from the pus is a Gram-negative bacillus probably of the *Bacillus coli* group. The round ligament before it reaches the liver is solid, but within the liver it is much enlarged, and has a thick wall and lining, with a definite lumen from which pus escapes.

Dr. Langmead: With regard to the first specimen, it would be well if we could have a histological section made of the kidney. Its appearance is that of the kidney of acute lymphatic leukaemia. Histological examination would prove it absolutely, and it would show that the kidney was not only haemorrhagic, but engorged with lymphocytes.
Specimens from Case of Aplastic Anæmia.

By J. PORTER PARKINSON, M.D.

The section of the right kidney showed increase of the cells in the glomeruli: the epithelium of the convoluted tubules and ascending loop of Henle is swollen, degenerated, and in many places desquamated. The lumen of the tubules is filled with desquamated epithelial cells and granular débris. There is dilatation of the ascending loop of Henle and of the collecting tubules. This suggests chronic parenchymatous nephritis. The spleen was small and tough, with thickening of the fibrous capsule and increase of the reticulum. There is also increase of the fixed tissue cells with reduction in the number of lymphocytes and complete disappearance of the Malpighian bodies.

For the above I am indebted to Dr. Sanguinetti's report. The complete absence of albuminuria is interesting in connexion with the above changes in the kidney.

It seems probable that the poison of scarlet fever was responsible for the nephritis as well as for the loss of function of the red bone marrow.

The right kidney was large and very pale, the left kidney looked normal. A piece was removed from the right kidney, and after hardening sections were prepared from it. The section shows an increased cellularity of the glomeruli in which a few leucocytes are seen.
Clinical Section.

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Clinical Section.

President—Sir HUMPHRY ROLLESTON, K.C.B., M.D.

Case of Thoracic Actinomycosis.

By V. Z. COPE, F.R.C.S.

N. T., female, aged 13, said to have had pneumonia in October, 1918. She first attended St. Mary's Hospital in November following. At that time the right front of the thorax bulged forward from the second to fourth ribs. There was a discharging sinus outside the right nipple, and a fluctuating area near the sternum. The right front of the chest was dull on percussion over the involved area. X-rays show dense shadow over the right lung base. Present condition: Several partially healed sinuses; dullness much the same. An actinomycotic granule was obtained from the discharge and showed Gram-positive filaments branching in a dichotomous manner and arranged radially. Treatment by potassium iodide has led to some improvement. (See skiagram on p. 2.)

DISCUSSION.

Mr. KELLOCK: I have seen several cases of streptothrix infection of the thoracic wall and have always thought the primary seat of the disease was in the lung, there is always, as in this case, a history of some previous lung trouble, such as bronchitis or pneumonia, and one knows what a disposition the streptothrix organism has to produce adhesions and wander out from the organ primarily affected. I do not think it is the base of the lung that is always the affected part, for I have seen the thoracic wall infected as high up as the second intercostal space: this disposition to leave the original seat and invade surrounding tissues is often seen in the case of infection of the submaxillary or parotid salivary glands: these, I think, are infected from the mouth by way of their ducts, the affection of the gland itself may be slight, but the invasion of the tissues of the face or neck often extensive. When affecting the chest

1 At a meeting of the Section, held April 11, 1919.
Thoracic actinomycosis.
wall the condition is often supposed to be tuberculous and treated as such, but I agree that a tuberculous infection of the chest wall is very rarely seen dependent on pulmonary disease. As to the possibility of a thoracic wall infection being consequent on disease in the abdomen, liver, or appendix, this is not likely to be the case. When the appendix and liver are both affected it is more likely to be a simultaneous infection rather than a consequent one, for I have seen a case in which the appendix, the liver, and the pancreas were all affected, which seems to point to an infection from the intestinal tract of all three. With regard to treatment, of course iodide of potassium in very large doses has always been regarded as the routine drug, but it is disappointing as regards a cure; it has always seemed to me to have more effect in pure cases, and to be of comparatively little use when, as is so often the case, a secondary septic infection has been added. It is right to scrape or cut away the diseased parts when possible, but this is not often the case, and then we have to rely on constitutional treatment. I think I have obtained the best results from salvarsan; in one case of extensive infection of the anterior mediastinum which had involved the sternum and the thoracic and abdominal walls it did very great good, the disease was distinctly arrested, and the patient’s general condition very much improved. I should recommend Mr. Cope to give it a trial in his case, for I do not think any surgical interference here would be of any benefit.

Dr. F. Parkes Weber: Have any good results been obtained by X-ray treatment (in combination with the use of potassium iodide internally) in cases of thoracic actinomycosis? Mr. Kellock’s remarks on his favourable results from the use of salvarsan in cases of actinomycosis with septic counter-infections are extremely important. In septic conditions and in cases of septicaemia intravenous injections of salvarsan, when tried, have mostly not given good results. Salvarsan has been employed in cases of puerperal fever, for instance, without, I believe, doing the patient any good. In tuberculous cases trials of salvarsan treatment have proved most disappointing.

Mr. Cope (in reply): Benefit from exposure to X-rays has resulted in some cases, but the effect is very uncertain, and that treatment has not been tried in this case.

Sarcoma of Scapula treated by Radium.

By T. H. Kellock, M.C., F.R.C.S.

The patient, a carman by occupation, aged 46, was admitted to the Middlesex Hospital on January 8, 1917. About eleven months previously he had been kicked by a horse on the right shoulder; a bruise appeared, but this soon got well. About seven weeks before admission
he noticed that the right shoulder was swollen, but there was very little pain. The swelling gradually increased in size until, on admission, it had assumed the dimensions shown in the photographs. The tumour was very hard and fixed, and extended some little distance down the arm; there were several very large veins in the skin covering it. The movements of the arm and shoulder were much restricted, but not painful.

On January 12, 1917, 337·3 mgm. of radium were inserted into the tumour, and a small piece removed for microscopic examination. The

![Fig. 1.](image)

Taken before treatment (the parts had been painted with iodine previous to the insertion of the radium tubes).

report made on this was "round-celled sarcoma" (a section was shown). A little sloughing occurred where the strongest tube of radium (90 mgm.) had been inserted.

On February 9, 1917, the tumour had distinctly diminished in size. On this date the radium treatment was repeated, 322·3 mgm. of radium
being inserted for eighteen hours. After this the tumour steadily decreased in size and eventually disappeared.

It is now over two years since the treatment was carried out, and it will be seen that there is no recurrence; all that is to be seen now is some irregularity of the spine of the scapula and the scars where the radium tubes were inserted. The movements of the arm and shoulder are free, and the patient has been at active work ever since his discharge from the hospital on February 19, 1917.

**Fig. 2.**

Taken before treatment (the parts had been painted with iodine previous to the insertion of the radium tubes).

**DISCUSSION.**

**Dr. F. Parkes Weber:** In Mr. Kellock's experience are the tumours in which the cells are most "lymphoid" in character (that is to say, most nearly resemble so-called "lymphocytomata" and leukæmic nodules) those which have yielded the best results under radium treatment? It is notorious that excellent results have occasionally been obtained by X-ray treatment in
mediastinal new-growth (presumably sarcomatous or lymphosarcomatous in nature).^1

Mr. Kellock (in reply): It is certainly my experience that the growths of which the cells are of the lymphoid type are the most amenable to treatment by radium.

Fig. 3.

Skigram taken before treatment.

^1 Some may, however, have in reality been leukæmic in nature—of Sternberg’s “leucosarcomatosis” group.
Fig. 4.

Taken two years after treatment.
Fig. 5.

Taken two years after treatment.
Fig. 6.

Skiagram taken two years after treatment.
A Model to demonstrate the Methods carried out in the Mobilization Treatment of Knee-joints.

By J. Everidge, O.B.E., F.R.C.S.

(.4)—This shows the arrangement for those cases which are running an aseptic course after operation. It demonstrates a method whereby an increasing range of passive movement can be obtained, and is essentially for the prevention of the formation of adhesions. This method keeps the daily range of movement absolutely under control and eliminates the risk of the tearing apart of structures, sutured at the primary

![Fig. 1.](image)

Private M., fourteen days after a piece of shell was removed from the knee-joint. The joint at operation was opened, washed out, and closed. This patient walked well three weeks after the receipt of injury.
"toilet" operation. When firm union of these structures has occurred, active movement can easily be arranged for on this apparatus.

Note.—The illustration of the apparatus is not here reproduced; it may be seen on reference to the British Medical Journal, 1918, ii, p. 183.

Figs. 1, 2, 3 are illustrations of cases taken from a series of over a hundred treated by this method.

Fig. 2.

Fig. 3.
Penetrating knee cases.
Condition two and a half months after injury.
(B)—The arrangement used in connexion with the mobilization treatment of suppurating knee-joints. The limb is placed on the hinged back-splint with footpiece (a modified McIntyre). This splint is partially counterpoised, and is fitted with running-wheels beneath the

![Fig. 4.1](image)

"Active movement" splint. It can be made rigid by fixation of the adjuster below the knee. Note the adjustable footpiece supported by elastic springs.

1 Figs. 4-9 (pp. 12-15) are reproduced by permission of Messrs. John Wright.
footpiece (figs. 4-6). In this way active movements are carried out with optimum ease, and the patient's feeble strength is not wasted in the purposeless effort of raising the leg from the bed, since the force of gravity is neutralized by the counterpoising system. Active contraction of the leg muscles is arranged for; the hinged footpiece, held up by elastic supports, making this easy. Figs. 7, a, b, p. 14, illustrate a case treated in this way. For further details as to this method refer to the British Journal of Surgery. April. 1919, pp. 566-78.

Fig. 6.

Diagram of apparatus. The splint supporting the leg is partially counterpoised by the conical sand reservoirs A and B. The shape of the reservoirs enables an easy adjustment of the weight of sand, for, by means of a tube of outlet controlled by a tap in each, sand can be allowed to flow from one to the other. C is not a counterweight; it is a fixture, and acts as a cistern for the supply of any additional sand necessary to A, or through A to B. D is for waste sand (excess from B).
Fig. 7a.

Fig. 7b.

Demonstrating the apparatus in use.
Fig. 8.
Case 14, one month after shell wound of the knee which produced a septic arthritis.

Fig. 9.
The same case, showing that at this time a complete range of movement was possible.
Case of Acute Suppurative Arthritis of the Right Knee treated by the Method described in preceding paper.

By J. Everidge, O.B.E., F.R.C.S.

Private F. J. Shell wound of right knee on October 25, 1918. Piece of shell lodged in joint. External condyle of femur badly fractured. At operation, part of the condyle lying loose was removed. The internal semilunar cartilage was also removed. The joint was closed partly by suturing of the synovial membrane, partly by a muscle flap. Staphylococcal suppuration followed. The pathologist's report on the fluid removed from the joint was: Pus cells too numerous to count; cocci in every field; culture—staphylococci. The joint was at once opened up and active movements were commenced.

For ten days the temperature ranged at about 102° F. nightly, and the pulse 100 to 110. By the tenth day the joint discharge, which had been thick and yellow, was becoming clearer and resembling normal synovia. The pathological report on this fluid was: In fifty fields—polymorphs, 44; lymphocytes, 27; cocci, 3.

Progress from the tenth to sixteenth day was good, and the joint was obviously settling. During the following week a collection of pus in the popliteal space called for incision and drainage. Movements were carried on continuously.

The patient commenced walking in two and a half months from the date of his original wound. Now, five months after the original injury, all the wounds are soundly healed, he is able to flex his knee through a range of 60°, and walks well, being able to go daily from Kensal Rise to Fleet Street, in pursuit of his civil occupation.

Twenty-three cases of suppurative arthritis of the knee have been treated in this way, and eleven of these have good mobile knee-joints. Amputation was carried out in one case in the series.
Clinical Section.

President—Sir Humphry Rolleston, K.C.B., M.D.

Case of Lymphadenoma: Arsenical Pigmentation.¹

By Eliz. O'Flynn.

Patient, a boy, aged 10.

Past history: Attended Tite Street Hospital in 1916 for enlarged glands of neck. Admitted to the same hospital 1917. The glands were thought to be tuberculous—until an operation was performed and one of the glands microscoped. The changes in the gland consisted of an increase of fibrous stroma, diminution of lymphoid tissue and presence of large lymphadenoma cells. The only other illnesses from which he had suffered were herpes zoster and chicken-pox. During his stay in hospital arsenic was administered by the mouth (Fowler’s solution), in gradually increasing doses, from 6 minims to 30 minims daily. He became intolerant to arsenic; this was shown by gastric symptoms—i.e., nausea. Smaller doses were again tried and were tolerated. Total arsenic in three weeks, 5½ dr. Pigmentation was noticed at the end of patient’s stay in hospital. The old herpetic spots, however, remained unpigmented. In addition to arsenic the patient was treated by X-ray exposures and intramine. A slight temporary improvement ensued.

Subsequent history: The patient was admitted to St. George’s Hospital in February, 1919, with pneumonia. He was extremely ill for one week. The crisis occurred on the eighth day, and he then made an uninterrupted recovery. During this period no alteration took place in the glands. For about three to four weeks after the pneumonia

¹ At a meeting of the Section, held May 9, 1918.
there seemed to be some diminution in the size of the neck; measurements varying between 39½ and 40½ centimetres. There has been slight pyrexia varying between 99° and 100° F. The treatment has since consisted of the internal administration of phosphorus and benzene, and at present he is receiving intravenous injections of arsenic (novarsenobillon). Blood count: Reds, 4,704,000 per cubic millimetre; whites, 9,440,000 per cubic millimetre; haemoglobin, 65 per cent.; polymorphs, 57 per cent.; lymphocytes, 36 per cent.; mononuclears, 6 per cent.; eosinophils, 1 per cent. The general condition of the patient is better. Arsenical pigmentation as on admission.

The President: Dr. Parkes Weber made a collection of cases suggesting that pneumonia was prone to occur in persons taking arsenic but that such cases of pneumonia ran a favourable course. I am inclined to think that the evidence of acute pneumonia, when it occurs, may be connected with the presence of lymphadenomatous glands at the roots of the lungs, apart from the use of arsenic. The improvement in the condition of the lymphadenomatous manifestations thought to occur in this case raises the question of the curative effect sometimes exerted by one disease on another; for example, the beneficial effect of erysipelas on carcinoma of the mamma and the effect of Coley's fluid, composed of the mixed toxins of streptococci and Bacillus prodigiosus, on sarcoma. Dr. Harry Campbell¹ has recorded a case of malignant disease of the liver in which intercurrent influenza was followed by apparent disappearance of the hepatic disease, but eighteen months later the signs of malignant disease of the liver returned and the patient died. The association of abdominal pigmentation with leucoderma, possibly due to herpes zoster, in Dr. O'Flynn's case, at first sight, might suggest that the pigmentation and the herpes were both due to the arsenic, as was abundantly demonstrated during the epidemic outbreak of arsenical poisoning occurring among beer drinkers in the North of England and the Midlands in 1900. In his account of this outbreak, Dr. Reynolds,² who was first led to suspect its arsenical origin by the occurrence of herpes zoster, found that the pigmentation often showed well marked lighter spots like rain drops. In the present instance the history given by the boy's mother is very vague, but it appears probable that if he had herpes it was before the course of arsenic.

Case of Tuberculous Lymphangitis following Injury to a Tuberculous Wart of Long Standing; Complete Excision in continuity of Primary Focus, Infected Vessels and Glands.

By W. Sampson Handley, M.S.

E. M., aged 47, a piano maker, for many years had had two small warts on the knuckle of the left index finger. A sister had died of tuberculosis. In April, 1916, he accidentally cut one of the warts with a saw. Slight inflammation followed and a few weeks later he noticed a small firm swelling under the skin, situated on the dorsal aspect of the lower end of the radius. In January, 1917, he attended Dr. Pringle's out-patient clinic. Tuberculosis of the skin was diagnosed and he received X-ray treatment, but further swellings appeared up the arm and Dr. Pringle advised surgical treatment. He was admitted under my care on April 20, 1917.

State on admission: A patch of dry warty lupus, not ulcerated, and about an inch in diameter, was present on the knuckle of the left index. In a line between this lesion and the axilla were numerous nodular swellings (see figure). On the flexor surface of the forearm were several small subcutaneous nodules, covered by normal skin to which they were not adherent. At the junction of the middle and upper thirds of the forearm was a larger fluctuant swelling, \( \frac{1}{2} \) in. in diameter, covered by adherent, reddened and thinned skin. Just above the elbow, in the situation of the bicipital gland was another swelling, also softening. Several small nodules were present along the inner side of the upper arm, connected by vague cord-like subcutaneous thickenings. The whole formed a chain of nodules from the primary lesion to the axillary glands, which were much enlarged, hard and not tender.

Operation, April 26, 1917: An incision was made in the axilla and the enlarged glands were dissected out without division of the trunks reaching them from below. The incision was then prolonged downwards along the whole length of the arm to the primary lesion, bifurcating where necessary to encircle the larger nodules and the patch of lupus. The nodules, the connecting lymphatic channels and
Tuberculous lymphangitis following injury to a tuberculous wart of long standing.
the primary focus were then dissected out in one strip continuous above with the axillary glands. Included in the strip were the infected areas of adherent skin over the larger nodules. The wound, which measured 22 in. was then sutured. It healed by primary union. The patient returned to work as a munitioner.

On June 2, 1917, he was re-admitted with a small abscess over the middle of the biceps on the inner side of the arm. It was completely excised together with the infected skin over it. The wound healed by first intention, and he left the hospital on June 11.

In April, 1918, he noticed a warty growth at the side of the original lesion. He was treated by ointments but the condition grew worse. He continued at work, but the warty patch spread, and in April, 1919, he came again to see me. He had a patch of dry warty lupus, 2 in. in diameter, at the site of the original lesion. One or two subcutaneous nodules not adherent to the skin, could be felt on the front of the forearm. The lupoid patch was excised and the area grafted from the thigh. The nodules in the forearm were excised. His general health has continued excellent throughout.

On section the recurrent lupoid patch presented warty hypertrophy of the skin, with a tendency to downgrowth of the epithelial cell columns. The papillary region and the superficial layer of the dermis presented diffuse round-celled infiltration and numerous dilated lymphatic and blood-vessels were seen. The lymphatic vessels in places showed partial obliteration of the lumen by proliferation of the endothelium. A few typical giant-cell systems were present.
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Section of Dermatology.

President—Sir James Galloway, K.B.E., C.B.

Urticaria Pigmentosa.

By J. L. Bunch, M.D.

The patient, a boy, aged 6, has numerous brownish patches on the trunk, limbs and neck. Some are as large as a two-shilling piece, but the majority are smaller. The patches extend on to the neck, as far as the angle of the jaw, but the face is quite free. The lesions are not raised, unless they are irritated, when they come up as wheals, and well-marked factitious urticaria is present on any part of the skin. The patches appeared shortly after birth.

DISCUSSION.

The President: There can be little doubt about the diagnosis, and Dr. Bunch need hardly have a Wassermann reaction done. As a rule, the pigmentation is not as heavy as it is in this case, but if Dr. Bunch will look up the record of a case from Charing Cross Hospital which I published, which was worked out by my then clinical assistant, Dr. Brongersma, of the Hague, he will find that that case was spotted, with contrast of colour almost like a leopard. Sometimes the pigmentation in these cases is very extensive. In the number of the Journal of Dermatology referred to, there are illustrations of this, also from the same patient in the New Sydenham Society's Atlas. This was also one of those more unusual cases which commence at about the age of 17 or 18. Most of the cases commence earlier than that, but there is an interesting group in which the commencement occurs at from 17 to 22 years of age. Can Dr. Adamson tell us of any recent advances on the aetiology, or does the matter remain in this respect as Dr. Sangster, with Mr. Nettleship, described it years ago?

1 At a meeting of the Section, held October 17, 1918.
Epithelioma of Face.

By J. L. Bunch, M.D.

The man, aged 85, has a tumour about 2 in. long by 1 in. wide on the nose below the left orbit. It very closely approaches the eye. It has been present twelve months and has increased in size since he was injured in a carriage accident six months ago. It is slightly ulcerating; no glands can be felt. He has been X-rayed without benefit, and the question of operation now arises; or possibly the use of radium or diathermy.

DISCUSSION.

Dr. H. G. Adamson: I should hesitate about doing anything to it. X-ray treatment of large epitheliomata of the skin may be dangerous. It is seldom one can destroy the disease completely. Anything short of complete destruction only leads to more active growth. If X-rays were used in this case massive doses should be given, either six pastille doses of unfiltered rays at one sitting, or two to three pastille doses measured through 3, mm. of aluminium and repeated weekly for three weeks. With these massive doses there are likely to be symptoms from absorption of the destroyed tissues, which can be avoided by scraping away the growth before applying the X-rays. Diathermy might be used in this case. Dr. Cumberbatch has described this method and its results in malignant growths at a recent meeting of the Section of Electro-Therapeutics.¹

Dr. Graham Little: I strongly urge the trial of radium in this case, for even if the ulcer be not cured by its application it will probably at least heal over and so promote the comfort of the patient. I have recently had an instance of the value of radium in just such a case as this. It was a large rodent, involving the eyelid and cheek, in which remarkable improvement was maintained with radium. This was applied at the Radium Institute, the dosage being, I believe, 80 mgr., unscreened, for periods of one and a half hours at a sitting, repeated about every six weeks.

Dr. J. H. Stowers: I have had two cases, perhaps not so advanced as this, under my observation during the last eighteen months and in each of them radium has produced really satisfactory results. I sent them to the Radium Institute, where Dr. Lynham took charge of them and directed the treatment. Having regard to the patient's age, and the extent of the disease, I consider treatment by radium is the most suitable in this case.

¹ Proceedings, 1918, xi (Sect. Electro-Therap.), p. 29.
The President: Recently I saw, in private, a case of large epithelioma of this kind, though not in such a troublesome situation: it was on the forehead, and involved the scalp. It was surprising how it could have been allowed to become so extensive, as it was the size of the palm of my hand. I was then asked the question which is now being asked about this case: What is to be done? The patient was a gentleman aged 78, and I decided that it was wisest to leave it alone, as a complete operation was unthinkable. I agree with Dr. Adamson that to attempt to destroy the growth by means of the X-ray is out of the question: in many cases fungation is accelerated by the X-ray treatment. After massive doses I have seen patients suffering from toxæmia, if not from septicæmia. Therefore it is hopeful to hear from those with experience that they would give this patient the opportunity of radium treatment. A surgical operation in this case would, I think, determine the duration of the case with dispatch.

Dr. Bunch (in reply): When I was in Paris, at the clinic of a well-known surgeon there I examined some cases of well-marked epithelioma which he claimed had been absolutely cured by diathermy: he treated one or two during my stay. One was far back in the throat, and he said it had been septic. He had applied diathermy once before, and when I saw the case there was little more than a scar. He treated the patient again while I was there, and promised a cure. It was in the mucous membrane, and so I believe a surgeon would have been shy of tackling it. I shall have this patient treated by diathermy.

Guttate Morphæa.

By S. E. Dore, M.D.

This woman, aged 61, I saw for the first time yesterday, and diagnosed the condition as the guttate type of morphæa, so-called "white-spot disease." I showed a case here some time ago, which was also seen by Dr. Bunch, who has shown similar cases, and who agreed with the conclusions arrived at. I thought in regard to that case one could recognize three stages—namely, first the ivory or chalk-like patches, then a scaly stage, and thirdly, a stage of atrophy. These cases have been confused with lichen planus atrophicus and with macular atrophy. In the present case there is also definite scaling on the white patches, and I take these scales to represent an intermediate scaly stage, followed by atrophy, which is also shown in some patches on the shoulders. My belief in the diagnosis of guttate morphæa has been however somewhat shaken by Dr. Pringle's opinion that it is a case of
lichen planus. In favour of that diagnosis there is a history that she has had some erythematous circinate lesions on the legs and thighs some time ago, in fact there are some erythematous rings, in a faded condition, on the legs at the present time. It is worth while discussing whether white-spot disease is, properly, a morphea, or whether it is a lichen planus, and whether some cases of macular atrophy represent a terminal stage of one or the other. The lesions are situated in the typical position for white-spot disease, on the neck and shoulders, and the mucous membranes are not affected. The condition is of four years' duration.

DISCUSSION.

Dr. J. L. Bunch: I have come across one or two such cases from time to time, and I have published some cases, which Stelwagon quotes as typical cases of white-spot disease. The literature seems to be involved. Apparently it includes many atrophic diseases, apart from guttate morphea. Stelwagon would limit it to morphea guttata. My cases, which seem to have been regarded as typical, had no scaliness at any time. In one, there was slight erythema round recent patches. They went on to atrophy, and remained atrophic the whole time. I believe the present case is not one of the condition which would now be looked upon as morphea guttata, but it may be lichen planus atrophicus. I suppose sections have not yet been cut.

Dr. Graham Little: I believe this to be a case of lichen planus atrophicus, and this was my diagnosis before I had heard Dr. Pringle's opinion, which gives me greatly more assurance in making it. The case is remarkably like one shown by Dr. G. W. Sequeira, about which the same difference of opinion was expressed, and in which Dr. Stowers and I held the view that it was lichen planus. With regard to the name "white-spot disease," the American authors, who are the chief offenders in inventing and using it, expressly include under this heading diseases other than morphea, and I think it would be useful to discard the name, as tending to confusion.

Dr. H. G. Adamson: I think this case is morphea guttata. There is no evidence of lichen planus papules. It is not unusual for scleroderma to become scaly in that way. One may often see scaliness or plugging of the follicles at the central part of a patch of scleroderma.

Dr. Bolam: I suggest that this patient may be the subject of tubercle, as her condition is poor and she only weighs 6 st. It may possibly be an example of fibroid phthisis. With regard to the skin lesion, I think that the white patches are atrophic areas following on tuberculide. They have not the feeling one is accustomed to associate with morphea guttata.
Section of Dermatology

Dr. J. H. Stowers: Can Dr. Dore and Dr. Adamson wholly exclude lichen planus, seeing that the patient has had circinate patches upon the extremities associated with itching? This does not correspond with the condition spoken of as white-spot disease. It is probable that there are manifestations of disturbed nervous function. I regard this case as probably an instance of lichen planus atrophicus.

The President: The diagnosis would be helped very much if we could get a complete explanation of those definite finely scaly papules on the back. We are familiar with fine scaleiness in morphea; there is a fine scaling in badly-nourished conditions of the skin, even stretching may produce it on a thinned epidermis. The patches on the back and scapulae of this patient are definite small conical elevations, and they may simply be plugged follicles which have become inflamed, or there may be something more definitely inflammatory, or even granulomatous, and I suggest to Dr. Dore that a microscopic investigation of the skin including a papule would help the diagnosis. My inclination is to regard this as an example of the "guttate morphea" type rather than as a tuberculide of the lichen scrofulosorum type, or as atrophic lichen planus. Perhaps Dr. Dore will be able to tell us more about it later.

Dr. Dore (in reply): These chalky or mother-of-pearl lesions are to my mind, clinically at any rate, quite characteristic. In the cases of the condition I have seen, there have never been typical lichen planus papules, nor appearances similar to this in cases of atrophic lichen planus. I still therefore adhere to the diagnosis of morphea guttata.

Case of Multiple Idiopathic Hæmorrhagic Sarcoma of Kaposi.

By E. G. Graham Little, M.D.

The patient is a young woman, aged 22, the mother of one child who is now aged 6 and is perfectly well. The patient comes of pure Irish stock, traceable for at least three generations, and denies any Jewish ancestry. The condition began about the age of 8; the distribution as now seen seems to have been completed in about a year from the commencement of the disorder, and to have remained practically in statu quo. There is some pain complained of in the foot, but no other subjective sensations whatever, and she suffered no inconvenience until a few weeks ago, when she sustained an abrasion of the patch on the dorsum of the foot.

The disorder consists in a deep pigmentation the colour of "fumed oak," in three areas distributed as follows: There is a patch 2 in. long
by 1 in. broad over the dorsum of the right foot, just anterior to the
external malleolus: there is another and smaller patch about 1½ in. by
1 in. behind and a little above the external malleolus. In these areas
the pigmentation is a continuous sheet: the pigmented patch is scarcely
raised above the level of the skin, but the thickening is slightly percept-
able when the patch is palpated between finger and thumb. Over the
middle of the outer aspect of the right leg there is an area of about 4 in.
by 2 in., consisting partly of small patches of pigmentation and a large
number of discrete pigmented flat papules about ½ in. in diameter,
the colour of the small papules being somewhat lighter than the patches.
There has never been any tendency to ulceration.

Kaposi's idiopathic haemorrhagic sarcoma. The ulceration on the front of the
lower part of the leg is due to a biopsy.

In every other respect the patient is a well built healthy woman.
It is hoped that it will be possible to obtain a section of the skin for
later histological report.

Dr. Pringle has pointed out to me how closely this case resembles
that of the patient he showed here and reported in the July number
of the Proceedings.¹ The sex of my patient and the definitely non-
Jewish ancestry are points which add to the rarity and interest of
the case.

¹ Proceedings, 1918, xi (Sect. Derm.), p. 107.
DISCUSSION.

Dr. F. Parkes Weber: I agree with Dr. Little's diagnosis. Not long ago I referred to a case in which typical idiopathic multiple haemorrhagic sarcoma (Kaposi) seemed to commence about a naevus, and if the disease in Dr. Little's case had been congenital, one would have regarded it as a condition of vascular naevus. Very few cases of the disease have been recorded in which there was no Hebrew ancestry. One non-Hebrew case, in which the late Sir Jonathan Hutchinson was interested, died at Abingdon, near Oxford. I would like to emphasize the fact that the disease may commence at a quite early age, and that it may probably originate from a lesion which has every appearance of being a vascular pigmented (congenital) naevus. Almost all cases of the disease occur in Jews, the proportion of non-Hebrews being, probably, not more than 1 per cent.

Mr. H. C. Samuel: Is the fact that the condition did not appear until the patient was 8 years old a necessary and absolute proof that it is not a naevus? Many conditions which are congenital do not manifest themselves until later in life.

Dr. Graham Little (in reply): I went carefully into the question of the possibility of preceding lesions in this case, and the patient emphatically confirms the history I have given. I agree with what Mr. Samuel said as to naevus of late development, but I do not think this is a naevus.

Case of Circinate Persistent Erythema Multiforme.

By E. G. Graham Little, M.D.

The patient is a bookstall clerk, aged 34. The eruption began on his legs and arms in March, 1918, as small "pimples" which itched. He describes two varieties of lesion, a patchy amorphous erythema which itches moderately, and leaves a slight degree of pigmentation, and a ringed lesion, which smart but does not itch, and leaves either no discoloration or else a faint buff tint, which finally disappears. A few ill-formed erythematous patches such as he describes are to be seen, also numerous pigmented areas the site of previous patches, on the trunk and arms. But by far the greater part of the present eruption is made up of vivid erythematous rings with raised oedematous but not vesicating edge, enclosing spaces of the size of a shilling to a five-shilling

piece, or the ring may be broken in some part, thus presenting semi-
circular figures and portions of circles. The rings are extremely
numerous, and cover nearly the whole limb from the buttock to the
ankle on both sides. There are a few similar rings on the lower part
of the back and on the forearms and shoulders. The individual rings
enlarge from the initial papule, the evolution taking some six or eight
weeks in the case of the largest rings, such as those on the lower legs.

The man came to my department in August when I was away on
holiday, and my deputy describes much the same distribution as now.
I saw the patient in the first week of September, when there was no
sign of any skin disorder beyond the pigmentation, which still persists
on the trunk. The eruption seems to have come out again quite acutely
a few days after his visit to me. His health is otherwise good, and
he gives a negative Wassermann reaction. The diagnosis in my opinion
lies between three alternatives, of which I provisionally accept the
first: (1) Erythema multiforme perstans; (2) dermatitis herpeti-
formis “en cocarde”; (3) “erythème annulaire centrifuge” of Darier.

I believe I reported the first case of a rare affection which has since
been classed under the name “erythema multiforme perstans.” The
case which initiated the discussion of this type was shown by me first at
this section in 1912,1 and at the International Congress in 1913, where
very varied opinions were expressed as to its nature. This patient was
under my observation for several years, and the appearances of the
rings, the persistence of individual rings for several weeks or months,
and the slight buff pigmentation which is seen inside the rings strongly
recall this case. Further instances of the same type have been
shown here by Gray (1913), MacCormac (1915), and recently by
Stowers (1918). The presence of at least one or two vesicles—though
isolated and very different from the grouped vesicles of dermatitis
herpetiformis—and my experience of a case which I showed here as a
possible “erythème annulaire centrifuge,” which subsequently proved to
be an unmistakable dermatitis herpetiformis, make the diagnosis of
dermatitis herpetiformis possible. This however is largely discounted
by the moderate degree of itching. Finally the centrifugal spread, the
commencement of the ring with a rose spot, the comparative absence of
itching, the distribution of maximal intensity on the buttocks and
thighs, suggest the type named by Darier “erythème annulaire
centrifuge,” of which I have reported what is, I believe, a genuine
and so far unique case in this country.

DISCUSSION.

Mr. H. C. Samuel: What is much more important than the actual diagnosis is the aetiology of the condition. There seems to be some underlying toxaemia, and the patient gives a distinct history of post-nasal catarrh, and says he is constantly swallowing mucus and pus.

The President: As regards diagnosis, one point to which I drew Dr. Little's attention was that the patient complains of some irritation. Perhaps he is exaggerating, but the irritation is enough to keep him awake at night. And the odd thing about this irritation is that it is at the small point which he indicated on the upper part of the right thigh where there is a papular vesicle or vesicles. When these commence to appear, he begins to suffer pruriginous irritation. That makes one think of an early or a later condition of dermatitis herpetiformis. It is too soon to be sure of the diagnosis, but I am inclined to consider that it is early dermatitis herpetiformis, rather than erythema perstans or "erythème centrifuge."

Report on a Case of Tuberculosis Cutis of Six Years’ Duration in the person of a Male Patient, aged 53 (exhibited at the meeting held on July 18, 1918), confirming the diagnosis.

Communicated by J. H. Stowers, M.D.

Microscopic examination by Mr. T. W. P. Lawrence, pathologist, University College Hospital:—

(1) Slight hypertrophy of the epithelium and papilla, producing irregularity of the free surface.

(2) Hyaline thickening of the connective tissue fibres of the corium.

(3) Swelling and proliferation of the endothelium of the capillaries and arterioles leading, in places, to complete obstruction of the lumina.

(4) A stratum of closely-packed cells surrounding all the capillaries and arterioles, the cells having well-stained oval nuclei and considerable cytoplasm, indefinite in outline. The nuclei of many of these cells have undergone fragmentation.

(5) Absence of leucocytic exudation.

The lesion is a chronic inflammation, characterized principally by an arteritis and peri-arteritis. The cells surrounding the vessels have some resemblance to the "epithelioid" cells of tuberculous lesions, but there is no caseation and no formation of definite tubercles.

N—2a
Report by Dr. F. H. Teale, bacteriologist, University College Hospital: Guinea-pig inoculated (four weeks) with piece of ground-up lesion. Miliary tubercles in liver and spleen, few in peritoneum. Retro-peritoneal glands caseous; mediastinal glands caseous; tubercle bacilli isolated.

The President: I should be unwilling to throw any shadow of doubt on the expert report obtained and communicated by Dr. Stowers, but you will have noticed the bacteriologist made the suggestive remark that in the retro-peritoneal and other glands of this guinea-pig caseation was noted, and that, as you know, is not the result of recent tubercle. This must be borne in mind in considering whether this is or is not an absolutely conclusive piece of evidence.
Section of Dermatology.

President—Sir James Galloway, K.B.E., C.B.

Case of Melanotic Growths (Carcinomatous).\(^1\)

By George Pernet, M.D.

The patient, a woman aged 48, was first seen by me at the West London Hospital on August 27, 1918, for some growths developing on a black naevus situated about the lobule of the right ear. They are well brought out in the accompanying photograph. As a result of a knock, she thought, some bleeding occurred in front of the lobule three weeks

\(^1\) At a meeting of the Section, held November 21, 1918.
Case of Actinomycosis.

By E. G. GRAHAM LITTLE, M.D.

The patient is a child aged 12, of well-to-do parents, who live in a house in Potters Bar, surrounded by fields in which cattle graze. She was sent to me by Dr. Mercer, who diagnosed actinomycosis; indeed, the clinical features are very characteristic of that disease. The history is that a swelling was first noted about eight weeks ago, on the outside of the right cheek near the angle of the jaw. There was no abrasion inside the mouth. The swelling was practically painless, but the induration was very marked, and when I saw the case about two weeks ago the whole cheek was hard, swollen, and of a dusky red colour, with some characteristic puckerings of the surface in the site of nodular swellings about the middle of the cheek. My colleague, Dr. John Matthews, was able to demonstrate characteristic mycelial threads in tissue removed by operation from the nodular swelling, but attempts to cultivate the organism failed. No other cases have occurred in Dr. Mercer’s experience in the neighbourhood, nor have other children playing with this little girl shown signs of infection. The patient has made considerable progress under treatment with iodide of potassium.

1 See instances in Perrin’s article, “Sarcomes,” La Pratique Dermatologique, iv, p. 226.
Case of Extensive Pigmented Nævi.

By E. G. GRAHAM LITTLE, M.D.

The patient is a female child aged 3 months. She was born with the pigmented nævi now seen, which are most unusually extensive and deeply brown, almost black. The largest area is over the sacrum, where a patch some 6 in. by 3 in. occupies the lower part of the back. There are very numerous smaller but similar patches on the face and limbs. A peculiar feature is the complete absence of any development of hair on the pigmented areas; the somewhat pad-like feel of the deepest coloured patches has suggested the possibility of malignant change. I do not think there is any evidence of this myself, and have advised that no treatment should be given.

Case of Folliculitis Decalvans.

By E. G. GRAHAM LITTLE, M.D.

The patient is an elderly lady who has been under my care for some eighteen months with a slowly advancing cicatricial alopecia affecting the vertex and frontal area for the most part. There has been very little inflammatory redness at any time and practically no suppuration, but on close inspection there is the characteristic peri-follicular excavation, and slight redness. There have been no subjective sensations and the lady is otherwise in excellent health. Careful search was made for the accompanying lichen spinulosus which was described in connexion with this type of disease for the first time by Lassueur and myself and of which Beatty and Dore have since reported other examples. There is no trace of this symptom in this case.

DISCUSSION.

Dr. J. J. PRINGLE: I think Dr. Little’s diagnosis is accurate, and that the case is an example of the “folliculitis decalvans” or épilante, originally described and differentiated by Quinquaud, of St. Louis Hospital, about 1883, of which our knowledge became general after the Paris Congress in 1889. The only feature which seems not to be well developed in the present case is the tiny peri-follicular pustulettes which are so eminently characteristic of the

1 *Proceedings*, 1915, viii, p. 139.
disease in its early phases. But that, naturally, has been controlled by the treatment which has been adopted. In place of the pustulette we have the almost equally typical epidermic circumpilar collarettes at the spreading margin of the disease. These cases are constantly mistaken for lupus erythematosus. I have had three in the last year sent as lupus erythematosus of the scalp, and, from many points of view, I think it well to distinguish them carefully.

Dr. S. E. Dore: Can Dr. Pringle tell us if there is any real difference between Quinquaud’s disease and Brocq’s pseudo-péla? I think the term "folliculitis decalvans" is applied more to the early stages in which there is a definite peri-folliculitis. In pseudo-péla, cicatricial areas with a convex spreading margin are seen, but not the inflammatory collarette to which Dr. Pringle has referred. Are the two conditions distinct or are they stages of the same disease?

Dr. J. J. Pringle (in reply to Dr. Dore): I think Brocq’s "pseudo-péla" is a name descriptive of the last stage of shallow cicatricial condition of the scalp produced by folliculitis decalvans. I should be sorry if Quinquaud’s name should drop out in connexion with this disease, because it was his work which established it as a dermatological entity.

Case of Lichen Obtusus Corneus.

By W. Knowsley Sibley, M.D.

This woman is aged 71, and she has spent thirty-three years of her life at the Cape. She has now been back in England three and a half years. She says that ten years ago she had somewhat similar lesions on her legs and arms, and they disappeared, but that since she has been in England they have come back, in a worse form, and persist. There is a group on the outer side of the left leg, which she has had constantly for nearly four years: there is also a patch of hypertrophic tissue on the outside of the left ankle, a similar group on the right leg, on the outer side of the right thigh, on the inside of both thighs, and there is one papule left on the right forearm. Similar papules have occurred on the back of the hands, but they have completely disappeared after the application of X-rays. She has also one or two isolated papules scattered about her body. Apparently the irritation is, at times, extremely severe, and when I first saw her the areas were covered by scratch marks, and some of the lesions on the legs were bleeding. There are no lesions in the mouth.

With regard to diagnosis, I have suggested that it could be grouped
with the cases of lichen obtusus corneus. The first case of this disease seen in this country I showed before the Section in July, 1916, and the second I exhibited here early in the last Session. But this differs from those cases in many respects. In both the former cases, the papules were isolated and discrete, whereas in the present case many of them are grouped, especially the leg lesions, where the grouping is like that of a herpetic eruption. Notwithstanding that, there is some hypertrophic tissue between the papules. There is also considerable pigmentation left. The most marked typical discrete papule is on the inner side of the upper right thigh, which is the size of a pea, slightly umbilicated, presumably from scratching. The largest is in front of the right patella, which is a connecting link between lichen obtusus corneus and lichen hypertrophicus. Many of the lesions, taken by themselves, are exactly like those of lichen obtusus corneus. I show you a photograph of the left leg.

The symptoms have been considerably relieved by small doses of X-rays, and the patient is now much more comfortable. The Wassermann test is distinctly negative. The case is complicated by pus infections, but I do not think that accounts for the hypertrophic corneous condition of some of the papules.

The pathological report is as follows: "The section of a papule taken from the outer area of the left leg shows a very marked thickening of the stratum corneum, which is infiltrated with horn cells, the stratum granulosum appears to be destroyed, the stratum mucosum is greatly thickened and elongated, dipping a good way down into the dermis proper; there is also a dense cellular infiltration present, and the blood-vessels are dilated. There are no giant cells present."

DISCUSSION.

Dr. J. J. Pringle: I agree with Dr. Sibley's diagnosis of this puzzling case, and think his appreciation of its position in the lichen group is justified. I should not have arrived at any firm diagnosis had it not been for the dome-shaped elementary lesion high up on the right thigh, which corresponds in its characters with those of the very few cases which have been recognized as "lichen obtusus corneus" in this country. The fibrous feel to touch is similar, as is also the colour. One of these came under my observation in private, and the diagnosis was confirmed independently by Dr. Adamson and Dr. A. M. H. Gray. There is also a patch about the shin which corresponds exactly to lichen hypertrophicus. Therefore the view enunciated by the exhibitor that it is "a connecting link" is, I think, an acceptable one. The itching is, evidently, very terrible, as we know it to be in these cases, and we must not be misled by the very large amount of consecutive pyogenic dermatitis which has naturally resulted from scratching of the large area of skin involved over the centre of the leg.

Dr. Graham Little: I should deprecate the diagnosis of lichen obtusus in this case. The widespread pus infection of the lesions, for they are practically all suppurative, is surely very unlike the picture of lichen. The nodule which has been claimed as characteristic by the exhibitor seems to me to be more readily explained as a deep-seated pus infection, such as we see commonly in acne indurata. Africa is the home of inveterate pus infections of this very type. The histological report seems to me to be further directly negative of the diagnosis of lichen planus, for the granular layer is expressly said to have been absent, and this is, on the contrary, greatly accentuated in lichen, especially of the verrucose variety.

Dr. Semen: In a large number of cases in France which we have dealt with in the general hospital under Lieutenant-Colonel MacCormac, we have seen cases in which it was impossible to make a diagnosis between ordinary lichen planus and lichenization due to an infective condition
secondary to parasitic infection. In the absence of facilities for making biopsies, we were unable, in some cases, to come to a conclusion in the matter. As Dr. Little has said, one can get lesions extremely like those described, under ordinary circumstances, as lichen planus, but which are of infective nature, and in some cases there is no means, except the microscope, to settle it. In this present case there is nothing like a typical lichen planus papule. In France, we have been in the habit of looking carefully for mouth lesions, and very commonly we have found them when lichen planus is present. In a case like this one might possibly have found something on the tongue or the side of the mouth. Is Dr. Sibley's experience of treating lichen planus by X-rays satisfactory? I have tried it in numerous cases, without much success.

Dr. S. E. DORE: This case is, I believe, only the third of its kind which has been shown in this country. In the first case shown by Dr. Sibley there was nothing especially typical of ordinary lichen planus, but Dr. Adamson pointed out that it was identical with an American case published in the Journal of Cutaneous Diseases under the name of lichen obtusus corneus. In the present case the lesions on the upper part of the right thigh are identical with those in the case Dr. Sibley showed before. The case is of special interest because, as Dr. Sibley pointed out, it seems to show the transition stage between characteristic lichen planus papules and the obtuse horny lesions. Some of the patches which have been considerably scratched and are surrounded by a good deal of cicatricial tissue resemble those in a patient I showed here two or three years ago, with what I considered to be hypertrophic lichen planus, although it was thought by some to be an artefact. She is still under my care, but I have never been able to identify a lichen planus papule.

Dr. BUNCH: Does Dr. Sibley recollect, in the description of the American cases, if there were any lichen planus papules at any time? When I read the paper, the American cases struck me as very indefinite, and an undoubted connexion with lichen planus seemed very indeterminate. Does Dr. Sibley look upon lichen corneus, as described by American writers, as essentially a subdivision of lichen planus?

Dr. SIBLEY (in reply): No lichen planus papules were described in the American cases. I look upon lichen corneus as a subdivision of lichen planus. In the first case I showed, Dr. Adamson diagnosed it, and he considered there was a lichen planus papule by the side of one of the lesions. In that case there was no lesion in the mouth, nor in the other case I showed. This patient has had considerable pyogenic inflammation in addition. My experience of treatment of lichen planus with X-rays has been very satisfactory, but not so with lichen hypertrophicus. Lichen planus, especially in cases with much irritation, is relieved by one-third pastille doses of X-rays. I have had one or two cases of lichen hypertrophicus in which the treatment has made the condition distinctly worse. This patient was in such a condition as to impel one to do something for her, and the small doses of the rays she has had have made her feel a different being.
The PRESIDENT: With regard to the points to which Dr. Pringle drew attention—namely, the peculiar lesion on the right thigh and that near the ankle on the same side, so far as the general eruption is concerned, I think he would be a very bold man who would say it could not be well accounted for by recurrent chronic pyodermia, except for two points. I think that that slightly horny patch near the ankle is probably lichen hypertrophicus. But it is so complicated by pyogenic dermatitis that it is difficult to make a diagnosis. In such a case one would hardly expect to find lesions in the mouth: I understand this patient has not had any such, and she has none at present. I have not had much experience of the treatment of lichen planus by X-rays, but I will give one example. For several years, when I had charge of the out-patient department at Charing Cross Hospital, I had a patient who came under my observation repeatedly, and I frequently made use of her as an example to students of lichen hypertrophicus affecting the knee region, in front of the patella—a very common position. At Dr. MacLeod’s suggestion X-rays were applied, with a good result. I was able to follow the case afterwards, and learned that the eruption had greatly improved, and that the affected areas were flat and smooth.

Case of Senile Tuberculosis Cutis.

By W. KNOWSLEY SIBLEY, M.D.

This woman is a widow, aged 68. She tells us that, four years ago, a patch appeared on the lower part of the left cheek, and it has since been gradually extending, and now invades practically the whole of the left side of the face and a small part of the ear. Recently, nodules have appeared on the left side of her nose. It is not syphilitic; two Wassermann tests have been done and both were definitely negative. I have not previously seen a case of the condition commencing in a patient as old as this one.

DISCUSSION.

Dr. J. J. PRINGLE: This case illustrates very perfectly a type of senile lupus vulgaris with which I am familiar, and which I believe not to be extremely rare. I would draw attention to the very interesting way in which the ear is involved, as it is in lupus erythematosus and in the lupus vulgaris erythematoides of Leloir. As to the differential diagnosis of this case from one of syphilis, there can be no doubt whatever. Dr. Dore and I have, at the present moment, under our care an old Scotch woman aged 75, in whom the diagnosis of syphilis had been made and acted on before she came under my observation with complete lack of success. The disease began on the forehead in 1912, and was of the same timid type as in the case exhibited. I first saw her in November, 1917, and referred her to Dr. Dore for X-ray treatment. In March of the
present year the patient’s son-in-law wrote to me: “The treatment carried out by Dr. Dore has wrought almost a miracle,” a statement the justification for which I can endorse.

Dr. Graham Little: It is extremely rare to find lupus beginning at ages so advanced as in this case. Of course, one sees lupus at this age not infrequently, but almost always with a history of much earlier commencement. I reported a case beginning at the age of 72, which I believe remains a record. Colcott Fox in an early paper investigated the ages of onset in ninety-six cases and found it only once “between 60 and 70.” Crocker saw it twice commencing at the age of 63, and apparently at no greater age.

Dr. Travers Smith: A case of mine, a woman past 60 years of age, got her finger infected with tuberculosis while attending her husband who was suffering from phthisis. She had lymphangitis, and the condition spread up the arm.

The President: The question of the age at which tuberculosis can be acquired is an interesting one. There is a popular idea that old people are not easily infected with the disease. I recollect a case similar to that mentioned by Dr. Travers Smith. A woman, aged about 60, who had been attending her daughter, the subject of acute pulmonary tuberculosis, came to Charing Cross Hospital. The tip of the woman’s left forefinger was infected. The disease was regarded as serious, and the tip of the finger was amputated. The disease then spread to the middle phalanx of that digit, and another portion of the finger was amputated. Later the disease involved the hand, and the surgeon then suggested that the hand should be amputated. At this time she came under my observation, as she was alarmed by this process of gradual dismemberment. She had lymphangitis, and enlarged glands in the axilla, and at one time I thought she would develop pulmonary tubercle: but after I had had her under observation two or three years, the tubercular lesions healed, the pulmonary lesion quieted down, and the cutaneous lesions were completely cured.

Dr. Agnes Savill: Is it usual for lupus vulgaris which began in youth and remained quiescent in middle age, to become active and spread when the patient is over 70 years of age? I have at present such a case under my care.

Case of (?) Dermatitis Herpetiformis.

By E. G. Graham Little, M.D.

I showed this patient at our last meeting, when members will remember the extraordinary appearance he presented, of rings of erythema closely covering the skin from the pelvis to the ankle. 1

2 Ibid., vi, p. 81.
4 Proceedings, p. 7.
sent in a note of the case with the provisional diagnosis of "erythème centrifuge" of Darier, but with a reservation that it might prove to be dermatitis herpetiformis. I believe the latter diagnosis is more probable. I put the patient on arsenic after the last meeting, and when I saw him a week after, the eruption seemed to be aggravated, so that I stopped this drug. I have seen him again to-day after three weeks' interval. He has now a very pruritic vesicular and papular eruption, the distribution of which, especially on the axillae and penis, is suggestive of an intercurrent attack of scabies, and the rings have wholly disappeared. He also gives a history that his wife has recently developed an itchy eruption. It is therefore very probable that scabies explains a part of the present eruption, but I am sure that it is only a small part.

DISCUSSION.

The President: If I did not feel so confident in Dr. Little's diagnosis in spite of the fact that he told me that the characteristic feature of the eruption was a "ringed bulla," I should have felt inclined to diagnose scabies. But I remember the case at the previous meeting, and I remember those ringed eruptions and I think Dr. Little is right when he suggests that the scabies may have been intercurrent or an accidental infection, and that the man has, underlying, the circinate type of dermatitis herpetiformis. The eruption on his penis, the peculiar distribution of the eruption on the body, and the statement that his wife begins to suffer from itching, owing to a similar papular eruption, makes the diagnosis of scabies loom large at the moment.

Dr. Semon: Whilst subscribing to the possibility of the presence of two diseases in this patient, I cannot agree with the diagnosis of dermatitis herpetiformis. I do not see why the vesicular eruption may not be the small bullae which are sometimes seen in acute erythema multiforme, of which I regard this as an example. I regard the peculiar yellowish-pink centre with the peripheral reddish raised edge as characteristic of the erythema multiforme group. I think this is scabies with erythema multiforme, and that if there are, or have been, vesicles, they are a well known complication of erythema multiforme. The recurrent nature of the affection would also fit in with that idea.

[Note.—Since the meeting the patient and his wife have had full treatment for scabies. The rash in the axillae in the patient has certainly benefited, and, in fact, it has vanished, but concurrently with this improvement there has been a fresh outbreak of the ringed erythema seen earlier.]
Section of Dermatology.

President—Sir James Galloway, K.B.E., C.B.

Case of the Condition described as "Multiple, Benign, Tumour-like New Growths."¹

Shown for J. J. Pringle, M.B., by Henry MacCormac, M.D.

The case is one of the condition described by Schweninger and Buzzi under the title of "Multiple, Benign, Tumour-like New Growths," in vol. v, plate 15 of the "International Atlas of Rare Skin Diseases." The patient is an officer in the Cyclists' Corps, aged 21, and is exhibited by kind permission of the officer commanding the military hospital to which Dr. Pringle is attached. He is in perfect health, and no cause can be discovered for the development of the condition, which began in France in December, 1917, first in the presternal region, then over the back and abdomen, and finally over the thighs, upper arms and neck. In these latter regions the lesions are slightly pro-eminent papules; over the abdomen there are innumerable soft bladder-like tumours, which can be reduced by pressure and squeezed back into the skin through button-hole-like apertures; over the chest and back there is deep pitted, atrophic, scarring. In the left upper thoracic and right scapular regions, over areas the size of the palm of the hand, the atrophy of skin is more diffuse and, possibly, not the result of the absorption of pre-existing discrete tumours.

The description of the condition given by Radcliffe Crocker in the third edition of his "Diseases of the Skin," 1903, i, p. 649, is so completely comprehensive that it is quoted verbatim; it runs as follows: "Clinically the lesions are soft, round, or oval projections, from a lentil to a bean in size, more or less white, with a slight bluish or slate colour in some of them. Most of them are bladder-like, and can be pressed into the skin by the finger, projecting again immediately like a hernia. The larger ones are flattened and slightly puckered and

¹ At a meeting of the Section, held January 16, 1919
harder than the smaller, from which they develop. They undergo spontaneous involution, and leave only flaccid, loose, foveated scars. They appear very gradually and without sensory symptoms on the trunk, shoulders and thighs, and ultimately become numerous, as none disappear entirely, and others keep forming. Three out of the four cases were women. One had had syphilis, and she stated that the lesions appeared in a secondary eruption which did not ulcerate, but in the other cases there was no evidence of syphilis.” Further on Crocker remarks: “I have seen very similar lesions associated with fibromata of the ordinary form, when some of them have been absorbed. It is probable that they are the last phase of more than one pathological process.”

With this view that the condition is not a “substantive” disease, Dr. Pringle has expressed his agreement, and the case is shown partly with a view to evoking discussion on the point. It is worthy of note that the appearances in Schweninger and Buzzi’s plate are misleading, as the vivid red colour therein depicted is the result of treatment by thermo-cautery.

DISCUSSION.

Dr. F. Parkes Weber: These spots are already atrophying and becoming typical “rain-drop” spots of macular atrophy. Probably in process of time all the raised, fibroma-like spots will atrophy, and the slight coloration which there is in some of them will be lost, the case becoming a typical one of macular atrophy of the skin; the macular atrophy will last all the patient’s life, without doing him any harm whatever, and gradually the atrophic spots will become less noticeable. I consider the condition one of great rarity, because, although in the history of cases of macular atrophy the patients often mention a stage of raised spots, one seldom sees the stage in question: and certainly one is never likely to see another case in which so many spots are raised as in this case.

Dr. H. G. Adamson: Had I seen this case without knowing Dr. Pringle’s diagnosis, I should have supposed it to be one of “macular atrophy,” but I should have been puzzled by the unusual feature of the hard lumps that are to be felt in some of the lesions. Evidently it is the disease described by Buzzi, and I congratulate Dr. Pringle upon having recognized it as such. The question is whether all cases of what we have been accustomed to regard as idiopathic macular atrophy begin in this way. We know that in some cases of macular atrophy the lesions follow syphilitic lesions—I do not refer to scars, but to typical macular atrophy: and that others occur in tuberculous patients, possibly as a sequela to tuberculides. It seems possible that in patients who have neither syphilis nor tubercle the condition may begin in the way it seems to do in this example of Buzzi’s disease, but that we do not usually see this early nodular stage which precedes the macular atrophy.
Dr. Graham Little: I believe the histology of this very rare condition shows that the appearances are due to a defective production of elastic tissue in the sites of the swellings, so that a sort of hernia of the subcutaneous tissues, especially fat, takes place. A case has been recently reported in the Journal of Cutaneous Diseases, by Pusey, who questions Crocker's identification of his case with the original cases described by Schweninger and Buzzi.

Dr. H. G. Adamson: I do not think that the view which Dr. Graham Little puts forward is a new one: it is well recognized that the macular atrophy is due to loss of elastic tissue, and that this is the reason it is "baggy." Dr. Colcott Fox showed a case in which the patient had general œdema, so that all the macules were pushed out like tumours, disappearing when the œdema subsided. I think that the bag-like appearance of some of the macules is not peculiar to this case of macular atrophy: it may occur in all cases of macular atrophy.

Dr. S. E. Dore: As Dr. Adamson has pointed out macular atrophy may follow various conditions, such as tuberculosis and syphilis, and the peculiar raisin-like patches are common to all varieties, but in this case there is definite tumour formation, and I think multiple soft fibromas are the primary lesions. These fibromas atrophy, and cause the curious pockets which can be felt in the skin.

The President: The point which Dr. MacCormac might clear up for us is this: there is no doubt atrophy of the skin, which can be called macular atrophy, but the question is whether this preceding tumour-like appearance of the skin is of the nature of a fibroma, or whether it is some form of degeneration of the white connective tissues, associated with actual atrophy of the elastic tissue, allowing this hernial protrusion, including that of the corium, and to some extent that of the subcutaneous tissue. That it leads on to a condition of atrophy seems perfectly clear, but we agree we have never seen a case of what we call "macular atrophy" with this excessive amount of tissue, with increase in bulk, so far as the surface is concerned. The point I raise is likely to require a good deal of further investigation.

Dr. Semon: With regard to its being fibroma or macular atrophy, it is important to note that there is the complete absence of pedunculation about the growths. With the large number of tumours present in this case, if there had been a growth of the fibrous tissue or of the fatty tissue with it, in other words, fibromas, there would almost certainly have been some pedunculation. In my opinion this is strong clinical evidence against the probability of a diagnosis of fibroma.

Dr. F. Parkes Weber: With regard to soft multiple molluscous fibromata which atrophy and form a kind of shrivelled grape-like projections or excrescences on the skin, it is very rare to see more than three or four in one person.

m—2a
Dr. MacCormac (in reply): The case will remain under Dr. Pringle's observation, to whom I shall represent the opinions expressed; possibly sections may be obtained.

Case of Mycosis Fungoides.

By E. G. Graham Little, M.D.

The patient is a middle-aged man, sent to me by Dr. MacMunn. The disease began with a swelling on the cheek a little over two years ago. This developed into a granulomatous tumour occupying the side of the nose and the cheek below the left orbit, over an area of about 2 in. by 3 in. The tumour has receded somewhat from the nose and spread towards the ear. He was seen by two specialists, one of whom seems to have diagnosed syphilis, and appropriate treatment was recommended, but apparently without producing much, if any, improvement. The body was clear of disease until about seven months ago, when the present extensive rash made its appearance. This consists of large areas of slightly infiltrated, scaly, dry dermatitis, scattered almost over the whole trunk and covered surfaces of the limbs. This later eruption is hardly at all itchy. The development of the tumour before the general rash and the comparatively moderate itching make the case a difficult one to class, but I believe it is an instance of mycosis fungoides of the so-called "tumeurs d'embrée" type. I hope to show a section later.

Dr. S. E. Dore: I saw this patient in September, 1917, and at that time he had an ulcerating lesion on the face, and circular, punched out ulcers on the legs, which I thought were probably syphilitic. He also had what I thought was an eczematous eruption on the legs and on one palm. The lesions did not show much improvement under mercury and iodide of potassium by the mouth, and he was unable to have a Wassermann test done. I think Dr. Little's diagnosis of mycosis fungoides is probably correct, although the appearance of the tumour stage before the eruption, if this actually occurred, is very unusual.

White-spot Disease (Morphoea Guttata).

By J. L. Bunch, M.D.

The patient, a girl aged 9, has developed, during the past two years, several skin patches of an ivory-white colour, which are distinctly sclerodermatous. The largest patch is situated over the front of the
left tibia, is oval in shape, and measures 3 in. in its longest diameter. It is quite hard to the touch, and there is considerable thickening of the skin. In one place it has not recovered from the effects of a kick some three months ago, which caused superficial ulceration at the time and now redness. Otherwise the patch is ivory-white and quite smooth. Behind the right calf is a smaller white patch, also sclerodermatous, about the size of a shilling; and above the left ankle one the size of a sixpence. On the abdomen is a similar patch about the size of a pea, and between the scapula three more of the same size, with intervening areas of healthy skin. All these patches are quite white, shiny, and have all the characters of morphea. Above the left knee are several very small, punctate, white spots which cannot be said to feel sclerodermatous, and it appears possible that these spots will coalesce later and form an indurated patch like the one over the tibia.

There are well marked patches of erythema on the back and chest, and the child is said to develop such patches frequently, but they soon pass away. During the last two years the child has been living at Leyton, and has had experience of several air-raids and has become emotional and excitable, and the mother ascribes the skin condition to her nervous condition.

**DISCUSSION.**

The President: All these cases of morphea and scleroderma are of interest. Dr. Bunch makes a note of the kick on the shin, but I presume he does not suggest that it had anything to do with the causation of the disease, though no doubt it complicated it, so far as healing is concerned, owing to the scleroderma skin in the neighbourhood. The possibility of the association of this type of morphea with the linear scleroderma must always be present in our minds. The association of injury occurs to me, because within the last day or two I had a case sent to me in consultation from Bradford, the patient being a child of about the same age, with very characteristic linear scleroderma affecting the left pectoral girdle and the left arm. And it was not only arranged in accordance with the segmental somatic relationship of the arm, but the scleroderma had a peculiar spiral distribution, showing the way in which the arm developed from the somatic segment, not as an out-push, but in spiral fashion as the arm received its full development. The scleroderma commenced at the back of the hand, and produced contraction, so as to impede the movements of the hand from the ring finger upwards, working round the arm, terminating at the shoulder. The interesting point was that two or three years ago, when the disease commenced, the child had a blow on the back of the hand, which produced a bruise, and that took a long time to heal. The parents were convinced that the scleroderma commenced at that
time, and, as they think, in consequence of the injury to the back of the hand. Only in the sclerodermic area of the back of the hand was there anything in the nature of excess of growth. Where the injury occurred, the area was of a slightly keloid character. So when Dr. Bunch began to speak of the kick in this case, I thought he was going to launch into an aetiological association between the two. Whether these guttate types of morphœa are the same in aetiology as the linear type is still a matter for discussion, but as these cases are of exceptional interest in regard to aetiology and pathology, I shall be glad to hear the views of others. I fear there is not much to be done in the way of treatment. I do not think thyroid gland treatment is of any real value. But if the condition affects certain regions, continuous massage does good. I have shown cases of supra-orbital morphœa which caused much trouble in movements of the upper lid, and after continuous massage the skin showed a tendency to become more supple and comfortable.

Dr. S. E. DORE: I agree that Dr. Bunch's case is localized scleroderma or morphœa, with guttate lesions, but it differs from my cases and the case shown by Dr. G. W. Sequeira in the distribution and in the fact that there is a large patch of ordinary morphœa, as well as the guttate lesions. This shows that the guttate or "white-spot" patches are morphœic, and not atrophic lichen planus papules. I regard the atrophic macules in this case as a late stage of the guttate morphœic lesions.

Dr. H. G. ADAMSON: An interesting point is the association of blotchy erythema. That is not a sufficiently recognized association: an erythema, or urticaria, or even fluid in the joints in early cases of scleroderma, suggests that the condition is a toxic erythema. When I suggested putting the child to bed, I did so because the condition is in the acute stage; it is best to keep the patient in bed until the inflammatory redness and swelling has subsided. I recently had a case of scleroderma in a girl, the whole of whose arm was swollen; she was in what Crocker called the edematous stage. After a few weeks in bed this subsided and left scleroderma patches. There appears to be no distinction between the patchy type and that in which there is a segmental distribution. My view of the guttate type is that it is simply its early appearance around a hair follicle which determines it, and that the lesions afterwards coalesce into a patch. It is the beginning of the scleroderma around the more vascular parts of the hair follicle. In the present patient the patch on the leg is surrounded by smaller outlying patches around hair follicles. Both types, the band form and the guttate form, may occur in the same patient.

Dr. PERNET: There may be some difference in the aetiological factors at work, or in their incidence, in the usual morphœic and sclerodermic forms, and on the other hand, band and segmental forms; and also as regards those types with herpetiform distribution and sclerodactyia.

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Dr. F. Parkes Weber: In regard to the remarks of Dr. Adamson, I would remind members that in the early stages of the symmetrical generalized type of scleroderma the oedema-like appearance may be at first so striking (even on the face) that the condition may be mistaken for kidney disease. Then the swelling may disappear. It is during the hypertrophic, "oedematous," stage that there is still some hope of almost complete recovery.

Dr. Bunch (in reply): I think the history is clear that the white patch preceded the kick. The skin condition began two years ago, the kick was received three months ago. The erythema in this patient is new to me: I did not see it before to-day, when Dr. Adamson called my attention to it. I do not think Dr. Dore and I clearly understand one another about that previous case of "white-spot disease." I inclined to the view that it was lichen planus atrophicus, and therefore that it was not white-spot disease. If it had been morphea guttata I should have called it white-spot disease. If it was definitely an atrophic disease I think that would exclude it from the white-spot disease group. Some of the patches on the child's leg are very small and very slightly sclerodermatous. These punctate lesions will probably become more thickened later on, and I regard them as showing early, not late, stages of the disease.

Case of Pigmentation of the Mucous Membrane.

By H. C. Samuel.

The patient is a widow aged 65. She has had eight children and one miscarriage, all before the age of 35, when the present condition was first noticed on the lips. The lesions are pigmented spots, which first appeared on the lip, others following on the buccal mucous membrane and palate. There was also one isolated spot on the skin of the forehead, which was removed surgically by Mr. Mower White sixteen years ago. The patient's husband died of "consumption," and six children also died young of conditions which suggested tuberculosis. The patient was seen by Dr. Sibley twenty years ago, and I understand also by Sir J. Hutchinson. At that time the tachycardia was also noted, and is now quite a marked feature with a systolic murmur.

What is the aetiology of these pigmented spots? The marked resemblance to the inside of a dog's mouth suggests the idea that the whole condition is of the nature of pigmented naevus. The fact that they did not appear till the age of 30 does not negative that hypothesis.

I do not know whether there is any association between the present condition and the heart trouble. I brought her in order that I might ascertain opinions as to aetiology: whether members regard it as a naevus
or whether it has any connexion with disorders of the suprarenal gland. In Addison's disease, also associated with tachycardia and tuberculosis, there is a quite different history.

DISCUSSION.

Dr. Pernet: I have seen a few examples of this kind of case. I consider the condition is congenital. I have compared them with the black spots seen in the mouths of dogs.

Dr. F. Parkes Weber: Similar cases of pigmentation not due to Addison's disease have occasionally been brought forward in London. This case, I suspect to be an example of a kind of simple pigmented nævus of the mucous membrane of the oral cavity. If members care to look up the accounts of cases with patchy pigmentation in the mouth, they will find that a sufficient number have had pernicious anaemia to make one suspect there must be an association between the two conditions; such cases have been recorded by Hale White, H. French, A. Lazarus, H. D. Rolleston and T. G. Moorhead.

Mr. Samuel (in reply): I certainly regard this condition as nævoid, but it is suggestive that it did not appear until the patient was 30 years of age (although that does not necessarily exclude nævus), and that there should be such a marked family history of tuberculosis as well as of tachycardia in her own case. I think there may possibly be some association.

Case of Multiple Leuconychia Striata, associated with Leuconychia Totalis of One Thumb Nail.

By George Pernet, M.D.

The patient is a young woman aged 28, first seen by me at the West London Hospital on December 20, 1918. Six months previously the left thumb nail began to grow up white from the matrix end. This nail was entirely of an opaque chalky white. Four months later other nails began to show changes in the shape of white transverse bands, also starting from the matrix end. The photograph shows the condition at that time. The toe nails were not affected. About three and a half months ago all but five teeth in the lower jaw were extracted on account, I take it, of pyorrhea. The correlation between nails and teeth must be borne in mind. There was no history of any precedent illness to account for the nail changes, but the pyorrhea may have been the fons et origo or have played some part in its production. The patient has been and still is in good health, and Dr. J. A. Butler, who sent the case to me and overhauled her, had found nothing wrong with
her. The patient herself thought it was due to her occupation of handling and serving out butter and margarine. There was no sign of inflammation or infection about the matrices of the nails. I put her on mist. arsen. et strych., and there are already some signs of improvement. The association of leuconychia striata and leuconychia totalis is uncommon.

Dr. F. Parkes Weber: I think this is an early stage of leuconychia totalis, and in some of these cases it is impossible to find a predisposing cause. In certain cases, however, the subjects of the condition are distinctly neurasthenic, and this woman tells me she is very "nervy." I happen to have shown one of the earliest reported cases of leuconychia totalis in England. I do not know whether in any cases the leuconychia totalis has been known to have been preceded by leuconychia striata. Dr. Pernet's present case is the only one I know of in which leuconychia totalis of some nails has been associated with leuconychia striata of other nails. In a recent paper I have tried to collect the accounts of all cases of either condition. Dr. Pernet mentioned giving arsenic to this patient. One kind of leuconychia striata has been said to be due to arsenic, and has been named "leuconychia striata arsenicalis."

1 Weber and Krieg, Brit. Journ. Derm., 1899, xi, p. 120.
Case for Diagnosis, sent from University College Hospital.


The patient, a male, aged 27, only presented himself at the hospital this afternoon. He is a Belgian. There is no history of syphilis. The present condition began three years ago, and since then he has had several recurrences. There are papular, nodular discrete lesions, of lichen planus type. They are more or less generalized: some are aggregated together. In parts they are covered by grey scales. The skin of the penis is affected, but not its mucous membrane, nor that of the mouth. The scalp and face (moustache region) are also affected. The Wassermann test has not been made. Dr. Stowers is inclined to regard it as a variation of lichen planus, and I cannot suggest any other diagnosis, but a further investigation will be made.

DISCUSSION.

Dr. Semon: The extraordinary thing about this case is the absence of involvement of mucous membranes. On the penis the eruption stops short in a peculiar way on the prepuce, and the glans is not involved at all. I should have thought that in a case of lichen planus of that extensive distribution there would have been some involvement of mucous membrane. Dr. MacCormac will bear me out in the statement that we saw many cases of lichen planus together in France, and in a large proportion of these the mucous membranes were involved. I cannot suggest any diagnosis other than lichen planus, but I think a biopsy would be useful. The individual lesions, especially those on the anterior surface of the wrist and on the neck, are typically lichen planus, but the agglomerated masses of eruption do not help that diagnosis, and I have never yet seen a case in which the face and scalp were involved.

Dr. Pernet: On looking closely with a lens at some of the elements on the flexor surfaces of the wrists, one cannot come to any other conclusion but that it is lichen planus, and one would scarcely argue that the condition further up and on other parts is something else. I consider this must be lichen planus, but of a very exuberant type—lichen planus obtusus.¹ In my experience of lichen planus generally, I would say there may be extensive involvement of the skin and the buccal mucous membranes be little affected. On the other hand, I have seen cases of marked involvement of the mouth, with moderate or slight involvement of the skin generally.

¹ When the case was shown on the first occasion some time ago, I pointed out then the lichen planus appearance of some of the elements.
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The President: I am glad that obvious difference of opinion has been manifested in this discussion. When I first saw the case the distribution and the general appearance of the condition suggested to me pityriasis rubra pilaris. Then what Dr. Pernet said about the character of the wrist and neck lesions made me wish it were possible to reverse my first impression. But, even so, I am not inclined to be diverted from my original view. I think there is no aetiological relation between the two conditions, but long-lasting lesions of pityriasis rubra pilaris come to resemble the lesions of lichen planus extraordinarily closely. So I cannot arrive at a firm diagnosis. The diagnosis, three years ago, of granuloma of undescribed origin, would still make me hesitate before committing myself. The case is an unusual one, and I shall be glad if Mr. Samuel will ask Dr. Stowers to let us know the result of a histological examination, and to supply any further details that may be forthcoming. The case evidently provoked a good deal of discussion when it was originally shown. At that date Dr. Pernet did not rule out the possibility of its being xanthoma, and Dr. Adamson thought it was xanthoma diabeticorum, whereas now he believes it to be lichen planus.

Postscript by Dr. Stowers.—Since this patient was exhibited it has transpired that the case was shown and fully described by Dr. Dudley Corbett in 1914. A full account will be found in the Proceedings of the Dermatological Section reporting the meeting, and subsequent discussion, for December 17, 1914. It was then regarded by some members as an infective granuloma, by others as a new and previously undescribed disease. It is intended that a further report shall be made on the case at a future date.

Case of Arsenical Pigmentation and Hyperkeratosis occurring in the Course of Dermatitis Herpetiformis.

By E. G. Graham Little, M.D.

The patient is a woman, aged about 30, who has been under my care at St. Mary’s Hospital for the past two years, during which time she has been taking small doses of arsenic. She has been a very interesting patient throughout. She has typical dermatitis herpetiformis which has been controlled with singular certainty by arsenic, her dose to produce control being 3 minims three times a day, rarely increased to 5 minims when the smaller dose seemed to hang fire. She has taken the drug nearly continuously, for experiment has shown that if she

leaves it off the rash returns within a few days, but she has kept
the dose down to the smallest that relieves her of the rash. A few
months ago she began to show the typical "rain drop" pigmentation of
the abdomen, which has slowly increased, until now the greater part
of the surface of the chest and abdomen are affected. Still later she
began to show a granular condition of the palms and soles, which were
thickened and covered with minute hyperkeratotic elevations. There
is no disturbance of the reflexes nor numbing of sensation, and no
symptoms of acute arsenical intoxication have been observed at any
time. The question arises whether one is justified in allowing the
patient to follow her own wish, which is to run some risk as the price of
comparative immunity from the distressing symptoms of her disease.

DISCUSSION.

The President: In early days there existed certain groups of out-patient
hospitals and clinics. Some dealt with eye diseases, some with skin diseases.
It was stated that patients who emerged from the eye disease clinic had drops
put in their eyes as a matter of routine: patients emerging from the skin
clinic had arsenic prescribed to them. Hence it is not surprising that patients
developed arsenical poisoning in its various types, and the number of post-
arsenical phenomena which we used to see was much greater than now.
Still, the question does occasionally arise as to whether it is wise to continue
the drug or not, in what may appear to be medicinal doses.

Dr. Semon: In a recent issue of the British Journal of Dermatology, Mr. McDonagh pointed out that the toxic manifestations of arsenic could be
controlled by intramine.

Dr. Pernet: There is some danger of cancerous degeneration in these
keratotic lesions. In a case I published with Radcliffe Crocker, the patient
had to suffer amputation of the thumb and index finger.

Dr. S. E. Dore: An important point seems to me to arise out of this
discussion, namely, that in order to get much benefit from arsenic in dermatitis
herpetiformis it is necessary practically to poison the patient, in other words,
the eruption does not yield until sufficient arsenic has been given to produce
symptoms of arsenical toxaemia. All the cases I have seen controlled by this
drug have shown toxic symptoms sooner or later.

Case for Diagnosis.

By H. G. Adamson, M.D.

The patient is a nursing sister, and the condition for which she came began in June, 1917, in Baghdad. She was bitten by mosquitoes, and the bites were followed by sores, which suppurated. The condition was diagnosed as Baghdad boil, and she was treated by X-ray applications. Apparently the suppuration continued until she was on her way home in January, 1919, and then the lesions began to dry up. She now has eight patches, varying in size from that of a threepenny-piece to that of a shilling: one on the cheek, one on the neck, three on the left arm, one on the right arm, and one on the ankle. These are circumscribed patches, dusky-red, with slightly raised margins, and a centre which, in some, is crusted, in others is grey and striated, making it rather suggestive of lichen planus in large patches. Others of the patches are stippled and look like those of lupus erythematosus. From the history of suppurating lesions, I think it may have been Oriental sore.

I brought her not only that I might have views on the diagnosis, but also in regard to treatment.

DISCUSSION.

Dr. MacLeod: With regard to the suggestion that it is Oriental sore—by that I mean a sore due to Leishmania tropica—the absence of induration is against that diagnosis, and it is more likely to be a streptococcal infection.

*At a meeting of the Section, held March 20, 1919.*
or due to some micro-organism of the type which causes veldt sore. Good results have followed the treatment of Oriental sore by curettage and X-rays.

Dr. A. EdDowes: If I had not heard the history, I should have thought this was like so many cases I have seen in soldiers back from the Front. In the first case of apparently the same nature which came under me, staphylococci were found in the lesions. Therefore I called them subacute boils. I think this is a chronic form of such infection, and that it will yield to local treatment appropriate for that condition.

Dr. Semon: We had two cases in France in soldiers from Mesopotamia; the situation was, as in this case, on the cheeks and forearms, and in both cases Captain Small, R.A.M.C., found the Leishman bodies in the discharge. They cleared up in three months. X-rays are recommended as a good treatment for Oriental sore. We applied the rays to one of these cases, but not to the other, and there seemed to be no difference in the progress of the two. Probably as soon as patients leave the Tropics the foci tend to clear up, as they certainly appear to be doing in this case.

Dr. Whitfield: I thought that Leishmaniasis cleared up if you gave salvarsan, both internally and locally, but I have no personal experience of treatment of the disease.

Dr. Pernet: I have seen several cases of Oriental sore, especially at Biskra. They were not quite like this case, being more crusted and indurated at borders. An Italian I saw in the street at Biskra had five of these sores scattered about the face. They had been present five months. He had had no treatment and did not appear to worry about them. In the garrison hospital at Biskra I was shown several cases: they were mostly multiple. I have dealt with a "Chitral sore," which is the same thing. Thorough curettage and the use of acid nitrate of mercury cured it. In "The Adventures of Haji Baba of Isphahan," by Morier, there is a very good description of Oriental sore. The condition may clear up spontaneously, leaving a scar. The appearances in the present case appear to be in the involuting phase.

Dr. Adamson (in reply): There is a difference of opinion as to the value of salvarsan for this. I had one case in hospital in a soldier whose condition cleared up with the use of galyl: it cleared up less quickly than syphilis does.

Postscript.—Pathological report, April 8, 1919: "Film from scrapings shows Leishman-Donovan bodies in small numbers." Further investigations are being made.

1 In Algiers, Professor Brault told me that the clou or bouton de Biskra, was also known as "le chancre du Sahara," and that dogs and horses also suffered from the disease about the nostrils.—G. P.
Case of Multiple Neuromata of the Skin.

By E. G. GRAHAM LITTLE, M.D.

The patient is a South African soldier, aged about 30. He was quite well until about two years ago when he began to develop nodule-like those now seen on the right leg, without any history of injury. He has now some hundreds of small bluish, reddish and hard nodules, the size of a split pea, in the substance of the skin, extremely tender to touch, so that even the slight friction of the trousers causes him pain. There are a few similar lesions on the left leg, and none elsewhere. There are no soft tumours such as are found in Von Recklinghausen's disease, which was one diagnosis suggested. The section shows that the bulk of the tumour is made up of nerve-fibres so that neuroma is a justifiable diagnosis, notwithstanding the extreme rarity of this condition.

Case of Dercum's Disease.

By E. G. GRAHAM LITTLE, M.D.

The patient is a woman aged 54, of stout build, formerly the wife of a publican and then accustomed to take alcohol to some excess but not at all in the last sixteen years of her widowhood in which her circumstances have been somewhat reduced. The tumours for which she sought my treatment date from about seven years ago, during which time they have slowly increased in size. At present she has two large pad-like masses about 8 in. by 6 in. in surface measurement forming pendulous swellings above each elbow, apparently consisting of diffuse lipoma, without any discrete nodules, and of a dark violet redness from cyanosis. They are extremely cold to touch, quite different from the temperature of the body. She has two much smaller pads of similar tissue on the back of each wrist. The tumours are painful and disturb her sleep. During the last six years she has lost 4 st. in weight, although at the same time the tumours have grown larger, so that although she is still a stout woman (12 st. at present) the tumours cannot be said to form part of a general increase of fat. There is a somewhat obscure history of her mother having died of diffuse tumours.
described as cancer. The remarkable symmetry of the adipose deposits, the typical diffuse pad-like swellings, and the pain associated with them seem to me to make the diagnosis of Dercum's disease (adiposis dolorosa) probable. An X-ray photograph of the thyroid shows no diminution in size and no calcareous deposits as were found in two of Dercum's cases. The remarkable cyanosis is probably to be explained by the poor vascularity of the fatty new tissue.

DISCUSSION.

Dr. Pernet: In Dercum's disease there is no discoloration: as far as I remember, the few cases I have seen were of the colour of the ordinary skin.

Dr. Whitfield: Most cases of Dercum's disease have a calcified thyroid, and if that were the case here, it would support the diagnosis against the tubercular hypothesis. At King's College Hospital, not long ago, I had a case when I was acting as Internal Physician. Thyroid was given for a short time, but it did no good at all.

Mr. H. C. Samuel: Is Dercum's disease associated with mental changes? I understand a certain number of the cases have been found to possess a peculiar mentality, so that a woman aged 38 may behave like a child of 8. This patient's mental state seems to be normal.

The President: The distribution of the fat deposit is an important point in the diagnosis of such cases of irregular adiposity. It is not unusual to see a considerable increase of subcutaneous fat on the outer aspect of the arms, especially in the case of women. The skin covering the fat is usually discoloured owing to varying degrees of stagnation erythema, and often tender and painful. Such cases have been frequently commented on by Dr. Weber, Dr. Adamson, by myself and others. This patient seems to be of this type. But it is very doubtful if such a case should be grouped with the cases described as "Adiposis dolorosa." Dr. Dercum illustrated his early papers with representations of the peculiar and often symmetrical distribution of the painful fatty masses and the picture is different from that presented by this patient. Much has been written on the various forms of lipomatosis since Dercum's original paper, especially with reference to the relationships existing between such states and imperfect function of the glands of internal secretion. In some cases good results have been obtained after the administration of extracts of the thyroid and other endocrine organs." Has Dr. Little used thyroid or any other endocrine medication in this case.

Dr. Little: No.
Case for Diagnosis.

By H. W. Barber, M.D.

I have had this case under observation since early in February. Though I am showing it as one for diagnosis, I incline to the view that it is pellagra.

The patient is aged 50, and is a painter and paperer by trade. Since October, 1914, however, he has been in the Army on home service.

Family history: His father was subject to "fits," from which he has now recovered. The patient is married, and his wife is alive and well. They have had eight children, of whom the sixth, a girl, has recently had "fits."

Previous history: He has himself been subject to "fits," apparently epileptic in character, since childhood, and on account of these he was discharged from the Navy at the age of 20, after six years' service. At this time he had sores on his penis which were diagnosed as syphilitic, and for which he was treated for about three months with mercury pills and black wash. No secondary symptoms developed. At the age of 36 he again contracted sores on his penis, and his foreskin sloughed off; shortly afterwards he was in St. George's Hospital for about twelve weeks with synovitis of his knee, which, he says, was diagnosed as being due to syphilis, and he was treated with medicine only. Two years ago he was treated at the Herbert Hospital, Woolwich, for "erysipelas" of his face, but the attack was not accompanied by fever. He has also been treated at the same hospital for "neuritis" of the arms with radium baths and massage. He states that he has noticed swollen glands in his neck for the past four years. No history can be obtained of his having had arsenic given him internally. He has had no fits during the past two years.

History of the skin eruption: At Christmas, 1917, while on sentry duty on the marshes near Dartford, he noticed a small swelling on the forefinger of his right hand like a "gnat-bite." The next day this hand became swollen, red, and tender, and a few days later the left hand became similarly affected. Soon afterwards the nails of both hands began to crack. He was treated for "rheumatism," and given mist. sodii salicylatis. Three months later the feet became affected in the same way, and in the summer the skin of his ears, nose, and cheeks
became reddened and very tender. Just before admission to Guy's Hospital he noticed a similar condition of the skin over his elbows and knees. During all this time he was subject to attacks of very severe pain in his hands and feet, so severe indeed that he even developed suicidal tendencies, and he became very depressed. With these attacks the skin of the affected parts became more inflamed and swollen, and was exquisitely tender.

Condition on admission to hospital: The patient came under my observation on February 11 of this year, and he was admitted under Dr. Lauriston Shaw, who very kindly allowed me to take charge of him. At that time the hands and feet, both on their extensor and flexor surfaces, were involved in an acute erythrodermia: the skin was reddened, infiltrated to the touch, and extremely hyperaesthetic. The epidermis of the palms and soles was desquamating in thick scales, and the nails of the hands were brown and fissured. Examination showed that a similar, though less acute, condition of the skin was present in the following situations: Over the elbows and front of the knees in bilaterally symmetrical patches, on the buttocks over the ischial tuberosities and around the anus, on the ears, the nose, and the butterfly area of the cheeks. Apart from the erythrodermia there was very evident brownish pigmentation of the skin in the affected areas, most marked on the wrists, whence long brown streaks extended up the forearms, on the extensor surfaces of the feet and ankles, over the malleoli, and on the buttocks. The patient had generalized tremors and seemed in a very nervous state. Further examination showed that below the angle of the right lower jaw there was a visible mass of enlarged hard glands, and other glands were palpable on both sides of the neck; the epitrochlear, axillary, and inguinal glands were also enlarged. There was superficial leucoplakia of the mucous membrane of the inner surfaces of the cheeks. It was also observed that the skin of the tip of the nose was atrophied, giving to the organ a "pinched" appearance. The knee-jerks and ankle-jerks were definitely exaggerated: arm-jerks not obtained. Plantar reflexes flexor. As regards sensory changes there was no apparent disturbance of the sensations of touch, heat, and cold in the skin of the affected parts, but very marked hyperalgesia as tested by a pin-point. The limitation of this hyperalgesia to the areas involved in the eruption was very striking. Nothing abnormal was found in the chest and abdomen, and there was no albumin nor sugar in the urine.

Two days after his admission to hospital large symmetrical bullæ
developed on the dorsum of each foot, and the patient complained of great pain in his hands and feet. The bullae subsided in a day or two, and did not become infected. A differential leucocyte count showed increase in the mononuclear cells (37 per cent.). The Wassermann reaction was performed twice in Dr. Eyre's laboratory, and was negative on each occasion. A gland was excised from the neck, and was kindly examined by Dr. Nicholson, who reported that it was involved with squamous-celled carcinoma. No primary source has as yet been discovered. While in hospital the acuteness of the skin condition has subsided, though he has had two or three attacks of acute pain in his feet and hands. The large epidermal flakes on the palms and soles have completely desquamated.

The appearance and distribution of the eruption led me at first to consider the possibility of its being pellagrous, and further consideration has strengthened this view. There is one point in his history which I think is suggestive, and that is that in the Army he was employed chiefly as a cook, and he states that during this time he lived mainly on puddings, having no appetite for meat, and eating little or no vegetables and fruit.

The aetiology of pellagra is still a matter of discussion, but I understand that the late Dr. Sandwith believed it to be a deficiency disease, in the same class as beri-beri. If this case is not pellagra, I cannot suggest any other diagnosis.

DISCUSSION.

Dr. G. PERNET: I consider this is a case of pellagra. It must be borne in mind the patient has been under treatment and has improved, so we must make allowances. Dr. Barber says it was red and scaly when first seen. I have seen a number of cases a little way out of Rome. On the occasion of the International Dermatological Congress of Rome in 1912, Dr. Sambon bade us visit a pellagrous village, very primitive and more like a kraal. But there the people are constantly in the open air and exposed to strong sunlight. Dr. Barber had the advantage of observing the case in its inflammatory stage, and from what he has told us I think his diagnosis is correct.

Dr. F. PARKES WEBER: My experience of pellagra is limited, but Dr. Sambon showed me some cases in asylums in England, which he assured me were absolutely typical. In Dr. Barber's case there seems to be no seasonal change such as there is in pellagra cases. The face suggests to me diffuse scleroderma, and the patient has a pinched nose such as that resulting from sclerodermic tightness of skin. Perhaps the condition is an atypical form of generalized scleroderma which will become more typical later on.
Barber: *Two Cases of Granulosis Rubra Nasi in Boys*

Dr. Semon: I saw this case at Guy's Hospital with Dr. Barber, and there were then enormous bullae on the dorsum of the feet, and bullae are a common complication of pellagra. There was also marked hyperaesthesia. If it were ringworm, would there be this marked reaction, and how could the pigmentation be thus explained? The only feature which seems difficult to explain on the pellagra view is the condition of the nose, the pinched appearance of which has been mentioned.

Dr. Barber (in reply): Surely the condition of the nose in this patient is typical of pellagra, for the dermatitis of pellagra is followed by atrophy. I showed him to some Australians who had seen cases of pellagra in Egypt, and they said at once that the condition of the nose is typical, and that the atrophy usually showed itself on the nose. As Dr. Semon said, I do not see how the occurrence of bullae can fit in with the diagnosis of psoriasis, and as the patient has not had arsenic, the marked pigmentation is against psoriasis or a tinea infection. I have had the patient under observation, and a striking feature has been the recurrent attacks of acute pain in the hands and feet; he is extremely hyperalgesic even now. The nails in pellagra are occasionally affected in the way you see these are.

**Two Cases of Granulosis Rubra Nasi in Boys.**

By H. W. Barber, M.D.

The condition is not now so marked in either patient as when I first saw them for then both showed very well the excessive sweating over the nose, distinctly so in the central furrow of the lip. Both have had chronic ill-health, both have cold hands and feet, and are of the "scrofulous" type. I shall be glad to hear views as to the cause of the condition. The appearance of both these patients suggests that they have latent tuberculosis.

**DISCUSSION.**

Dr. A. Eddowes: Have these boys adenoids? They seem to have, especially the younger. If so, such would provide a source of irritation to the end of the nose.

Dr. Graham Little: I have had two cases, which did very well after treatment with carbon dioxide snow.
Alopecia of the Scalp and Eyebrows associated with Graves's Disease.

By H. W. Barber, M.D.

I am showing this young woman because she illustrates the connexion between alopecia and disturbance of the thyroid gland, a subject in which I am very much interested in connexion with influenza. Her hair began to fall out three years ago, and she became quite bald last summer, losing the hair of the eyebrows and eyelids too. Apparently the pubic hair is normal. When I first saw her, at the beginning of the year, her thyroid enlargement was more pronounced than now; she also had exophthalmos, though only slightly. She showed the flushing of the face and upper part of the neck, and very fine tremor, and tachycardia. I kept her under observation the whole of one afternoon, and her pulse varied between 130 and 145 per minute. Her general condition has improved under observation; her alopecia is still complete. I shall be glad of suggestions for treatment. I have not given any gland preparations, but perhaps a combination of them might do her good. With regard to the cases of post-influenzal alopecia, the worst cases I have had have shown large thyroids, tremor, and tachycardia, and in fatal cases of influenza one always finds the thyroid enlarged. I have some preparations of thyroid cut from cases of influenza, and I think they will show conditions similar to those in Graves's disease.

DISCUSSION.

Dr. Adamson: I think all these cases of total alopecia are instances of alopecia areata at first. When the hair comes off in illness it is never a complete falling off. I do not think the association of alopecia with Graves's disease is very uncommon. The association is of interest, because one view held of alopecia is, that it is due to some internal gland secretion disturbance.

Dr. S. E. Dore: This is not the first time that alopecia areata has been described in association with Graves's disease. Dr. Burney Yeo, many years ago, described a case in which there was enlargement of one lobe of the thyroid, with exophthalmos on the opposite side, and alopecia of the eyebrows and eyelashes on that side, followed by the converse. ¹ Dr. Jenkins ² recorded

a case in the British Journal of Dermatology, following an operation on the thyroid (in this case there had been a previous attack of alopecia areata), and Dr. Sequeira has seen two cases of alopecia areata following partial thyroidectomy.1 The evidence for regarding alopecia areata as a direct result of excessive or definite secretion of the thyroid gland appears to me to be inconclusive and its association with Graves’s disease is so rare that it would seem to be rather a part of the general nervous disturbance of that disease or simply coincidental to it.

Case of Melanotic Nævo-carcinoma (previously shown).2

By George Pernet, M.D.

This patient, a woman, aged 48, was brought before the Section two months ago. She has continued under the care of Dr. Lynham, at the Radium Institute, who is to be congratulated on the good results he has obtained. I am sending round the photograph of the original growths for comparison. They have quite disappeared. Moreover, Dr. Lynham has been treating with radium the part below the lobule of the ear where there was some thickening.

DISCUSSION.

Dr. Whitfield: Many years ago after working at the subject of moles and nævo-carcinoma I ventured to express the opinion that there were two types of nævo-carcinoma. The first is so hopelessly malignant and affects glands so early that some observers are of opinion that operation makes them even worse. The second type is malignant microscopically, but its course is more like that of a rodent ulcer. I remember one case in which a small ulcerating tumour developed in the centre of a widespread pigmented growth. At the first operation the surgeon removed the tumour only. Two years later the malignant disease having recurred the whole pigmented area was removed and the patient did perfectly well. I secured the tissue removed at both operations and found microscopical evidence of malignancy in both pieces. At the discussion on malignant tumours held by this Section a few years back I showed a number of photomicrographs illustrating my contention. I have seen one case which the medical man was bold enough to treat by freezing with CO2: it was not quite cured, but nothing very bad occurred, and the growth was successfully removed later. The point is, I think, that there where there

is a widespread pigmented area with a growth at one corner or in the centre, the variety is probably the not very malignant type, whereas where the whole area is tumour and there is no wide surrounding pigmentation, and also where one gets the mushroom-shaped tumour, the variety is of the ultra-malignant type. This case would, therefore, in my view, be of the mild type, and I am not surprised that radium has done some good. In my opinion wide removal will be followed by cure if the subsequent course shows that radium will not cure it.

The President: There is no doubt that radium does well in some of these cases. At the time Dr. Whitfield was at work on this subject I read a communication on, and showed photographs and sections of melanin-containing growths, at the meeting of the British Medical Association at Montreal. It was very pleasant to find that Dr. Whitfield’s observations fitted in with the discussion which took place at that meeting. Dr. Whitfield is right as to the classification which he has made in regard to the degrees of malignancy. It is not every pigmented growth which is severely malignant.

Dr. Pernet (in reply): I had the opinion of my surgical colleague on this case, and he was not anxious to operate, but agreed that radium should be tried. The black mark has been in existence from birth. It is a nevus. The patient knocked it accidentally, and then the growths developed.

Case of Unilateral Band Scleroderma and Morphæo-scleroderma of the Left Leg.

By George Pernet, M.D.

The patient is a married woman, aged 31, who first came under my observation at the West London Hospital on February 14, 1919, for thickening of the skin spreading up the left leg. Two years before that she had a knock over the left outer malleolar region, which was followed by a good deal of local swelling for three or four weeks. According to her account she caught cold in it. Rheumatic fever developed and she was in bed for eight weeks. The swelling about the seat of the injury quite disappeared.

About the end of August, 1918, thickening of the skin began in front of the left external malleolus and spread upwards in band form as far as the head of the fibula. Then thickening occurred from about the middle third of the left thigh and spread upwards to the fold of the nates on the same side, in which situation there is a mixture of scleroderma and of
Pernet: *Unilateral Band Sclerodermia*

lilac-bordered morphea. A narrower band of sclerodermia appeared on the instep, extending forwards from just in front of the malleolus. These bands are characteristic of sclerodermia and very adherent to the underlying structures, causing a good deal of pain about the left ankle in flexion. The diagram shows the arrangement. The left leg looks slightly smaller in girth than the right: 11.4 in. in circumference just below the tibial protuberance as against 12 in. This is probably due to some disuse of the left limb on account of the afore-mentioned pain. The treatment has been massage and thyroid. Whether the injury played a part in the production of the sclerodermia I cannot say.\(^1\) There is a spiralizing tendency about the distribution in this case from the outer side of the leg towards the back of the thigh, corresponding to the embryological torsion of the limb and of the nerves.\(^2\)

\(^1\) Vide Radcliffe Crocker's Atlas, Plate xlviii. A girl, aged 5, in whom the sclerodermia of right foot began on site of a scar, result of a blow.

DISCUSSION.

Dr. Eddowes: These band sclerodermias have interested me ever since I showed a case of a girl in whom one side of the face was imperfectly developed, and who had a band of scleroderma down the neck and up behind the ear. There was a history of toothache for several years, which had been treated with mustard plasters and strong liniments. I think the scleroderma was due to irritation of the teeth and damaging the growth of the parts anatomically related. I have known of other disturbances acting in much the same way: for instance, a patch of erythema remaining for a long time over the lower jaw, which I attributed to prolonged irritation from cutting a wisdom tooth. I remember, many years ago, putting up a broken leg, and doing it very carefully, yet I was surprised to find, on taking it down, that a hard oedema had developed in the leg. The tendency of such cases is to improve after a time—though some of them take years to do so.

The President: I spoke on the subject at one of the recent meetings, and of that meeting Dr. Pernet will find a record of a case I had just seen, in which there was well marked linear scleroderma affecting the left arm. The story was definitely given that it followed a blow on the back of the left hand. We discussed the matter with those concerned at the time and the conclusion in my mind was that blows are well remembered by the victim, whereas the insidious onset of a linear scleroderma does not make any impression on his mind. Parents regard a blow and the skin condition as in the relation of cause and effect, but I think they are mere coincidences. Still, it is curious that at the site of the blow, which had produced a superficial ulceration, the linear scleroderma occurred in a form which was thickened and hypertrophic, whereas the rest of the sclerodermic area, up to the shoulder, and involving part of the pectoral girdle, presented the fine, tissue-paper-like scleroderma. I am familiar with Crocker's cases, and I am interested in what Dr. Pernet now says, but I regard the blow on the site as a coincidence.

Dr. Semon: Is prognosis in this case serious, and is the condition likely to spread?

Dr. F. Parkes Weber: My impression is that linear scleroderma, on an extremity, sometimes ceases to spread when it reaches an advanced stage, as in the present case. It may remain for years without getting worse. I take it that in the present case there will be no recovery in some parts, namely, in those parts which have already become scar tissue.
Case for Diagnosis.

By S. E. Dore, M.D.

The patient is aged 34 and has suffered from the eruption almost continuously for twenty years. He has seen many dermatologists and been treated at numerous hospitals for his complaint of which various diagnoses have been made, including those of prurigo and dermatitis herpetiformis. At the present time the eruption affects chiefly his buttocks and thighs, and his arms and face slightly, but he states that no part of his body has been free. There are a few small vesicles on his hands and fingers which appear to be the initial lesions and, as the result of scratching, these are followed by excoriations and pigmented finger-nail sized patches, which are most numerous and accentuated on the trunk and lower limbs. He has an acute attack every spring, which lasts throughout the summer, the eruption being more or less quiescent in the winter. He was treated at Margate for tuberculous glands at the age of 10, but his general health is now good. He admits to a highly nervous and excitable temperament.

DISCUSSION.

Dr. F. Parkes Weber: Could not this be an atypical case of the class of so-called hydroa aestivalis? That condition gets very much worse during the hot season of the year, and it may also affect parts which are not exposed to the sun’s rays, parts which are affected in the present case.

Mr. Samuel: Dr. Parkes Weber’s suggestion is excellent, because the patient volunteered the statement to me that he has only to expose himself to the sun and immediately he gets an attack on the part exposed.

Dr. Agnes Savill: This patient came to my hospital six years ago, where the remedies for summer prurigo were tried. It was in the summer I saw him and the eruption then was more profuse than at present. His sister was suffering from the same condition.
Section of Dermatology.

President—Sir James Galloway, K.B.E., M.D.

Pigmented Hairy Mole benefited by Impetigo Contagiosa.¹

By Alfred Eddowes, M.D.

I am indebted to Dr. Steele Perkins for kindly volunteering to show the striking change for the better that a pigmented hairy mole on his ear has undergone, as the result of its becoming the seat of severe impetigo, which also affected his head, face and neck, and for which I recently treated him. Though I did not take a culture from the scab I have no doubt the case was one of typical impetigo due to streptococcic infection.

This case reminds me that many years ago I read of chronic sinuses sometimes being cured by an attack of erysipelas.

Case for Diagnosis.

By E. G. Graham Little, M.D.

I have brought this case for diagnosis: I have not yet made up my mind what it is. For the last two or three years, possibly longer, this man has had a condition of the fingers and toes which is, apparently, persistent. It is not to be confused with Raynaud’s disease, which is spasmodic. He also has swelling of the nails, a whitening of the extremities of the nails. The condition on the feet is probably associated with that on the hands. I think probably it enters into the acro-dermatis neurotica group. There are definite nerve symptoms, as he has pain and tingling of the tips of the fingers, and there is some diminished

¹ At a meeting of the Section, held May 15, 1919.
sensation. There is no clue to it in his occupation, that of jeweller, and he does not handle anything which would be likely to cause trouble in his hands.

Dr. A. Eddowes: Perhaps this is one of the "deficiency diseases" which will attract our attention more and more as time goes on. He may be requiring vitamines, of which he has been deprived during the war, especially the "antineuritic" vitamine.

Another Probable Early Case of the Miscalled Multiple Idiopathic Hæmorrhagic Sarcoma of Kaposi.1

By J. J. Pringle, M.B.

This case is undoubtedly an example of the same condition as that presented by a man shown by me to the Section on July 18, 1918, when the diagnosis I suggested received considerable support, especially from the President of our Section. The four photographs I hand round provide ample testimony to this fact when compared with those published last year. In this instance the lesions present are less abundant, but the microscopical evidence—unsatisfactory in the former case—is of a more convincing character as supplied by the sections and microscopical report furnished by Dr. MacCormac.

The patient is a man, aged 32, born in Bedfordshire, of purely British stock, who has been employed for the last seventeen years in a photographic chemical laboratory making dry plates. Nothing in the nature of his employment appears to have any bearing on the aetiology of his disease; and it is worthy of note that the hands are unaffected and that he has not got varicose veins. He came under my observation on February 25 of the present year. He had previously attended the skin department of one large general hospital and two "special" skin hospitals since July, 1913, where his case apparently did not evoke any particular interest. He states that the condition began in June, 1913, as "freckles," which subsequently became "spots," below the left internal malleolus. As these were accompanied by considerable pain in the feet and ankles he soon sought medical relief at a hospital. The "spots" next appeared below the right internal malleolus, then on the dorsum of the right foot near the toes; but he is unable to give any

definite account of the order in which the lesions subsequently manifested themselves. The pain, previously noted, ceased in the latter part of 1915, since which time his chief trouble has been due to the ulceration of the patches about his ankles, obviously of septic origin, which has readily healed up in hospital under rest and simple antiseptic treatment. The purplish vascular growths which were so prominent a feature of the previously exhibited case, are not so conspicuous in this man, although three are present on the dorsal surface of the toes. There were, however, two pad-like flat prominent plaques of soft, rather resilient, consistence on the dorsum of the right foot, which have subsided under two half-pastille doses of X-ray. The man is in excellent general health: his urine is normal; he shows no signs of cardiovascular disease.

Dr. MacCormac’s description of the microscopic appearances is as follows:

**Histological Report.**

“For the purpose of microscopical examination small pieces of tissue were removed (1) from a very early lesion in front of the right ankle where pigmentation was the predominating feature, and (2) from a situation where the disease had advanced to early tumour formation on the terminal phalanx of the second toe of the right foot.

(1) In the section of the very early lesion the epidermis was but little altered from the normal; some oedema was present, and in the basal layer an excessive deposit of pigment could be detected. These constituted the only striking abnormal features in the epidermis. In the dermis small collections of round and spindle cells were arranged around the blood-vessels. The capillaries showed some swelling of their endothelium and seemed to be present in larger numbers than usual. All through the dermis pigment was laid down usually between the cells, as linear collections of tiny granules. These deposits were also associated with the vessels, being best developed amongst the perivascular cell collections.

(2) In the more advanced condition there was some oedema of the rete giving rise to histological vesicles in places; papillation was imperfect. As in the case of the early section the most striking changes were found in the dermis. Here the capillaries were increased to such an extent as to resemble in some degree the appearances found in sections of a vascular naevus. Considerable perivascular cellular collections, similar in type but more extensive in degree to those already described,
were a prominent and striking feature. The deposit of pigment was denser and was laid down in larger masses, especially in association with the perivascular cell collections. Throughout the dermis the lymphatic spaces were dilated. Other collections of cells were grouped about the sweat glands, and here also some pigment was found.

An examination of these two sections leads to the opinion that the main appearances follow upon and are largely associated with the vascular changes. The general histological characters conform to an inflammation rather than a new growth.”

I suggest that the case and microscopic sections be submitted to the Pathological Sub-committee for report.

DISCUSSION.

Dr. MacLeod: The only way in which the nature of this case can be proved is by a microscopical examination, because the tissue of multiple idiopathic sarcoma is fairly characteristic. Six or seven years ago Dr. Parkes Weber and I had a case in which we worked out the histology, and it fitted in with that of other descriptions.

Dr. Graham Little: The case which Dr. Pringle showed in July he transferred to me, and I had him in St. Mary’s Hospital six weeks for the purposes of observation of the condition. I got a fresh section, and an admirable report on it from Dr. Kettle, our pathologist, who has had a very wide experience of tumour formations, and his report is a long and valuable document. That report I shall be pleased to hand over to Dr. Pringle, if he would like to have it. I also showed a case almost immediately after Dr. Pringle’s, and neither of those two patients was of Jewish or Polish extraction, but English, and the present patient is native-born too. At that date Dr. Parkes Weber told us there were records of only three other non-Jewish cases. That number must now be increased to six. Dr. Kettle reported that there was no suggestion of sarcomatosis in the section, but that it was a chronic inflammatory condition.

Dr. F. Parkes Weber: I did not see Dr. Pringle’s first case, though I saw a photograph of it. I think one would hesitate to include the present case under Kaposi’s so-called multiple hæmorrhagic (pigment) sarcoma, because of the curious distribution around both malleoli in both feet. I have not seen that in any typical instance of the condition. I nevertheless regard the condition as granulomatous (and Kaposi’s so-called sarcoma is likewise now supposed to be a granuloma). The peculiar fibrotic, hæmorrhagic, chronic inflammatory change with pigmentation around the ankle bones on both sides might have a connexion with tubercle, but there is no evidence of tuberculosis in the present patient.
Dr. J. H. Sequeira: I have had the opportunity of having sections cut of several cases of this kind, and the observations which Dr. MacCormac has just made fall in entirely with the reports made by Professor Bulloch and Dr. Turnbull on my own cases. Dr. MacCormac referred to an apparent vascular overgrowth, and Dr. Turnbull was of the opinion, as Dr. MacCormac is, that this is not a neoplasm, but an inflammatory increase of blood-vessels. I think we all agree that the term "sarcoma" applied to these cases is absolutely absurd. It is interesting to find that the late Sir Jonathan Hutchinson described cases identical with this as "symmetrical purple congestion of extremities," and that expresses a very important clinical feature of the condition. A Polish Jew at present under my care and whom I showed at the International Medical Congress in 1913, has this characteristic purpuric congestion of the extremities with very occasional and slight tumour formation, which is easily controlled by X-rays. I should not, at the present time, have recognized the pigmented condition behind the ankles in Dr. Pringle's case as the disease under discussion: but the history which Dr. Pringle gives, and the fact that the condition has cleared up remarkably under X-rays, with the histological picture, makes me think we should place it in that group. Perhaps the light which will be thrown on it by the Pathological Committee will help us.

Dr. H. G. Adamson: I would like to point out the resemblance of the lesions in this case to those of Schamberg's progressive pigmentary disease. The patches here seem to be made up of the same sort of punctate lesions, partly vascular and partly pigmentary, as those seen in Schamberg's disease, and Dr. MacCormac's histological description also fits in very well with that of Schamberg's disease.

Dr. Barber: I agree with Dr. Adamson about this case. I have a case under my observation at Guy's Hospital very much like it, except that so far there have been no obvious tumour formations. I think that in this condition the primary lesion is the purpuric one. My impression of the probable pathology of these cases is, that they are due to the chronic absorption of toxins of haemolytic streptococci. My patient had extremely septic tonsils, from which a pure growth of intensely haemolytic streptococci was obtained. In some cases the teeth may be the source of the trouble.

The President: After the free discussion which has taken place on this case, I agree that the reference of it to the Special Committee is desirable. In the history put before us there are two points that I would refer to. Until comparatively recently, we had the idea that we never saw true Kaposi's disease except in people from Eastern Europe. Dr. Pringle's and my experience of the condition goes back a long time, and there was one remarkable case, that of a Polish Jew, which we studied together, and he remained for a long time under our care. But during the last seven years we have had cases of this sort reported in persons who did not belong to that race, a fact which is important if the condition is the same. The second point, which I would emphasize once more, is how frequently these vascular phenomena of
inflammatory nature, possibly due to pyogenic toxins, show themselves, in the
first instance, in places such as behind the external and internal malleoli.
Those areas of skin must be regarded as areas of diminished resistance, and
both Dr. Pringle's cases showed, in the early stage, changes in those parts.

Dr. PRINGLE (in reply): I am gratified that the case has been so well
received and discussed. It is not necessary for me to say much in reply,
because the Pathological Sub-committee, to whom it will be referred, will
investigate it and report upon it fully. The only criticisms I have to meet are
those of Dr. Parkes Weber, Dr. Adamson, and Dr. Barber. Apparently
Dr. Weber has never seen cases of the condition which I suggest this to be in
the same extremely early stage: Dr. Sequeira however has, and, on the whole,
his views and mine of the case are identical. Dr. Adamson and Dr. Barber
think it is "Schamberg's disease." I pointed out, when the cases first came
under my observation, that some of the older lesions were indistinguishable
clinically from those of that rather indefinite condition. I am not aware,
however, that prominent vascular outgrowths have been met with in that
disease, and the development of the morbid process in the case shown by
outlying, discrete vascular and purpuric spots, is not consonant with the
characteristic progressive extension by continuity of the condition associated
with Schamberg's name.

The following report has been submitted on Dr. Pringle's two cases
of Kaposi's disease (?) by the Pathological Sub-committee of the
Dermatological Section of the Royal Society of Medicine:—

Case I.

Epidermis.—The epidermis is somewhat irregular, with band-like
downgrowths. In certain areas the prickle-cells are oedematous and
swollen and in places there is a tendency to intercellular vesicular
formation. The oedema has resulted in slight parakeratosis. There
is an increase in the intracellular pigment here and there in the cells in
the basal layer, in the form of granules which look like melanin.

Corium.—The vessels of the papillary and subpapillary layers are
dilated and there is oedema of the surrounding fibrous tissue. The
endothelium of certain of the capillaries is proliferated. Chiefly around
the vessels of the subpapillary layer there is a cellular infiltration
consisting of small connective tissue cells, round or oval in shape, and
a few polymorphonuclear leucocytes. There are also deposits of what
appears to be blood pigment, which is extracellular and is most marked
in the deeper layers of the corium and in the neighbourhood of the
sweat glands.
Case II.

(a) Section of Early Lesion.

_Epidermis._—The epidermis is irregular, flattened, with projections which slant sideways into the corium. The epidermal cells are swollen and oedematous in places as in Case I and there is the same excess of pigment in the cells of the basal layer.

_Corium._—The most striking feature is the collection of cells in the upper part of the corium, which are of small round or oval connective tissue type similar to fibroblasts. Collections of pigment granules are also present, especially about the sub-papillary layer and deeper down in the neighbourhood of the sweat glands.

(b) Section of Later Lesion.

The features present in the section of the early lesion are here repeated in an exaggerated degree.

_Epidermis._—The thickening, oedema of the cells, and parakeratosis, are more marked.

_Corium._—The cellular deposits are more dense, extend throughout the corium, and the dilatation of the blood-vessels is so striking as to bear some resemblance to the appearances of a vascular nevus. There is proliferation of the blood-vessel walls and deposits of what appears to be blood pigment, which is chiefly extracellular.

Summary.

The appearances vary according to the stage of the lesion, and consist simply of a growth of connective tissue cells with vascular dilatation, oedema, and a deposit of blood pigment, which would suggest an organizing connective tissue formation rather than a sarcoma or an infective granuloma. The changes in the epidermis would appear to be secondary.

These changes are similar in the main to those which occur in so-called multiple idiopathic sarcoma; they also bear some resemblance to those found by Schamberg in connexion with the disease which bears his name, but clinically the two conditions differ in that in the latter nodular or tumour formation is absent.

James Galloway, Chairman.

J. M. H. MacLeod.

Henry MacCormac.
Case of Erythrodermia with Lymphatic Leukæmia.

By J. H. Sequeira, M.D.

The patient, a man aged 64, had been shown by the exhibitor at a meeting of the Section on October 21, 1915. The provisional diagnosis made was erythrodermia with lymphocytosis, but mycosis fungoides was suspected. In the discussion on the case Dr. Parkes Weber thought the case was one of Kaposi's lymphodermia perniciosa. Dr. Adamson, who had had the patient under his care at St. Bartholomew's Hospital, believed from the biopsy he had made that the case was one of mycosis fungoides.

The patient has been under my observation at intervals since 1915 and no material change has taken place in the skin condition though the patient is evidently failing in health. He is a characteristic case of erythrodermia, the type known in the French clinics as "l'homme rouge." From the scalp to the feet the skin is of a brick red colour, with marked swelling of the extremities and some scaling of the legs. The scalp and eyebrows are denuded of hair, but the eyelashes remain. The hair of the beard, moustache, axillary and pubic regions is scanty. The spleen is not enlarged and no symptoms have been observed referable to the alimentary tract. The lungs are emphysematous, and the area of cardiac dullness is diminished. The urine was free from albumin and sugar (urea 1.1 per cent.). The temperature throughout the patient's residences in hospital was normal.

During the past few months the mental condition has been abnormal. The patient is very depressed about his condition and frequently passes his motions and urine in bed. There was never any loss of consciousness and ordinarily there is complete control of the sphincters. The soiling of the bed is that met with in certain types of mental case. The Wassermann reaction on several occasions has been completely negative. There is no history nor evidence of syphilis. The lymphatic glands in the cervical, axillary and inguinal regions are enlarged, but none exceed a walnut in size. The blood examinations (Dr. Panton) show a moderate degree of leukæmia with marked proportional increase of the lymphocytes. Counts made on April 12, 1919, and May 5, 1919:—

Red blood cells ... 3,599,000 ... 4,650,000
Hemoglobin ... 70 per cent. ... 55 per cent.
Colour index ... — ... 0·6
White blood cells ... 22,800 ... 16,000

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<td>Large hyaline cells</td>
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Improvement followed the intravenous injection of novarsenobillon. The condition of the blood has changed very little since the examinations made in 1915. It will be noticed that there is no very great increase in the number of white cells. The special feature is the large proportion of lymphocytes.

A portion of the skin was excised, but beyond showing chronic inflammatory changes there was nothing to favour the diagnosis of mycosis fungoides. A cervical gland was removed for examination, and Dr. Turnbull reported that there was definite evidence of lymphatic leukæmia.

DISCUSSION.

Dr. F. Parkes Weber: I think we should still retain the term "lymphodermia perniciosa of Kaposi" for such cases: it should be retained as a clinical term for this type of lymphatic leukæmia. When the case was previously shown, I suggested that it was one of lymphodermia perniciosa. At the beginning of such cases there are no decided leukemic blood changes, but when the skin changes are accompanied by the blood changes, the diagnosis can be easily made. Are the mediastinal glands affected in the present case? I suggest that in all such cases those glands should be examined by the Röntgen rays.

Dr. Sequeira: That has been done, and there is no change.

Dr. G. Pernot: I have seen and followed up a case of the kind, which was under the late Dr. Radcliffe-Crocker. It is probable that tumours will form later on in Dr. Sequeira's case, if the patient survive long enough. In Radcliffe-Crocker's case, the head of the patient swelled ultimately in a most alarming manner, and tumours developed in the scalp and skin of the chest. The tumours were convex and hemispherical in shape, like the bolts used for clamping girders. They were quite unlike the usual mycosis fungoides tumours.

1 For full description see Radcliffe-Crocker: "Diseases of the Skin," 1903, ii, pp. 952-3.
Dr. Graham Little: For five years I have had under observation a case very much like this. The President and Dr. Pringle saw it with me. Dr. Pringle will support the assimilation, as he suggested the diagnosis. The difficulty is, that there is nothing to point to blood abnormality, but the clinical features are very similar: there is the loss of hair, the thickening of the surface, thickening of nails, the extreme itching, and little nodular enlargements, but no tumour formation. Two years ago I diagnosed it tentatively, in consultation with the family doctor, as mycosis fungoides, but it was not entirely clear, and the absence of tumour formation made me rather hesitate to form a final opinion. I am now convinced it is the same kind of case. He has been given extensive X-ray treatment by a first-rate medical electrician, but, on the whole, this treatment made it worse. The only drug which, in my experience, has done good is arsenic, 80 minims of collosol arsenic three times a day: I was pushing it to such a degree that I feared arsenical intoxication. To some phases of the illness that made a definite difference: the skin became thinner, the itching was lessened, and the great sweating and consequent thirst were much allayed. But it was not a permanent improvement, and one had to withdraw the drug because of the reduction of the knee-jerks and other symptoms. But the patient remained in robust health the whole time.

The President: Early cases of leukaemia are rarely seen, and that is the reason why our knowledge of its natural history is scanty, and our diagnosis is apt to fail. At present I have, at Charing Cross Hospital, a case of undoubted myelogenous leukaemia, in which the patient has about 20,000 white blood cells, but the myelocytes number from 40 to 50 per cent. In general health the patient is quite well, but it is an early stage of this remarkable disease. And here, according to Dr. Sequeira, we have a remarkable condition of leukaemia with the number of leucocytes very small, but with certain other results of the disease far advanced. As already stated by Dr. Graham Little, I recently had the opportunity of seeing with him a case resembling the patient before us. How the diagnosis can be firmly made when there is not a change in the blood count, is difficult to say. Until blood changes are present, I doubt whether a diagnosis can be made.

Dr. F. Parkes Weber: In these cases of lymphodermia perniciosa the increase in the white blood cells is never very great, as far as I know.

Dr. J. H. Sequeira (in reply): I do not like the term "lymphodermia perniciosa." I cannot believe this man is making an excessive proportion of lymphocytes in his skin, and there are no lymphatic tumours in the skin.
Section of Dermatology

Case of Lichen Planus Annularis.

By J. H. Sequeira, M.D.

The patient, a single woman aged 34, stated that three years ago a few spots appeared on her arms, but they disappeared without treatment. Eighteen months ago she fell on her knees, and a few days after the fall patches came upon the front of both knees, where they had been bruised. The patches were described as starting with small discrete red spots which spread and coalesced, leaving white areas of skin between. These areas have extended, and fresh patches appeared from time to time.

The eruption has the characters of lichen planus of the annular type, the most extensive being on the extensor surface of the legs, just below the knees. Some of these are as large as a two-shilling piece, the centre of the spots being smooth and of normal skin colour, while at the margin are aggregated small shining flat-topped papules, in some parts with a small scale. On the front of the forearms, just above the wrists, are smaller areas the size of a sixpenny-piece, and others again on the upper arms. The eruption itched at first.

There is a history of a sore in the mouth, but there was no leukoplakia when the patient came under observation. The patient had scarlet fever and rheumatic fever as a child. She has always been anaemic, and suffered from menorrhagia until the uterus was curetted in 1916. In 1917 she had the vermiform appendix removed, and myomectomy was performed at the same time. The Wassermann reaction was negative.

Case of Leuconychia.

By H. C. Samuel.

The patient is a man exhibiting leuconychia of the totalis type. The nails of both hands are affected, but the left hand is the worse. The nails are unequally affected. Toe nails are normal. He is a bacon cutter at a large grocery store, and his hands are constantly soaked in the bacon brine. He has been in this trade for the last twenty-five years, and the nails have been gradually assuming their present characteristics for fifteen years. He himself assigns the condition
entirely to the salt in the bacon, in which, as I have said, the hands are constantly bathed. There is no family history of leuconychia, leucodermia, alopecia, nor any other skin disease, nor has he ever had any other skin trouble whatever.

Dr. Pernet showed a case here recently\(^1\) of a woman with leuconychia striata and totalis, and she put the appearance down to her work, which was also in the grocery department—viz., the handling of butter and margarine.

In view of Dr. Pernet’s remarks in his case, the teeth of this man have all been removed before he came under my observation, and the inference that he must have had unhealthy teeth may only be coincidence. May not the occupation of this man be a very important ætiological factor in the causation of his condition? It seems to me more than a coincidence that Dr. Pernet’s patient should also have connected her leuconychia with some chemical or physical action of the butter or margarine.

**Case of Leucodermia and Melanodermia associated with Leuconychia.**

**By H. C. Samuel.**

The patient is aged 6. The left lower costal and lumbar regions show a melanodermic sheet upon which are small leucodermic spots arranged in lines. On his nails are some small early spots of leuconychia. I have also brought his elder brother, who exhibits the early stages of leuconychia striata on several of the nails of his fingers. The father, who is not here to-day, has recently developed well-marked leucodermia and melanodermia at the angle of his lower jaw—they are not at all unlike those on the boy’s trunk, that is to say, there is a melanodermic patch with small dotted macules of leucodermia upon it. In addition the father shows some leuconychia of his fingers. The father’s brother had very well-marked leuconychia striata of all his finger nails. I am unaware that there is any association between leucodermia and leuconychia as shown by this case, although I believe it is not unknown between alopecia areata and leuconychia, and of course leucodermia, alopecia and canities are very frequently found together.

\(^1\) *Proceedings (Sect. Dermat.)*, p. 28.
So that, perhaps, there may be an indirect association between leuco-
dermia and leuconychia. I should also be interested to hear from
members whether they have ever, either in their own experience or
that of their colleagues, any record of such a marked family history of
leuconychia.

What is the pathology of leuconychia? Neither of the two cases
suggests that it is due to the presence of air in the nails, which has been
suggested as the explanation of the whiteness.

Dr. F. Parkes Weber: I do not know of any case (excepting Mr. Samuel’s)
showing the association of leucoderma with leuconychia. The leucoderma
here is of a very peculiar appearance, and it would be interesting to watch its
progress. It does not look like the commencement of ordinary vitiligo.

Case of Trichorrhexis Nodosa.

By Mrs. Addison, M.B., B.S.Lond.

The patient is aged 28. When first seen she had been using for
a year a lotion containing chloral hydrate and borax, and washing her
hair once a week. About nine months ago patient noticed that her
hair was beginning to break off short over the right side of scalp,
When first seen by the exhibitor in March, 1919, there was a patch,
about 4 in. in diameter on the right side of the scalp, of short hair, and
a similar patch, about 1 in. across, on the left side. This patch developed
later than that on the right side. The hairs were about 1½ in. to 2 in.
long, and dry and harsh looking. On close inspection whitish-grey
nodules were visible at the tips of many hairs, and also at varying
distances down the shafts. Microscopically the nodes are seen to be
typically those of trichorrhexis nodosa.

Report by Dr. Mary Schofield, Pathologist to the Royal
Free Hospital.

Report on Hairs.—“On close examination, without the aid of a
lens, small greyish-white nodules can be seen along the individual hairs
completely encircling the shaft, and invariably present at the distal end,
indicating that the shaft has broken through at one of these nodules.
Microscopical examination shows that the nodules correspond with
fusiform swellings of the hair shaft, which in the larger nodules has
split into numerous fine fibrillæ, the hair finally breaking off transversely
Case of Angioma Serpiginosum

By A. M. H. Gray, M.D.

The patient is a girl, aged 4, apparently in perfect health. There is no personal nor family history which appears to have any bearing on the present condition. At the present time she has a patch extending from...
the junction of the middle and lower thirds of the left leg, up the thigh, almost to Poupart's ligament, and occupying the front and inner aspect of the limb. The mother states that a small patch, about the size of a threepenny-bit was present at birth in the lowest part of the present patch, and that it has gradually spread upwards, and that progress still continues. On casual examination the patch seems to be composed of dilated capillary vessels, the whole presenting a reticulated appearance. On closer examination, however, the lesions will be seen to vary according to their age. In the newest part of the patch—namely, the upper edge on the thigh—the elementary lesions are seen to consist of slightly raised angular papules, best seen by reflected light. They are of the colour of the surrounding skin and occur in groups, presenting a tessellated appearance. The angular outline is obviously produced by the natural lines of the skin, as in lichen planus, which it closely resembles. Underlying these papules vascular points or small dilated vessels, which disappear on pressure, can be seen. In the region round the knee a further stage of development is noticed, the centres of the groups noted above have cleared up, leaving apparently normal skin, while surrounding each of these areas, which do not usually exceed 1 cm. in diameter, a raised margin is seen containing vascular points and dilated capillaries. If one now looks at the oldest lesions—namely, those at the lower and inner border of the large patch—these circular areas are still visible but the raised margin has now become paler and fewer vessels are present. The pale raised margin has now a keloidal appearance as though some acid had been poured irregularly over the part, but no induration can be felt on palpation.

So far no biopsy has been performed, so no account of the histology of the case can be given.

The diagnosis is by no means clear to me. In the cases of angiomatous serpiginosum which I have seen or read of the vascular lesions were all of the "cayenne-pepper grain" type and not mixed with ordinary telangiectases as they are in this case, nor have I been able to find any description of an angiomatous serpiginosum associated with the papular arrangement noted above. On the other hand this slowly spreading vascular growth with its reticular distribution has many features of the "angioma serpiginosum" cases, and is unlike the systematized vascular naevi. It is hoped to report on the case further when a histological examination has been made.
Case of Lupus Erythematosus.

By A. M. H. Gray, M.D.

The patient is a married woman, aged 25, who for the last year has been working at a shell factory as an inspector. She has had nothing to do with handling explosives, her work being limited to gauging shells.

Last year she had a nervous breakdown as a result of air raids, and to this she attributes her present condition. The affection began about six months ago with a red patch on the left hypothenar eminence; this gradually spread till it involved the areas now seen. Shortly after the appearance of the first patch a similar patch made its appearance in the same situation on the right hand and also spread till it attained its present dimensions.

The distribution of the lesions is illustrated in the diagram. The dotted areas are of pale purplish-red colour, with a more prominent reddish border and rather depressed, less red centre. Some of the lesions are very faint, and are obviously erythematous in character. They show no signs of scaling. The few patches shown in the diagram as circles with a dotted outline are areas of pale atrophic scarring without telangiectases. The areas shaded in fine lines are the most striking feature of the case. The skin in these areas, which occupy chiefly the distal extremities of the fingers on the ulnar aspect of both hands have just the appearance of an atrophy following X-ray applications. The skin is smooth and shiny: it is extremely thin and transparent; the natural ridges and furrows have disappeared and telangiectases are everywhere visible: opaque white lentil to pea-sized patches are also present but no pigmented spots. Most of the orifices of the sweat ducts have disappeared but a few still remain and the orifices of these are larger than normal. Sweat droplets can be seen exuding from them. The nails of the affected fingers are dystrophic and show marked longitudinal striation.

In addition to the lesions on the hands a patch the size of a threepenny piece can be seen on the upper lip; this has similar characters to the erythematous patches on the hands with a distinctly atrophic centre. Similar patches are present on the lobule of each ear.

The lesions on the face are so characteristic of lupus erythematosus that there can be little doubt as to the nature of the lesions on the hands. I have not been able to find any description of such extensive
Case of lupus erythematosus.
atrophy with telangiectases in this condition, and I therefore think it worthy of record. I also think it throws much light on a very interesting case of atrophy of the face with telangiectases which was shown in October, 1914,\(^1\) by Mr. H. C. Samuel, and which was thought by most Fellows at that meeting to be an X-ray burn, though there was no history of any X-ray applications. The history of the onset being associated with severe mental shock was also a prominent feature in that case.

Dr. J. J. Pringle: He would be a bold man who would make a diagnosis of lupus erythematosus on the grounds of hand condition alone, although cases I have seen would, I think, give me the necessary courage to do so. In this case I think the other lesions clinch it.

**Two Cases of Epidermolysis Bullosa Hereditaria.**

By GEORGE PERNET, M.D.

The patients are a boy, aged 7, and a girl, aged 5, brother and sister, who attended the West London Hospital recently for scattered pus lesions, but underlying this the condition of epidermolysis was noticed. The legs are mainly affected by the latter. The fingers are not involved. The mother states there are ten children in the family, and five of them are or have been affected in this way, all males except the present girl. The two eldest brothers, aged 30 and 29, are said to have "grown out of it," and are married. Another son, aged 23, is still a sufferer, and a bad one at that. According to the mother, the two eldest brothers attribute their relief to marriage, and the one aged 23 considers that marriage would cure him. The father suffered from the same condition as a boy, but he "grew out of it" too. The two children shown are the only members of the family I have seen.

**Case of Lymphangiectodes.**

By GEORGE PERNET, M.D.

The patient is a girl, aged 19, with a characteristic area of lymphangiectodes (lymphangiomatia circumscriptum) over the right hip, showing the typical vesicle-like elements in groups, but the formation is crusted in parts as a result of secondary pus infection. The case has not yet had any treatment.

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Section of Dermatology

Case of Keratoma Senile.

By H. C. Semon, M.D.

This woman is aged 62, and hyperkeratosis is by no means uncommon at her age. She has, however, suffered from the condition for the last fifteen years, and the "warts and spots" have always been worse in the spring and summer months. There are keratomata, and sessile papillomata on the face and backs of the hands. Pigmented macules and small atrophic spots with loss of elastic tissue are also to be noted. Identical lesions are met with in xeroderma pigmentosa, a condition which occurs in young children, and is frequently fatal before the age of 21 owing to the formation of epitheliomata, especially in areas exposed to sunlight. In this patient's case it will be noted that the degenerative changes are particularly active on the uncovered parts, and Dubreuilh was the first to point out that there is considerable analogy between the two types of cases.

The family history of this patient is interesting and highly significant. Her father had similar "spots" on his face and hands. Ultimately he died of cancer of the bladder. One sister died of cancer of the breast another of cancer of the breast after an operation.

The treatment of the condition is not satisfactory, as both warts and papillomata are constantly recurring. As in xeroderma pigmentosa the most reliable agent is the X-ray. The growths will also yield to CO₂ snow. For purposes of demonstration I selected the right side of the face—formerly worse than the left side—for X-ray therapy. She has had only two exposures of X-rays of one-third of a Sabouraud-Noirée pastille each, through \( \frac{1}{4} \) mm. of aluminium plate. It will be seen that the right side has now fewer lesions than the left. In connexion with radiotherapy it must be emphasized that lesions very similar or even indistinguishable from those in this case can be actually produced by prolonged and repeated, or unshielded exposures to the X-rays. This close resemblance is responsible for the fact that many dermatologists prefer the use of CO₂ snow for such cases. Dr. Norman Paul, of Sydney, in his recent admirable little text-book on the cutaneous cancers, advocates in addition the use of radium.

Dr. MacCormac: A clear distinction should be drawn between xeroderma pigmentosa and senile keratosis. In the first disease the malignant change is part of the process: in the senile changes described by Dr. Semon malignant...
degeneration is a more or less accidental feature. The suggested analogy between the condition exhibited by Dr. Semon and an X-ray dermatitis is not quite a true one. One of the most striking features in skin that has received an excessive amount of irradiation is the disappearance of the elastic tissue, which possibly accounts for the telangiectases. In the same way the "sailor skin" described by Unna is a form of dermatitis which may under certain circumstances undergo malignant degeneration, but this is not the rule. Malignant disease of the skin may be primary or follow upon some form of chronic irritation, such as senile keratosis. Such changes may occur in any situation, and are found after many different forms of chronic irritation.
Keratosis Follicularis (Darier's Disease). 1

By J. L. Bunch, M.D.

This man, aged 49, has always been a farm labourer in Gloucestershire, but he has not done much work since last Christmas. He says that the disease from which he is suffering first occurred on the chest, and then gradually spread to the rest of the trunk, to the face, and, much more recently, to the left arm: in fact his left arm has become worse during the last few months. The disease is, in my opinion, keratosis follicularis, or Darier's disease. Sections show the psorosperms which usually occur in this disease. The Wassermann reaction is negative. It is interesting that the doctor who sent this patient to me a month or two ago now writes that he has discovered that one of this man's two daughters has similar lesions on the face and head, and he promises to send me either sections or tissue from which sections may be cut: he says that, as far as he can tell, it is the same disease. I believe that heredity in Darier's disease is extraordinarily rare, and I shall be glad to know if members have had cases of this kind under their own care. This superficial ulceration has been apparently going on for some time, and there is some objectionable odour about the condition. The man's scrotum is a good deal involved, and shows a well-marked nodular condition, almost vegetating, but not of so deep a red or brownish shade as the nodules on the chest. The involvement of the face and scalp is very marked. The nodules are partly discrete, partly tending to coalesce, but showing no sign of the ulceration which is present on the chest and arms. The inguinal and axillary glands, especially the former, are enlarged.

In addition to antiseptic treatment, he has also had radiant heat baths: he has not been treated with X-rays, nor had injections of drugs, such as "606," nor mercury. I shall be glad if anyone can suggest a remedy which in their experience is efficacious in this disease.

1 At a meeting of the Section, held June 19, 1919.
Dr. J. J. Pringle: The first cases of this disease in Europe were shown and discussed by Darier at the First International Congress of Dermatology in Paris, in 1889. The condition was described independently by J. C. White, of Boston, U.S.A., in the same year under the name of "keratosis follicularis." Darier described coccidia-like bodies, which he called "psorosperms," but which have since been proved to be simply epithelial cell inclusions. I have never
had a well-defined case under my care, although cases have been referred to me in which the diagnosis was suspected. I think in many instances the disease begins in the groins, regions which are conspicuously involved in the patient exhibited.

Dr. Arthur Whitfield: The only case of the kind I have had is now under my care, a private patient. This patient gives a negative Wassermann, which is what one would expect. I have tried various things on my patient. X-rays are useless, so is blue light. Salvarsan was not given. He had been arsenicated pretty heavily before I saw him, but it had had no good effect. I
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tried a series of local applications of salicylic acid, which is what is recommended in the textbooks but I do not think it does much good even temporarily. Then I tried two other things. One was liquid extract of thuya, which has a reputation for removing warty growths of the skin. It failed in this case however. What I believe did him most good was painting with a 1 in 1,000 solution of mercuric iodide—the biniodide dissolved in potassium iodide. I tried it because the late Dr. Colcott Fox told me that many cases of these multiple flat warts in children went away after being painted for some time with biniodide of mercury. There is no doubt it subdues them, and some go right away, but fresh growths appear. It is a very early case. I took him into a nursing home and dug them out with a tiny spoon and cauterized the part with zinc chloride. They did not recur, but the fact that he had fresh ones rendered the treatment rather hopeless. His own suggestion is that I should take him in once a year and clear him up. He now has rape-seed sized bodies scattered about, some are on the face and they hurt him when he shaves. The disease is so early that one might doubt the diagnosis, but the histology is characteristic.

The President: Dr. Pringle may remember a case which we suspected long ago, when I was clinical assistant to Sir Stephen Mackenzie, a case attending the London Hospital. I made the microscopic examination of the lesions, and the sections were shown at the Dermatological Society of London. At that time there was an influx of new knowledge about cell inclusions in cancer and psorospermosis, and these bodies were easily identified with the cell inclusions of degenerating epithelium. I recollect that in Sir Stephen Mackenzie’s case salicylic acid was used with some advantage. There was a good deal of the eruption about the axillary folds, which caused some discomfort, and the salicylic acid seemed to have a flattening and comforting effect. I can imagine that treatment with X-rays would not be a very advisable proceeding. These cases are very rare, and I am glad we have had an opportunity of seeing this patient.

Dr. Bunch (in reply): The sections show clearly the so-called psorosperms, or degenerated epithelial cells, thickening of the stratum corneum and blocking of the hair and sebaceous follicles with horny material. There is some infiltration of small cells in the deeper layers of the epidermis.

Case of Delhi Boil.

By Henry MacCormac, C.B.E., M.D.

The patient, a man, aged 42, developed the first lesion on the left cheek in 1916 and a second on the left hand in June, 1917, while on service in Mesopotamia. A third sore appeared on the right upper arm in 1919 after his return to England. The lesions on the face and hand are about the size of a half-crown, and show central ulceration and
crusting with a surrounding infiltrated and erythematous area. The skin is considerably undermined and a smear prepared from the discharge showed numerous examples of the causal parasite, *Leishmania tropica*, the discovery of which is a necessary step in establishing the diagnosis, since many instances of tropical ulceration clinically similar to Delhi boil arise from other forms of infection. The third lesion is in an early state of evolution and consists of an indurated elevation the size of a split-pea; ulceration has not yet occurred. The disease appears to cause very little discomfort to the patient except just before ulceration begins, when considerable pain is experienced.

As this disease will probably be seen more frequently in the British Isles in the future, owing to the return of infected soldiers from the East, and as the forms of treatment hitherto used have on the whole proved unsatisfactory, it may be of interest to briefly describe a method employed by Mr. Lyster. This consists of ionization with a solution of sodium hypochlorite, only a feeble current being used, not more than 1 ma. to the square inch. By this means rapid healing is brought about. Mr. Lyster has now treated six cases in this way with good results.

**DISCUSSION.**

The President: The diagnosis, I think, can hardly be in doubt, in view of the microscopical preparations which are here for us to see. I recollect seeing a case treated many years ago by excision of the whole of the involved area under a general anaesthetic. It would be a good thing if we could get satisfactory results from the method of treatment which has been mentioned.

Dr. J. J. Pringle: I recently had a case of this kind at a military hospital to which I am attached, in which six of these sores were successfully excised, the incisions being made wide of the lesions, and four or five months afterwards the result was quite satisfactory. I agree that it would be of great advantage if the method of treatment advocated by Mr. Lyster turns out to be successful, as the cure of the disease, if left to itself or treated in the customary manner by scraping and caustics is extremely slow. At the same military hospital I have seen a case with extensive and deep ulceration of the abdominal wall, which I at first took to be tertiary syphilis, but the Wassermann test was negative, and the Leishman-Donovan bodies were easily demonstrated. In February, 1914, a lady consulted me with two sores of this nature on the back of the left forearm, singularly like primary syphilitic sores, but without induration. They were said to be of about a fortnight's duration. Reference to my notes enables me to report her statement, that during a recent yachting cruise in the Mediterranean she had only once been ashore, and that for half an hour in October at Tunis, where "she was sure she was bitten."
Dr. Vincent Dickinson: During the last four or five years Italian contemporary medical literature has been full of this subject, particularly its relation to kala-azar, and its treatment by antimony. Dr. Cannata and Dr. Caronia published large numbers of cases in which beneficial results were obtained by intravenous and intramuscular injections of antimony. When the drug was used crude, the effect of intramuscular use seemed to be very painful and untoward results ensued. One of these observers succeeded in formulating an organic preparation of antimony, acetylaminophenyl stibiate of sodium, which, he claimed, was quite innocuous and painless. I have not recently seen reports in Italian papers about the treatment by hyperchlorites, but observers in Italy seem to be very pleased with the results they have achieved with antimony, not only in the external manifestations in kala-azar but in internal Leishmaniasis as well.

Dr. Haldin Davis: In the official communications given to medical officers as to how to treat these diseases in the East, mention is made of treatment by intravenous injection and antimony, but antimony was not provided in the hospitals. I saw many cases of this condition out in Egypt, and in most cases they got well after scraping and cauterization with carbolic. I think the chronicity and obstinacy must vary a good deal in various patients. We have an exaggerated idea as to how obstinate they are, because here we only get those which are most resistant. At St. Bartholomew's Hospital recently we had a case in a nurse, and she got well after the use of X-rays.

Dr. A. Whitfield: Dr. MacCormac suggested that these cases might become fairly common in this country; does he therefore think that the subjects of it will spread it here, or does he mean that we shall have a good many cases back from tropical regions? As far as I know, cases have not been described as occurring unless in particular regions. And Dr. MacCormac suggested that the lesion on the arm was very likely inoculated by the patient himself; but I had an idea that inoculation was the result of bites of flies. That may explain Dr. Pringle's case in which the lady was such a short time exposed. I have not found any information forthcoming as to the incubation period of this disease, whether short or long, regular or variable. Can Dr. MacCormac tell us?

Dr. MacCormac (in reply): With regard to the antimony treatment of this condition, Dr. Low informs me that he has obtained some successful results with this method, but it would appear to act more beneficially in kala-azar, a disease caused by a closely allied parasite. Dr. Davis, I understand, thinks the chronic nature has been exaggerated. As my own experience has been limited to a few cases I am unable to speak on this point, but in all that came under my observation the disease had been in existence for a considerable time, and had proved extremely rebellious to all the remedies employed. It is true that the sores eventually get well, but as they may spread widely before doing so, it is obviously desirable to control the morbid process as soon as possible. Delhi boil will probably become more common
in this country because of the large number of soldiers infected with this complaint while serving in the East, many of whom will shortly be returning home. There is no means of telling whether these men can infect individuals in this country or not. Apparently where the disease is endemic inoculation may occur either directly, or through the intermediary of flies, but one or more of the necessary factors may be wanting in the British Isles. The incubation period appears to be uncertain, and statements on this point are of an indefinite nature.

Case of Dermatitis Herpetiformis.

By Alfred Eddowes, M.D.

This boy has suffered from the eruption for two years. Last winter it did not disappear, but during the previous winter it was very much better. He complains of intense itching, especially at times. When I first saw the patient, and observed these pigmented nodules with very little sign of scratching, I thought it was urticaria pigmentosa. But on reflection and watching the case a little longer, I concluded that it was probably dermatitis herpetiformis. There is a very symmetrical distribution, and the lesions do not occupy the common sites: the patches are mainly over the sternum and clavicle and below the umbilicus. The fact that the lesions are chiefly papular and nodular is against the case being one of dermatitis herpetiformis. I have not seen vesicles or bullae so far.

DISCUSSION.

Dr. A. Whitfield: When I saw this case I concluded that the boy had an itchy skin with, possibly, an occasional papular urticarial lesion, which he dug out: that is, it was the type of disease which we class as neurotic excoriations. I should be inclined to give him, for some time, syrup of calcium lactophosphate, which is much more efficacious than calcium lactate or chloride; also it is extremely palatable, which the other drugs are not. He might have a local application of liquor carbonis, or something of that sort. I think he has pruritus of internal origin, probably kept up by scratching, though the habit, apparently, is also very strong. If we can get the itching to stop, the scratching habit may die out.

Dr. J. J. Pringle: Dr. Eddowes has not advanced the diagnosis of dermatitis herpetiformis with any great conviction and I think his hesitation is fully justified. Neither the elementary lesions, their grouping, nor their general characters bespeak that disease. I think the case is an undoubted example of a common enough condition bearing the rather unsatisfactory name of "acne urticata," which usually occurs in neurotic women. The lesions are
deep-seated lumps into which they dig their nails with extreme satisfaction to themselves, thereby producing so-called neurotic excoriations. With regard to the treatment of the condition, I am glad to hear what Dr. Whitfield says: it gives one hope for greater success in the future than in the past.

Dr. Stowers: I appreciate the conditions which led Dr. Eddowes to diagnose this case as one of dermatitis herpetiformis, but on learning the full particulars of history and mode of onset I fail to be convinced that his view is well supported. The frequency with which we see prolonged cases of so-called lichen urticatus, and its allies, in young children, affords us the opportunity of noting the later and often very chronic manifestations associated with the itching habit, the chronic pruriginous class of neurotic origin leading to excoriations, of which this is probably an instance. Dr. Whitfield’s suggestion as to treatment is one we should bear in mind and test for ourselves as it tends to simplify our methods which are often complicated and troublesome. The view expressed by a member that the patient is the subject of a cutaneous tuberculide can be excluded, I think, unless the general conditions and a microscopic examination of sections support it.

The President: So far as the histology is concerned, unless the lesions are very characteristically those of urticaria pigmentosa, we shall not get much advantage from examining sections. I should agree with the diagnosis suggested by Dr. Pringle and Dr. Whitfield.

Dr. Whitfield: The treatment I mentioned does not originate with me: it is a modification of Wright’s treatment. In lichen urticatus it is very much more specific than in any other form of urticaria in children. Recently I have been treating the child of a well-known bio-chemist in London, and he said it seemed as specific as the giving of yeast extract to a polyneuritic pigeon: therefore that testimonial is not that of a lay opinion but of an exact observer. It is one of the great characteristics that Colcott Fox pointed out in his work on the subject that factitious urticaria is very rarely present in lichen urticatus, and he made a great point of distinguishing lichen urticatus from the true and very rare urticaria of children: he said the lichen urticatus was not identical with urticaria though probably allied to it.

Dr. Eddowes (in reply): With regard to the suggestion that this might be a case of acne urticata. That condition generally leaves scars, with the open mouths of cicatrized follicles. Here the healing is flat. The fact that one cannot produce swelling by friction is against the case being one of urticaria pigmentosa or any other kind of urticaria. The lesions start as little erythematous nodules which I think more closely resemble the scattered lesions which one finds in dermatitis herpetiformis. I shall be glad to act on the suggestion as to treatment which has been made; and if I can watch the boy long enough, I shall be pleased to bring him again. I have made no special inquiry as to tuberculosis, but the family appears to be fairly healthy.
Section of Electro-Therapeutics.

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The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.
Section of Electro-Therapeutics.

President—Mr. Cecil R. C. Lyster.

Stereoscopic Radiography.

By the late Sir James Mackenzie Davidson, M.B., C.M.

To-night I am bringing to your notice by practical illustration certain points in X-ray work which at this time seem to me of some importance. Stereoscopic radiography has proved itself of immense value, but it still appears that many workers approach the subject as if it were a difficult or complicated process. Although I am advancing no new facts this evening, I wish to give one or two demonstrations which may be helpful in elucidating the really simple principles upon which stereoscopy is based.

This subject has two aspects: (1) The condition of the vision of the observer; (2) the proper preparation, placing, and viewing of the X-ray photographs. As to the first, the individual to see stereoscopic pictures correctly must possess binocular vision. By that is meant that each eye is able to see the two stereoscopic pictures and obtain therefrom a single combined impression on the brain. It does not follow that he must necessarily be able to see equally well with each eye, because people who have distinctly unequal vision may yet have a good binocular capacity. On the other hand, it may happen that an individual has excellent vision in each eye and yet fails to get a combined impression—i.e., he can only see with one eye at a time. It appears to me that there is also a third condition of interference, though a little difficult to determine, in which people in full possession of binocular vision, and able to see ordinary stereoscopic pictures of landscapes and the like quite correctly, yet for a time have considerable

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1 At a meeting of the Section, held October 18, 1918.
difficulty in seeing stereoscopic X-ray pictures in their proper relief. But with these individuals the difficulty is completely removed by a little practice in viewing. In work of this kind all these points are of considerable importance, because they explain the fact that many individuals do not know what we are talking about when we speak of the vivid relief of the stereoscopic picture; and it is a matter of very great importance to find out whether the X-ray worker and also the surgeon who works with him are in possession of correct binocular vision. To those who can see the stereoscopic radiographs properly, the immense importance of the stereoscopic picture is obvious, and requires no special elaboration, and there can be no doubt that the comparatively slow progress of this important branch of work has been delayed and hindered by the fact that many workers are quite unaware that they do not possess proper binocular capacity. The ophthalmic aspect of the question could, of course, be very much more fully dealt with, but I have only wished to indicate the great importance of ascertaining the condition of the worker's powers of vision.

The second aspect of the subject has to do with the pictures themselves. Many writers on stereoscopic radiography, it seems to me, complicate the subject by directions as to varying the displacement of the tube with its distance from the plate, and also with the thickness of the part to be radiographed. For many years I have used a displacement of the tube of 6 cm., which may be taken as the approximate interocular distance—i.e., the distance between the pupil centres when the eyes are looking at an object about 10 in. to 15 in. distant. I have found no difficulty in these conditions in getting a correctly proportioned picture in the stereoscope.

I will now proceed to show a very simple arrangement which will illustrate the production of a stereoscopic picture. I have here two little electric lamps, each of them enclosed in a small square box with a coloured screen in front. In the one case it is a red screen and in the other a green. I have also a small translucent screen supported in a wooden framework upon which shadows of objects can be cast by these two lamps; and here is a small wire device with a circular base and a smaller circle at its apex so that it forms a skeleton cone. When this is held in front of the screen so that the larger circle of the base rests actually upon the screen and the smaller circle is at a distance of some inches, a shadow of this cone can be cast by the light, and the appearance of the shadow will entirely depend upon the relative position of the light which produces it. If the lamp is held opposite the axis of
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this skeleton cone two circles will be seen on the screen, the shadow of the base circle, and, in the centre of it, the shadow of the smaller circle of the apex. If the light producing the shadow is moved to the right or to the left the shadow of the base which rests in contact with the screen will maintain its position, but the shadow of the small circle will be shifted to the side opposite to that to which the light has been moved. If the two lights are placed side by side so that the mid-distance between them is opposite the centre of the axis of this wire cone, two shadows of the cone will be seen, one coloured red and the other green, and one might at first interpret that by saying that the red shadow was cast by the red light and the green shadow by the green light. As a matter of actual fact, the shadow that appears red is the one that is cast by the green light, and the shadow that appears green is the one produced by the red. At first one is apt to overlook this, but it is quite obvious that the green light will illuminate the whole of the screen except where the shadow of the little circle is produced, and in that shadow there will be no green light; the red light, on the other hand, will illuminate the whole of the screen, and will mingle with the green except where the dark shadow is produced by the green light, and this will, of course, be illuminated by the red lamp and will look red, but nevertheless the shadow is really produced, as is evident, by the green light. I may have dwelt upon this little point tediously, but it has a very definite bearing upon the theory of the stereoscopic picture.

I have here a large number of spectacles which have been very simply made by piercing two eye-holes in a piece of black cardboard and inserting red and green gelatine respectively, so that when one of these spectacles is held before the eyes one eye looks through the red gelatine and the other through the green. If the shadow of the cone on the screen be viewed in a suitable position with a pair of these spectacles held in front of the eyes, each eye will see its own image: the red will enable the eye which it covers to see the red shadows plainly, whereas it will neutralize the green, which will be comparatively invisible to that eye. The other eye, which is covered by the green, will only see the green shadows and not the red. The result will be a vivid perception of perspective, and if the experiment is properly arranged the shadow of the cone will be seen in true proportion; in fact it will appear as if one were looking at the cone itself instead of only at its shadows. It is possible in a short time to learn and to teach all the variations in the size and shape of the stereoscopic image

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by varying the distance of the observer from the screen, or by altering the displacement of the lamps. One can learn more in a few minutes by practical experiment on these lines than by much reading about theory.

The results obtained with these shadows by ordinary light are exactly what would be obtained with X-rays if two X-ray tubes were substituted for the little lamps, and this little apparatus helps one to realize practically the principles upon which a stereoscopic fluoroscope depends. The day will come when all important X-ray examinations will be conducted by stereoscopic fluoroscopy. Unfortunately, with X-rays, we have no simple means of enabling each eye to see its own image. In the apparatus with the coloured glass the eclipsing of each colour by the other is a very simple arrangement, but with X-ray apparatus an expedient that will give a similar result can only be obtained meantime by some revolving shutter. At the present time the Americans have a stereoscopic fluoroscope which, from what I have gathered of its principles—I have not actually seen the mechanism—ought to be a success. Anyone who possesses a Snook type of machine and two of the new rectifying Coolidge tubes only has to have some reliable synchronous motor operating an eclipsing shutter to get at once a means of producing the stereoscopic picture on the fluorescent screen.

I would strongly urge the younger workers present to devote their energies to the construction of this type of apparatus. It would well repay their efforts. About twenty years ago I constructed a stereoscopic fluoroscope with great difficulty with the ordinary coil and tubes and commutators. It gave a good stereoscopic picture on the screen, and I demonstrated its action in the X-ray Department at Charing Cross Hospital. The difficulty of carrying it out with the tubes and commutators then in use prevented its further development, but it seemed to me that the problem with modern development is very much simplified, and it only remains for someone to construct an apparatus such as I have suggested in order to bring the matter into practical use. It is very remarkable to observe the shadows standing out in vivid relief. Any part of the object that can be reached may be touched with the utmost precision; if a bullet is in the sinus of a wound a probe can be put on to it with the same ease as if one were looking at the object in the ordinary way. In the case of the chest, the movements of the ribs and diaphragm in breathing and the beating of the heart inside the ribs, make a study
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which, once seen, can never be forgotten. In 1898 I demonstrated this in the X-ray Department at Charing Cross Hospital. I used to place bullets in a large wooden box or bag full of sugar, and the students, when viewing the object on the stereoscopic fluorescent screen could seize the bullet with a pair of forceps and pull it out every time.

One point of very great importance in producing correct stereoscopic photographs, which if not realized may lead to serious error, is that, upon the displacement of the tube to produce the second image, the most complete immobility possible in the part being photographed must be maintained. If this condition is not observed an erroneous stereoscopic picture is produced. To take a common example, if a lead mark is put upon the skin of the chest and a stereoscopic picture is taken, unless at each exposure the breath has been held in exactly the same phase, the resulting picture when viewed in the stereoscope will often show the ribs in wrong positions, and at times the lead mark placed upon the skin will give the most realistic impression of being inside the chest. This is entirely due to a misleading parallax produced by the independent motion of the part being photographed. An easy way of illustrating this convincingly is to take a photograph of a hand lying flat on the table, and between the two exposures slightly to move one of the fingers. The two resulting negatives on casual inspection will appear to be similar, but, nevertheless, when viewed in the stereoscope it will be seen that the hand and the fingers other than the finger that moved, give a correct impression of being on the same plane, but that the finger which was moved, although the movement was almost imperceptible, appears bent forward and in advance of the other fingers—an impossible plane, because all the fingers were resting on the table.

This brings me to the physiological optics of stereoscopic vision. Why is it that we see an object in a certain position in the stereoscope, and when the negatives are changed over to opposite sides, see the picture the other way round? In the development of his visual apparatus from infancy the individual discovers that when an object is near he has to converge his eyes to see it, and when it is farther away he has to diverge. Therefore the convergence and divergence of the eyes become associated respectively with near and distant objects. In the simple apparatus I have already brought before you it will be observed, when the spectacles are held in front of the eyes to see the coloured shadows in relief, that this principle of convergence and divergence goes to explain how the cone is seen, whether with its small end towards the observer or away from him. I have here some diagrams
which represent the shadows of the cone, and it will be found that in the case where the distance between the centres of the larger circles is greater than the distance between the centres of the smaller circles, the right eye seeing the image on the right side and the left eye the image on the left side, the stereoscopic picture will have the effect of bringing the small end of the cone nearer to the observer, and putting the base farther away. If, now, the diagrams be transposed, and the right and left occupy each other's positions, so that the distance between the centres of the larger circles is less than the distance between the centres of the smaller circles, and be so viewed that each eye sees its own image, the cone will appear reversed, the base being nearer the observer and the apex farther away. It is a matter of the convergence and divergence of the eyes in order to take in the corresponding points of the diagram, and the principles which are so easily demonstrated in a line drawing apply equally to the hundreds of corresponding points in the pair of X-ray photographs.

I will now endeavour by means of two lanterns and the coloured screens to demonstrate to you on the viewing screen some stereoscopic photographs which, if you will view them with the coloured spectacles supplied for the purpose, will appear to you in relief. In all stereoscopic pictures, especially on the reduced slides now so commonly used, it is discoverable with a fine pair of compasses put on to the various corresponding points of the two pictures that the distances between them differ quite appreciably, and it can be worked out by means of these compasses which points in the picture will appear nearer the observer when viewed in the stereoscope and which more remote.

This brings us to the practical application: how are we to proceed to take and view stereoscopic radiographs so as to get the true effect? The distance of the tube displacement being kept constant at 6 cm., the angle of view will be the same if the screen is placed midway between the observer and the tubes. Therefore, in the Wheatstone stereoscope the distance of the picture from the centre of the mirror ought to be the same as the distance of the tube from the photographic plate when the picture was taken. It will be quite obvious, then, that stereoscopic radiography is just as simple as ordinary radiography. The point of view having been decided upon, a single picture can be taken, and then the displacement of 6 cm. made, parallel to the edge of the plate, and the second picture taken; these two negatives, viewed by any means
which enables each eye to see one picture at a time, will give the effect of true stereoscopic relief. The most convenient method is to reduce the negatives to lantern size and so mount them that they can be viewed in a lenticular stereoscope. This gives excellent detail, and a vivid stereoscopic picture which can be used by the surgeon, even while he is operating. It is a simple matter to arrange for an assistant to hold up the lenticular stereoscope and for the surgeon to look through it during operation. This has the additional advantage that if the picture is required in a hurry, the negatives while wet can be put in the reducing apparatus, and stereoscopic slides produced in a very short time. It is a method that I use myself and strongly recommend in cases where stereoscopic pictures are required.

So far we have dealt with a method of triangulation that the human eye and brain can carry out. But if measurements are to be given of the position of foreign bodies the triangulation has to be done mechanically. In order to do this quickly and accurately, I have devised an apparatus which I now wish to demonstrate to you. The principles upon which it is based are exceedingly simple and well-known. The whole object I had in view was to apply these principles in such a simple way that in this time of stress and strain when skilled workers are few and the opportunity to train men is brief, comparatively unskilled people would be able to use the apparatus and produce accurate results for the surgeon.

The tube is held in position in a well protected box, and arranged to be vertically below the point at which two rectangular wires intersect. The arrangement enables this position to be fixed, and yet the tube and the cross wires can be moved in synchronism vertically up and down in the usual way so as to occupy any desired position in relation to the couch below. With the screen above the wires, and the tube lighted up, the arrangement is moved about over the patient and adjusted so that the tube finally occupies such a position that the shadow of any foreign body appears in some definite relation to the cross wires—say that the point or the base of a bullet is made to touch the intersection. When this is done it follows that this given part of the projectile is vertically below the cross wires, and at that point the skin is marked. Then, by means of a travelling clamp which can be fixed with the pressure of the foot and the turn of a T-shaped handle, the box and the fluorescent screen with its wires are displaced to the left to the distance of 6 cm. The shadow of the foreign body will in that case, of course, be displaced to the right to an extent which varies according
to the depth at which the foreign body is lying. A small travelling wire can now be moved by means of a screw into precisely the same relation to the foreign body as when it was centred, and the little indicator attached to the travelling wire will enable one to read off on a calibrated scale the depth of the bullet below the cross wires. I do not know of any more accurate or speedy way of screen localization than this. If photography is desired, the horizontal arm carrying the screen and cross wires, which is hinged, can be moved aside, and a small indicator put on the marked spot on the skin; the plate is then laid on the part, and stereoscopic photographs are taken in the usual way; or the cross wires can be laid on the skin, with their intersection at the marked spot, the displacement carried out, and the localization effected as ordinarily. This apparatus is arranged to work under the couch, either with the usual top or a stretcher top. If other positions are desired, a separate apparatus ought to be used. Attempts at producing apparatus to do every kind of work are, I think, fatal mistakes.

I hope that the demonstration I have had the honour of giving you to-night may be of some assistance in furthering the adoption of methods of proper education in X-ray work.
The Examination of the Vermiform Appendix by X-rays.

By E. I. Spriggs, M.D., F.R.C.P.
(Senior Physician, Duff House),

With Photographs and Drawings by O. A. Marxer
(Radiographer, Duff House).

(ABSTRACT.)

[For the paper in full see the Lancet, January 18, 1919, pp. 91-97.]

After a summary of the literature of appendix radiography, in which our indebtedness to American workers was acknowledged, the authors described the methods which they had found of value. The patient is prepared with castor oil and given a meal, similar to that recommended by George and Gerber, consisting of three quarters of a pint of buttermilk and 150 grm. or less of barium sulphate. Careful screening, with suitable manipulation, is needed to observe the mobility of the appendix and the presence or absence of active movements or of tenderness. Photographs must also be taken freely, as the chief points which help to decide whether the appendix is healthy or diseased —namely, the filling and emptying of the appendix, its position, and its outline, are observed best on the photographic plates. The manipulations which had been found most useful were detailed.

The following figures were given by the authors: The first hundred

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1 At a meeting of the Section, held November 15, 1918.
cases in which they observed the passage of an opaque meal through the alimentary canal were left out of account, as the ileo-cecal region was not examined with such care as is now given it. Of the next two hundred cases with a standard meal, in many of which castor oil was also used, the appendix was seen clearly in a few. They then began to use the barium and buttermilk meal after preparation by castor oil. In the fourth hundred cases the appendix was seen twenty-four times; in the fifth hundred, thirty-five times; then fifty-four times; then seventy-two times; and in the eighth and last hundred, eighty-six times.

An account was next given of the radiography of the normal appendix and photographs were thrown on the screen.

The paper then described the examination of cases of appendicitis. In acute appendicitis the patient is not usually fit to be X-rayed, neither is such a method of diagnosis needed. In chronic appendicitis the authors have found X-ray examination of great value, especially in the subjects of vague abdominal symptoms of unknown cause. In thirty-six cases a diagnosis of chronic appendicitis had been made and confirmed by operation. Radiograms of many of these were shown, accompanied by coloured drawings of the appendices after removal.

The chief points to which attention must be paid are: (1) The filling and emptying of the appendix; delay. (2) Shape: constriction and dilatation. (3) Fæcal concretions: vacuoles. (4) Mobility. (5) Hyperactivity: spasm. (6) Tenderness. (7) Position. These features are placed in the order of their importance in a series of cases. Each was illustrated by lantern slides and clinical details.

The signs of present inflammation are, in addition to pain and other clinical symptoms: a tender point (though tenderness requires care in its interpretation) and varying dilatation of the lumen from hyperactivity and spasm; whilst evidence of former disease, recent or remote, is given by concretions, abnormal outline, delay in filling or emptying, adhesions, severe kinks, and in certain cases at least by the absence of a shadow. The proportion of cases in which no barium sulphate enters the appendix is small when the methods described are used, but the authors do not think it justifiable in the present state of our knowledge to assume that an appendix is abnormal because it does not fill, though they would regard it with suspicion, especially if the observation were repeated.

The paper was founded on the study of the photographs of about 300 appendices.
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Dr. A. C. Jordan: Dr. Spriggs has drawn attention to the fact that the normal appendix is much greater in length in the living subject than it appears after removal, and his pictures certainly bear out his statement. With regard to "tenderness" of the appendix, one is able, in many cases, to elicit pain by pressure upon the appendix, but some authorities hold the view that this pain is, in reality, a reflex phenomenon, and that no conclusive experimental evidence exists to prove that the visera are themselves susceptible of sensation. With reference to Dr. Spriggs's remarks on the subject of ileal stasis, I would go even farther than he. In the normal subject we can give a definite time-table of the progress of an opaque meal—two or three hours for the stomach to empty, five or six hours for the small intestine, and so on. As soon as stasis begins, however, and delay occurs in the lower ileal coils, duodenal distension and pyloric spasm are produced. Consequently the time-table is upset. The gastric contents escape slowly, and reach the lower ileum in small quantities. If there were no delay here, these small amounts would enter the cecum at once, leaving the lower ileal coils nearly empty. Instead of this we generally find a large accumulation of bismuth in the lower ileal coils, while there is still bismuth in the stomach. This I regard as a certain proof of ileal stasis. I have brought a few lantern slides. The first was mentioned by Dr. Spriggs when he was good enough to refer to mine as the first X-ray picture of the appendix to be published in this country. It appeared in the Archives of the Roentgen Ray for January, 1911. The next two illustrate ileal stasis due to fixation of the proximal portion of the appendix; the terminal coil of the ileum and the appendix are shown kinked by the same band. The next three illustrate cases in which the appendix can be proved, by careful manual examination with the fluorescent screen, to be producing ileal stasis in a similar manner, although the appendix itself does not appear on the skiagrams, its lumen being obstructed or obliterated. In each of the five cases shown the fixation of the appendix has led to ulceration in the duodenum. Although "appendicitis" is not to be regarded as a disease in itself, there can be no doubt that Dr. Spriggs's seven headings for determining diseases of the appendix will be found of the greatest value in forming a diagnosis.
Personal Experiences of Burning caused by Secondary Radiation.¹

By James Metcalfe, M.D.

The effect of secondary X-ray radiation on the tissues so far does not appear to have been thoroughly demonstrated. The first suggestion for the use of secondary radiation as a therapeutic agent was made in a paper read before the Section of Electrology and Radiology of the British Medical Association in 1910 by Sir J. J. Thomson. He presupposed that the effect desired from Röntgen radiation was only to be obtained from the very softest kind of X-rays. Hard rays he judged were only of value in making radiograms. They were not absorbed by the tissues, so had no physiological or therapeutical action. This we now know is a fallacy. Professor C. G. Barkla made further experiments on the subject which were published in the Journal of the Röntgen Society for October, 1911. He found that when Röntgen rays struck any metal the secondary rays emitted were of a specific quality peculiar to the substance bombarded. It does not matter what the quality of the X-rays used is, as the radiation produced is of the same type. Hard rays or medium rays all act alike, the only point to observe being that the incident radiation must be harder than the radiation characteristic of the body used. A soft radiation for instance will not excite the radiation of a metal of high atomic weight such as lead. The whole point of the paper was intended to demonstrate the fact that the softness or hardness of the ray emitted from the metal was simply a question of

¹ At a meeting of the Section, held at Guy's Hospital, December 20, 1918.
atomic weight—the hardness of the ray increasing with the atomic weight of the metal. Iron rays would be absorbed within a one-hundredth of a millimetre from the surface of the skin; copper a little deeper; silver rays would go much deeper. Tin of higher atomic weight than silver would give more penetration. Iodine would be a still more powerful penetrant.

Dr. Hernaman-Johnson read an exhaustive paper on this subject of secondary radiation before our Section, on February 16, 1912, and another paper dealing with the same subject before the Röntgen Society on May 5, 1913. Dr. Hernaman-Johnson made many interesting experiments in connexion with secondary radiation. He demonstrated their utility in several cases of disease which he recorded. But at a later period he came to the conclusion that his results, with the exception of silver and one or two other radiations, were not due to secondary radiation at all. This he assumed because he found that if he exposed a sensitive plate to the action of these secondary rays a trivial obstacle to their penetration—such as the interposition of a piece of notepaper—cut off all action produced on the plate. The only photographic result obtained was due to primary scattered radiations of the same quality as those resulting from the exposure to the hard X-ray tubes used.

Dr. Hernaman-Johnson suggested certain new technical terms for the various types of secondary rays. Anadrastic rays would be those secondaries acting in a direction opposite to that of the primary beam: those acting in the same direction he termed "syndrastic," and those acting on scattered particles of the metallic substance in the tissues in all directions "pandrastic." However, the only type of any practical importance is the anadrastic—acting in the opposite direction to the primary beam.

I will now give the result of secondary X-ray radiation induced unwittingly on myself in the course of my work. I speak of the condition advisedly as "burning," not as dermatitis, as the appearance and symptoms were all those of a specific burn. Some months ago I felt some irritation on the back of my left wrist. After a short time a small spot formed which extended until it became the size of a threepenny-piece. I was living in the country at the time, and as there were innumerable small insects in the garden that were continually producing similar spots on the skin in other members of my family, I thought that I had been bitten by one of them. I removed a wrist bracelet watch I wore on my left wrist and placed it on the right
one. The little sore on the back of my wrist was quite round, very inflamed, discharged a copious serum, and gradually extended its circumference. I dressed it with a simple ointment and rested my arm as much as possible. It became the size of a shilling-piece, and eventually as large as a half-crown. By this time I had quite forgotten the fact that I had worn a silver bracelet watch on this wrist previously, and could not conceive why the burn extended so steadily. I attributed it to septic infection, and several friends to whom I showed it also thought this the probable cause. Dr. Hernaman-Johnson applied the tungsten arc lamp to it, but this really inflamed it more. In contrast with the earlier stages the burning now became more pronounced—the ulcer had a callous unhealthy appearance, and discharged serum copiously. I came to the conclusion that it was an X-ray burn, and I went to see Dr. Sequeira, who agreed with me. He examined it critically, and said that the papillae of the true skin were not destroyed, and he thought the condition would eventually heal up. As stated, I had forgotten until now that I had formerly worn my silver bracelet watch on this wrist, so came to the conclusion that it must be a case of secondary radiation. My watch had a thick leather strap intervening between it and the skin, but this evidently was not sufficient to cut off the action of the secondary silver rays. The definite circular form of the patch was conclusive to my mind that the condition was induced by anadriastic rays—not due to scattered radiation from the primary beam. Dr. Sequeira recommended hot fomentations of boracic acid and the use of Burroughs Wellcome's hazeline cream, which is put up in collapsible tubes. I found the cream of the greatest use, soothing and effective in healing. To stop all danger of injury from further work I went off to the seaside. After I had been in Bournemouth for nearly three weeks I felt some slight irritation on the outer side of my left leg and the inner side of my right one. In these positions there are pieces of nickel forming portions of my sock suspenders. On the outer side of the left leg the irritation was under the larger rounded clip of the suspender and on the inner side of the right leg under the small buckle fastening the strap round the leg. The wounds extended until the left one was the size of a shilling-piece, and the right one that of a sixpenny-piece. Rubber and tape intervened between the metal and the skin. Both sores burnt a good deal and discharged a copious yellow serum. Of course I came to the conclusion that these were also burns due to secondary radiation, but the coincidence was peculiar that they should have occurred within three weeks of each other. I always wear gaunt-
Iredell, Marston and Smith: *Diathermy in Gynaecology*

letted lead rubber gloves, so the rays getting at my silver watch must have been of an extremely hard character.

I have been working with X-rays for the last fifteen years, and have never had any dermatitis. It was a curious coincidence again, that after immunity for such a long period two such widely separated parts of one's anatomy should be attacked in sequence. Were the conditions any different from those in which I have been working during recent years? I could not discover that they were. The X-ray tubes I was using were the same as those I have always worked with in recent years. The burns healed slowly but steadily, and had quite healed up in between two and three months' time.

I draw attention to the following points:—

(1) The danger of metal of high atomic weight in near contact with the skin.

(2) The intervention of leather, rubber, clothing, &c., is quite evidently not sufficient to prevent this.

(3) It appears that the action of secondary rays is therapeutically much more powerful than photographic experiments seem to show. Whether they possess any advantages superior to those of the primary X-ray beam is however doubtful.

**Diathermy in Gynaecology.**

By C. E. IREDELL, M.D., A. D. MARSTON (Temporary Surgeon-Lieutenant, R.X.), and G. BELLINGHAM SMITH, B.S.

Some months ago a bed in Queen Ward, Guy's Hospital, was set apart for the investigation of the action of diathermy in the treatment of carcinomata of the uterine cervix. It was decided that only those cases which were inoperable should be treated, and this paper is a record of the results obtained to date. The first factor that usually makes a growth of the uterine cervix inoperable is the spread of the disease anteriorly to the bladder and posteriorly to the rectum, the surgeon being thereby prevented from operating because he cannot completely remove the growth from these organs. Possibly, however, the diathermy used to produce a cauterizing effect may destroy the cancerous growth attached to these two organs without their walls being permanently injured. Moreover, after operation recurrences, if
they do occur, appear first in the seat of the operation, and therefore they may possibly be treated in the same way.

The following is the operative procedure: The patient is anaesthetized and placed in the perineal position, and the soft breaking down growth is scraped away with a sharp spoon. This is a very important proceeding as we find that the less the diathermy current is used the less is the constitutional disturbance following the operation. The indifferent electrode is about 3 in. by 1\(\frac{1}{2}\) in. and consists of bare metal. It is held by the Sister on the thigh or buttock of the patient and as soon as it is felt to be warm the current is switched off and the electrode moved to another position. The operation lasts about fifteen minutes and the whole mass of growth is treated with a bullet-nosed electrode \(\frac{1}{8}\) in. in diameter. This is held in the right hand and the temperature is estimated from time to time by the left forefinger. A current of 1 amp. is used and we have so far never seen any harmful local effects from its application. The temperature should be rather above that which can comfortably be borne by the finger. If a smaller electrode is used a much higher temperature over a smaller area is very quickly produced, while, if a larger one is used, the temperature with 1 amp. is probably not high enough to exercise any beneficial result. With the use of a powerful machine the operation could probably be done more speedily. As it is almost impossible to bring the patients down to the Actino-Therapeutic Department from Queen Ward, we have made use of a portable diathermy machine, which our electrical engineer, Mr. Pullin, found would work as well off an interrupted continuous current from the lighting main as it did from the alternating current. The mains, however, are not thick enough to carry a current which will produce diathermic oscillations registering more than 1 amp. on the hot wire ammeter. The amount of constitutional disturbance on the day following the operation is in proportion to the amount of breaking down of tissue produced by the heat. The scraping has no constitutional effects.

The standard with which we started was to give an anaesthetic and operate three times, with a day’s interval between each operation, and then send the patient out, and repeat the series in a month’s time. In practice, however, we found we could seldom do the diathermy more than twice a week. It may be objected that as the cervix is insensible to heat and pain there is no need to give a general anaesthetic. We have treated several cases in this way and with satisfactory results as regards pain, bleeding, and discharge, but have always found that the
Notes on Diathermy Apparatus

By C. M. Dowse, B.Sc., A.M.I.E.E., and C. E. Iredell, M.D.

The construction of the apparatus used for diathermic treatment is of great importance to the electro-therapist from two points of view, namely, safety and efficiency. The alternating currents of high frequency which are employed to produce heating effects are usually obtained by
repeated discharges of a condenser, and there are two main forms of the apparatus in general use.

In one case low frequency alternating current is fed into a step-up transformer and the voltage raised to about 700 volts. Across the secondary terminals a spark gap is connected, and parallel with this there is a condenser and an induction coil. In the other form of apparatus an induction coil replaces the transformer, and the supply is from some source of continuous current with periodic interruptions.

There are also two ways of connecting the patient, shown in figs. 1 and 2. The former of these is preferable from the points of view of safety, and ease of regulation of the diathermic current. There is no direct connexion between the patient and the circuit in which the high frequency current is produced, but by means of an air core transformer $P_2 S_2$ energy can be safely transferred to the circuit in which the patient is connected. The current can be varied conveniently by changing the distance between $P_2$ and $S_2$ or by inclining the axes of these coils.

The method of direct connexion shown in fig. 2 is not to be recommended, as, if the condensers C C break down, the patient is in connexion with the high voltage low frequency circuit and a dangerous shock might result, especially when a transformer is being used. Although these condensers are made with a high factor of safety the possibility of rupture of the dielectric cannot be ignored, as we have already, at Guy's Hospital, broken down both the paraffined paper condensers supplied by the makers of our apparatus, and also two out of three made in the Works Department from the glass of old photographic plates. There is little doubt that the molecular strain produced in the dielectric by high frequency currents sometimes causes disintegration of the material after much use, or at any rate a serious reduction in dielectric strength. Cases are known in which glass tubes that had been subjected to prolonged electric stresses fell to pieces some hours after the stress was removed. It should be noted also, that the condenser may be strained by surges due to faults, or irregular sparking, and hence it is desirable to reduce the electric flux density as much as possible where the edges of the metal plates of the condenser touch the dielectric. This is achieved by placing the condenser in oil, which must reach well above the top of the metal plates. This increase of surface density should be avoided in all parts of the high frequency circuit, as otherwise considerable losses will take place due to brush discharges. To a large extent these can be prevented by avoiding all sharp edges
and rounding metallic surfaces such as the condenser plates and the electrodes.

If it should be necessary to use the direct connexion of fig. 2, the danger of breakdown may be reduced by replacing each condenser C by two condensers in series, each having twice the capacity of C. This reduces the pressure difference across each capacity to one half of its former value, leaving unaltered the total capacity of the circuit, and hence the oscillation frequency.

![Fig. 1.](image1)

![Fig. 2.](image2)

It is of some interest to consider the question of the possibility of occurrence of effects other than thermal. Although the frequency of the electrical oscillations may be very high, in certain circumstances an unpleasant pricking sensation may be felt. This effect probably depends partly on the strength of the current, and may become troublesome, for instance, when the current is passed from the back to the abdomen, as a larger current is then possible than when the electrodes are held in the hands; but it also appears to vary with the frequency of the spark discharges. Some preliminary experiments which we made with a Poulsen arc as a diathermy generator indicated that it was possible to obtain pricking with undamped oscillations, which was somewhat unexpected. We propose to make a more complete investigation of this effect.
The efficiency of the whole apparatus cannot be fully discussed now, but two important points may be briefly considered. The connecting wires in the primary circuit joined to the condensers and the spark gap should be as short as possible, and should be made of a number of fine strands twisted together, as in this manner the effective resistance is reduced to a minimum. Any change in the configuration of this circuit may change the value of the frequency of the oscillations produced, and if the secondary circuit be previously accurately tuned, a considerable fall in the value of the secondary current will ensue. The inductance coil for instance may consist of only one or two turns of wire, and it is quite probable that the inductance of the leads is greater than the inductance of the coil. The same effects may occur, but in a smaller degree, in the secondary, which should be tuned so as to be in resonance with the primary circuit. This occurs when the product of capacity and inductance is the same in both circuits. The curve showing the relation between this product and the current in the secondary circuit is called a resonance curve. Fig. 3 shows two such curves obtained
from apparatus of the type shown in fig. 1. The full line curve was obtained with the electrodes short circuited, the dotted curve when the electrodes were held in the hands. The difference in character is very marked. The curve with short-circuited electrodes falls very steeply from the maximum on both sides, so that a small change in the capacity or inductance in either circuit will produce a large change in the current. Fortunately the curve obtained when the resistance of the body is included is much less steep on one side, so that an increase in capacity will not materially alter the current, but a reduction in the value of the capacity will involve a serious reduction in the diathermy current. The difference in the shapes of the two curves is partly due to the damping effect of the resistance of the body, and this will be less marked if the electrodes are connected to parts of the body which include a much smaller resistance. It is obvious from what has been said that it is most desirable that the secondary condensers Cs Cs (fig. 1) should be readily variable in value so as to compensate for changes in disposition in both secondary and primary circuits. This does not seem to be provided for in commercial types of apparatus. The resistance of the human body is high as compared with metals, and were it not so any diathermic effect in the centre of the body or of a limb would be impossible. In a metallic conductor the passage of an alternating current of high frequency only produces heat in a thin skin on the surface, to which the current is confined, and any internal heating is the result of conduction from the outer skin. This effect diminishes very considerably as the specific resistance of the substance through which the current is passing is increased.

Another point to be noticed is that the heating effect depends not only upon the square of the current strength, but also upon the value of the resistance through which the current is sent. Thus two patients may have very different electrical resistances between corresponding points, and hence the reading of the ammeter in series with them will not give a satisfactory indication of the relative amounts of heat produced. It must be remembered, in this connexion, that the resistance to high frequency currents is very different from that obtained by tests with continuous currents.

Some of the experiments referred to were performed in the electrical laboratory of the Northampton Polytechnic Institute, to the authorities of which the authors wish to tender their thanks for the facilities provided.
The Treatment of Malignant Disease by Diathermy and Fulguration.

By C. E. Iredell, M.D., and Philip Turner, M.S.

Of all the modern methods of treating cancer few, if any, seem likely to replace surgery. Some, however, seem likely to be most important aids and additions to surgical treatment, and of these new methods the electrical are among the most promising. This paper is based upon a series of cases in which fulguration and diathermy were employed during the years 1909-12. The record of these twenty cases will, it is hoped, give some idea as to what has been done, as to what may be expected from these methods of treatment, and as to the class of case in which either method may be used with advantage.

Fulguration.

This method is the application to a cancerous area of a spark, produced by the ordinary high frequency apparatus which is in common use. The apparatus is tuned up so as to give a spark which must be at least 3 in. long. The growth should first of all be removed as completely as possible by ordinary surgical means, and it is an important point that if masses of growth which can be seen are left an early recurrence is certain. It is desirable, though not essential, to connect the patient to earth by binding an electrode 3 in. or 4 in. square to the leg, and connecting it by a wire to a gas or water pipe. This prevents any bystander from getting a shock should he touch the patient. A cable from the top of the resonator is connected with the electrode, which consists of a wire mounted in an insulating handle. The spark is directed from the end of the wire to the surface of the wound. The best electrodes have a movable sheath of porcelain which can, if required, be pushed down for 3 in. or 4 in. over the point of the wire, thus forming a channel through which the spark can pass. This is most useful in all but shallow wounds. In fact, in deep wounds, it is impossible to direct the spark to the bottom of the wound without it, since the edges of the wound form the shortest path. Just as the lightning flash is very erratic in its course, so, to a less extent, is the fulguration spark, and for this reason the insulating sheath is of great
assistance. Unfortunately, after two or three minutes, the spark makes the porcelain so hot that it cracks unless great care be exercised. It is therefore advisable to spark on to the more superficial parts of the wound without using the porcelain sheath, keeping this for the more inaccessible parts. The presence of moisture is important. While the spark will pass to any solid object that is not an insulator, and with some difficulty to substances which are insulators, no spark will pass if the electrode is brought near the surface of water, or even if it touches it. Practically, however, the spark will always pass to a surface which is moist, but it saves a good deal of trouble if the surgeon, by attention to haemostasis, makes the wound as dry as possible before commencing the fulguration. Otherwise much time is wasted in sponging. In our cases the duration of the electrical treatment has varied from ten to twenty minutes according to the extent of the disease, except in one case in which the treatment lasted forty minutes.

Diathermy.

This form of treatment is merely a method of producing heat. Diathermy is distinct from fulguration, the only point of resemblance being that the high frequency apparatus is used in each. Diathermy differs, however, from all other methods of treatment in which heat is employed, first, because the heat is produced in the tissues themselves by the resistance of the body to the electric currents, and, secondly, because the temperature and quantity of heat produced can be regulated with greater ease and accuracy than in any other method. If two similar electrodes connected with the poles of the diathermy apparatus be placed on opposite sides of a thick piece of meat, heat is developed, at first on the surface of the meat, but subsequently spreading, so that the centre becomes warm also. If the process is continued long enough the meat begins to coagulate under the electrodes and the process extends so that at last a cylinder of coagulated meat is produced, with cross section the same as the electrodes. If, instead of the electrodes being the same size, one is smaller than the other, the smaller will get hot more quickly and coagulation will start there. If one electrode be large and the other the size and shape of a bullet, coagulation will only appear in the region of the smaller electrode. It is interesting to compare the action of the actual cautery with that of diathermy, and this can be conveniently done with egg albumen. If a Paquelin's cautery be made red hot and the end placed in egg albumen
it will be noticed that a white ring of coagulated albumen forms round the metal, which may actually char the albumen in contact with it. At the same time the fact that the ring is not very wide shows that the heat has not penetrated very far. If now a large metal electrode be placed in the solution, and the other pole of the diathermy machine be connected to the cautery which has been allowed to cool, and the current be turned on, coagulation will again occur near the cautery. It will however not merely surround the cautery but will extend from it towards the large electrode. The coagulum will not, at all events at first, be so firm and so white as in the former case, but it will be very much wider, and it will be difficult to mark the exact spot where the coagulation ceases.

In actual practice we place a large electrode (6 in. by 12 in.) on the back of the patient. The growth to be treated should be removed as far as is practicable by surgical means, though this is not so essential as with fulguration. Preliminary removal of as much growth as possible means that there will be fewer after-effects due to absorption from the destroyed tissues, and also that there will be less chance of damaging important underlying structures such as large vessels and nerves. The small electrode is now applied to the diseased surface until superficial coagulation has been produced and the tissues have been heated to a considerable temperature. At present it is not possible to measure the temperature of the tissues under treatment, and as the tendency of the heat is often to set up capillary bleeding it is not easy to estimate the amount of coagulation. If the temperature is high enough to render the tissues moderately unpleasant to the finger it may be considered sufficient, but we hope that it may be possible to devise some more accurate form of measurement.

As fulguration and diathermy were new methods and the dangers associated with them unknown, the cases were carefully selected. In all there was either some factor which in ordinary circumstances would lead one to regard the case as inoperable, or other methods of treatment had been tried and had failed.

Of the twenty cases, seven were treated with fulguration, ten with diathermy, while in three the two methods were combined. Seven were cases of large fixed secondary epitheliomatous glands in the neck, in some of which the primary growth had been successfully removed at a previous operation, while in others the primary growth in the mouth had either recurred or had not been treated. Five were extensive rodent
ulcers in which other treatment had failed, two were epithelioma of the face, two epitheliomata of the nose, two advanced cases of carcinoma of the breast, and one a case of recurrent sarcoma of the upper jaw. There was also one case of extensive breaking down tuberculous glands in the neck.

In a new form of treatment, especially where powerful electric currents are employed, it is of the greatest importance to know whether there are any particular dangers associated with it, and we will therefore first deal with two cases which were followed by unexpected and unfortunate results, though only one had a fatal termination. In this case there was a large secondary growth in the right submaxillary region, fixed to the lower jaw and extending to the muscles of the tongue. The growth was removed as far as possible through a large submaxillary incision and the deep parts of the wound treated with diathermy. Though the growth bulged into the floor of the mouth, the mucous membrane was not opened, but the electrode was applied to it, as well as to the muscles of the tongue. The middle third of the wound was left open for drainage, but though the patient did well for ten days the wound then became septic, and there was a secondary haemorrhage. This was checked, but bleeding recurred on two or three occasions, on the last of which it proved fatal. In this case it would probably have been wiser to use fulguration. Had this been done there would have been less damage to the mucous membrane and tongue muscles, there would have been less sepsis and sloughing, and the vessel—a branch of the lingual artery—would probably have remained secure. In the other case the indifferent electrode, which was held against the patient’s neck, produced a burn, this being due either to the electrode not being in electrical contact or to the heat being too great. The extensive granulating surface had to be skin-grafted, and the resulting scarred surface was about as pronounced as would be the scar of a healed lupus vulgaris in the same region.

In another case there was a large mass of malignant glands in the neck extending from the lower jaw to the clavicle. This was removed surgically as far as possible, but near the clavicle a thick mass of growth had to be left, and this was treated by diathermy. About twelve days afterwards this thickness of growth separated as a slough down to, but not including, the carotid sheath. Had the diathermy been continued a little longer in this case there might have been damage to the large vessels of the neck, ending fatally, in spite of the tendency of vessels to escape injury.
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In all cases the strictest aseptic precautions were taken, and no suppuration took place, except in the case in which secondary haemorrhage occurred, and in those in which open wounds were left to granulate. In no case were any enlarged glands noticed due to septic absorption from the wound. On the other hand, in the case of a patient who had for years suffered from suppurating tuberculous glands with many sinuses, the wound healed perfectly and soundly after diathermy. We thus conclude that there is little or no tendency to suppuration after fulguration or diathermy, and that if sloughs form they either separate with a slight amount of sepsis or may be absorbed.

During the operation and immediately after it, we have noticed no ill-effects, nor is there any reason for thinking that these methods of treatment exercise any deleterious action. On several occasions we have treated growths in close proximity to the large vessels of the neck and the vagus nerve by fulguration, and no ill-effects were noticed, save for a slowing of the pulse of about 10 to 15 beats a minute. The destructive effect of diathermy is greater and it must be used with caution in the proximity of large vessels. The after-effects of fulguration are singularly slight and in almost every case on the day after operation the patients were much better than would have been expected considering the gravity of the original condition and the severity of the treatment. For a day or two after the operation there is a profuse serous discharge, but this soon ceases and healthy granulations appear. For a few days after diathermy there is generally a certain amount of depression similar to that seen after a severe burn and probably due to the same cause. It is interesting to note that after both methods of treatment the resulting scar is remarkably soft and supple, and that even in extensive cases we have not seen any formation of thick masses or of contractile fibrous tissue.

Seven cases of epithelioma of the mouth or tongue with secondary glands in the neck were treated, two by diathermy, four by fulguration, while in one case both methods were employed. In one of these an extensive growth of the fauces and tongue was removed and the base of the wound was treated by diathermy. This healed and remained quite normal, but some months later extensive deep secondary deposits appeared in the neck. In the remaining six cases it would be idle to claim that there was any marked prolongation of life, but, bearing in mind that all were advanced "inoperable" cases, it is interesting to note that with one exception all left the hospital with the wound healed, and with the same exception there was no recurrence of growth.
in the scar. Death in all cases was due to deep secondary deposits and metastases, and in six of the seven cases there was some diminution of the pain and suffering of advanced malignant disease.

Five cases of rodent ulcer were treated, three by diathermy, one by fulguration, and one by both methods. It is well known that some rodent ulcer cases do not permanently yield to X-ray and radium treatment. Apparent cure occasionally follows their application, but both methods seem to have lost their former beneficial effect. Sometimes even large amounts of radium administered for long periods fail to cure, and it is especially in these intractable cases that diathermy or fulguration are likely to be successful, diathermy being generally the more effective of the two. We showed two cases, one completely and the other partially successful, before the Clinical Section of the Royal Society of Medicine in 1912.1 Clinically these growths are generally of a malignant type, growing rapidly, and, until a histological examination has been made, are often regarded as having become epitheliomatous. All five cases healed well, and having been observed for over a year without any recurrence may be regarded as cured. In one case there was a fungating tumour in the parotid region 3 in. in diameter and extending beneath the deep fascia. There was a history of four years' duration and of apparent cure by X-rays twice, then of a further recurrence with failure to improve with X-rays. A wide excision of the growth was then performed but a rapid recurrence quickly followed and the patient was sent to us for treatment. The appearance suggested an epithelioma, but histological examination proved the tumour to be a rodent ulcer. Clinically it can scarcely be doubted that the growth was very malignant and that the prognosis was bad. The growth was removed as freely as possible and this was followed by both fulguration and diathermy. The wound healed well, and when the patient went abroad and was lost sight of over a year afterwards there was no sign of any recurrence. Four cases were epitheliomas of the face or nose without glandular enlargement but too extensive to allow of wide excision. Three of these may be regarded as cured, though one patient, who was aged 75, was lost sight of a year after operation. One case in which epithelioma became super-imposed on lupus-vulgaris of ten years' standing remained well for six months and then there was extensive recurrence.

Two patients who suffered from carcinoma of the breast were very advanced cases but the results were encouraging. In one case there was a large fungating growth 4 in. in diameter firmly fixed to the chest wall. Diathermy was used after as complete a removal of the growth as was possible by surgical means. The wound healed and she remained well for a year, when small local recurrences appeared and there was also severe pain in the back pointing to a secondary growth involving the spine. The other patient also had a deeply ulcerating mass firmly attached to the underlying structures and she was anxious to have something done on account of the offensive character of the wound and the discharge. In spite of a very wide excision necessitated by the size of the growth the wound healed well after treatment by both methods on different occasions. She remained well for six months and then developed enlarged glands above the clavicle and gradually went downhill, though a year after the operation the scar of the wound remained free from growth.

The case of sarcoma of the jaw was a patient of the late Mr. L. A. Dunn. The upper jaw had been excised and there was very profuse hæmorrhage which was only controlled with difficulty. The growth recurred some months later as a spongy mass the size of a walnut. After the severe hæmorrhage of the original operation Mr. Dunn was unwilling to risk any further cutting operation. Accordingly a diathermy electrode was plunged into the growth in two or three places with the result that in a week's time the growth sloughed away. The patient went home and at the end of a year there was no further recurrence.

The only case of tuberculosis in the series was of considerable interest. There was a very large fixed mass of glands in the neck, too extensive for excision, which had been incised and scraped on several occasions with the result that there were several deep sinuses. After a single application of diathermy the mass of glands shrunk up and the sinuses firmly healed. This case certainly suggests the desirability of a further trial of this method in old standing tuberculous sinuses and in suppurations in which other treatment has not been successful.

Conclusions.

(1) These methods are of great value in the treatment of extensive localized malignant growths in which surgical treatment alone is unlikely to be successful, owing to the extent and fixity of the growths, or to the proximity of important structures.
(2) Isolated secondary growths may be treated in the same way, but there is no effect on the development and growth of metastatic deposits. In such cases, however, the eradication of the primary growth often leads to a considerable prolongation of life, with a much greater degree of comfort.

(3) Diathermy is probably more effective than fulguration, but fulguration is less likely to damage large vessels or other important structures.

(4) Considering the severity of the cases the dangers of the treatment are slight.

(5) These methods of treatment are suitable for all accessible forms of malignant disease and to certain forms of tuberculous disease.
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President—Mr. Cecil R. C. Lyster.

Diathermy in Diseases of the Eye. ¹

By C. E. Iredell, M.D., and C. Meadows Ryley, Temporary Surgeon-Lieutenant, R.N.

In dealing with diseases of the eye the action of hot fomentations is well known and the hot pad—the source of heat being the electric current—is also used with considerable success. In this case the outer layer of the skin of the eyelid is warmed by conduction and presumably a certain amount of this penetrates the eyelid and reaches the eye. In using the diathermy current a special electrode is used. It is formed like an eye cup and contains at the bottom a metal connexion by means of which it is connected by a wire to the diathermy machine. This is filled with normal saline solution and applied to the eye in the ordinary way. The other electrode may be a metal handle, or if both eyes are affected they may be treated at the same time with similar cup electrodes. The eye, with the rest of the body, forms part of the electric circuit through which the diathermy current passes and therefore heat is generated in the organ itself instead of passing in through the eyelid by conduction only. We may point out here that in using a cup filled with normal saline as the diathermy electrode, there is no necessity for the patient to keep the eye closed as is the case with the hot pad, and, so far as our observations go, it makes little if any difference to the patient’s comfort, or to the efficacy of the treatment, whether the eye is kept shut during the passage of the current or not. The length of each treatment was from fifteen to thirty minutes and the current used about 0.3 amp.—i.e., to produce a pleasant warm sensation. We also

¹ At a meeting of the Section, held January 17, 1919.
wished to ascertain whether definite therapeutic effects could be obtained
with diathermy which could not be obtained with the hot pad and
whether in the latter case it was capable of doing harm. For this
reason we chose for our first case a patient whose affected eye was
blind.

Case I.—Male, aged 47. Had suffered from glaucoma for several years in
the right eye which had been blind for two years. The tension was +2 and
the pupil small; the whole eye was much inflamed and the patient had been
admitted to the hospital with the idea of having the eye removed on account
of the pain and headache. He had been treated with the hot pad for some
days with some possible slight improvement and when first seen actually had
the pad on. Then, as on all previous occasions, the application of the pad
seemed to bring on the pain. He was taken away and treated as described.
After the current had been turned on for a few minutes he said the pain was
much less. This result was the reverse of that found with the hot pad, which
at the time of application increased the pain. At the end of the treatment (half
an hour) the pain had gone. For the rest of that day and the following two
days the pain was very much less than usual. The treatment was repeated and
in a few days all necessity for removing the eye had disappeared and he was
discharged. In all eighteen treatments were given, this being probably more
than would otherwise have been necessary owing to the patient's irregular
attendance.

Case II.—Male, aged 37. Rheumatic iritis of several years' history. The
vision was diminished and the patient had considerable pain and discomfort.
After eight treatments the vision was normal and all inflammation had died
down. The lower part of the iris was still adherent to the lens and the patient
was otherwise quite well.

Case III.—Male, aged 46. Was sent for treatment suffering from chronic
gonorrhoeal iritis of twenty years' standing and there was much pain and
discomfort. After the first treatment the patient was quite free from pain.
After six treatments he was sleeping much better at night and after the
eleventh he was discharged, the condition being quiescent.

Case IV.—Female, aged 43, suffering from iritis. After six treatments the
inflammation was less and the patient felt better. She had treatment for
several weeks without improvement so far as vision was concerned, but the
pain was much less.

Case V.—A nurse, aged about 45, suffering from chronic iridocyclitis of
four years' standing. She had had pyorrhœa for which all her teeth had been
removed, but without any improvement so far as her eyes were concerned.
After two treatments she was distinctly better, and after twenty-three treat-
ments there was a general improvement, no sign of cyclitis remaining.

Case VI.—Male, aged about 40, suffering from chronic iritis, for which no
definite cause was ascertained. He had had eight injections which produced
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no improvement and was suffering from pain in the temple and at the back of the eye. This cleared up under treatment and after nine applications he was discharged much improved.

Case VII.—Male, aged 34. Chronic iridocyclitis of six years' standing for which no cause could be found. Much vitreous opacity was present, especially in the right eye. In this eye vision was originally $\frac{2}{6}$ and is now $\frac{2}{6}$. Left eye: vision originally $\frac{4}{6}$ with glasses; now $\frac{6}{6}$ with glasses. There is less opacity than formerly in the right eye and all opacity except one small mass has now disappeared from the left eye. He carries on his occupation of booking clerk without difficulty.

Case VIII.—Male, aged 33. For nearly ten years had had recurrent attacks of cyclitis with rise of tension in the right eye. The attacks lasted from fourteen to twenty-eight days. They were accompanied by fairly severe pain and much diminution of vision, with some rise of tension. Vision $\frac{6}{6}$ (some letters), and during attacks, vision $\frac{2}{6}$. No cause was found for the cyclitis, and many oculists of the first class had all failed to find any reason for the condition. The attacks came on at intervals of from two to four months. Iridectomy was performed nearly a year ago with no improvement. Nine months ago diathermy was started, being given for five minutes twice a week, the time being increased to fifteen minutes every other day. There has been a steady improvement ever since. The attacks have become less frequent and of shorter duration. The last attack lasted three or four days. There is very little rise of tension and there is practically no exudate on the posterior surface of the cornea. It cleared up without atropine, which was always necessary formerly. The diathermy has been continued through the attacks without any prejudicial effects arising.

Case IX.—Male, aged 46. Chronic secondary glaucoma. It somewhat resembled Case VIII, and was due to chronic iritis following oral sepsis. He had attacks of iridocyclitis with rise of tension about every six weeks, lasting about ten days. The teeth had been removed and the mouth cleaned up. There was no history of gonorrhoea and the Wassermann reaction was negative. No kind of treatment prevented the attacks but diathermy had been tried for about six months, ten minutes every other day. The attacks are diminishing in frequency and duration. The last was six weeks ago and it lasted five days. There was very little K.P., and the tension rose from 25 to 30 mm. Hg, as registered by the Schiødtz tonometer.

Conclusions.

In chronic inflammatory conditions of the eye diathermy has a marked palliative effect both as regards relief of pain and the reduction of the other inflammatory symptoms. No harmful results have ever been observed and in all cases treated a general improvement in the ocular condition has been obtained.
Diathermy in Abdominal Disorders.

By C. E. Iredell, M.D.

In an account of the treatment of a patient suffering from leprosy published in the Lancet some years ago, reference was made to the action that diathermy appeared to exercise in stimulating intestinal movement. In this patient an attempt was made to produce hyperthermia in the liver and spleen, with the idea that if leprous deposits existed in these organs diathermy might exercise as beneficial an effect on them as it had done on the enlarged and painful ulnar nerve. No obvious effect was expected or observed on either organ, but a rumbling sensation was produced in the abdomen during treatment, and one or more actions of the bowels followed in the course of the following few hours. This took place even if pills and white mixture had previously been taken with satisfactory results the same morning, and the patient did not expect any further action would result until the following day. The method adopted was to place the two electrodes, each about 6 in. square, so that the spleen lay between them, one electrode being situate on the abdomen and the other on the back. The current was then switched on and increased until a feeling of warmth under the electrodes was produced. It was continued for twenty minutes, and the liver was then treated in the same way. The hot wire ammeter registered about 2 amp.

Since then this treatment has been used in thirty-four cases. The length of each treatment has in many cases been extended from the forty minutes above mentioned to one hour, and in periods ranging from one week to several months. It was given three times a week or even more frequently. These thirty-four cases may be divided into two groups, the first of which contains those cases in which abdominal symptoms only are present, and the second those in which they are absent or of apparently secondary importance.

The first group consists of nineteen cases, comprising five cases of constipation, in which pain, if present, was slight, seven cases of abdominal pain due to adhesions after colectomy and other operations; three of constipation with neurasthenia; two of chronic colitis; and one each of lienteric diarrhoea and persistent vomiting. All these cases had undergone a great deal of medical treatment, and may fairly be classed as severe. In all there was distinct improvement,
usually within a week of beginning treatment. Two of the cases of constipation call for special notice. One patient had been admitted to the hospital under the care of the late Mr. L. A. Dunn, for what was thought to be intestinal obstruction. She recovered without a surgical operation, but remained very constipated, her bowels being opened only when purgatives were given three times a day. A bismuth meal showed that there was distinct iliac and colonic delay, bismuth reaching the sigmoid flexure one hundred hours after administration. She was very thin, had pronounced pigmentation round the eyes, and a very muddy complexion. Diathermy was started, and it was found three days later when the purgatives were stopped that she continued to have a daily motion. Three weeks later the treatment was discontinued, and it was found that she still had a daily motion. Another bismuth meal was given, and all trace of it was found to have gone twenty-two hours later. The other case was that of a patient who had been in the hospital for over a year for constipation, and on her discharge her report states that she was much better than she was on admission, and that she was having a motion of the bowels once a week after three enemata. Three years later diathermy was commenced, and the interval of one week which had remained the same during that time was reduced after three months' treatment to four days, and she felt correspondingly better for this improvement. In the case of the seven patients suffering from abdominal adhesions the relief of pain was immediate, the intervals between the attacks became longer, and in all except one the pain seems to have completely disappeared in a few weeks. Unfortunately relapses occurred in at least three of the cases, and another course of treatment became necessary. In the three patients suffering from neurasthenia the constipation was apparently cured, and the other symptoms improved. The two patients suffering from colitis—one of the mucous variety and the other of the colitis following dysentery—both recovered. The diathermy at first produced an increase in the quantity of mucus passed; this soon subsided, and after three or four weeks the condition cleared up completely. The patient suffering from vomiting after every meal improved directly treatment was started, and when last seen after a course of twenty-nine applications had had no vomiting for eight days. The patient suffering from lienenteric diarrhoea states that he was much improved by a course of treatment.

The second group consists of fifteen cases in which abdominal symptoms are absent or of only secondary importance. In one case the patient, suffering from acute rheumatoid arthritis, has given such a good
account of the effect of the treatment on him that I reproduce it in his own words, especially as it is typical of what is experienced by most patients undergoing diathermic treatment:—

"I felt a general improvement from treatment, but find it hard to indicate precisely in what direction. The chief points were:—

(1) Great improvement in my cough, which up to the time of treatment has been troublesome. Coughing was considerably less, and such as there was, was produced naturally and without strain or pain in the chest. I also found I could sneeze freely and fearlessly.

(2) Previous to treatment I had had blue fingers in the morning but not since.

(3) A feeling of ease in the stomach, previously lacking, and improvement both in regularity and quantity of motions.

(4) My ankles have become normal.

I further note that I seldom felt immediate benefit at the conclusion of the day's treatment, nor generally did I feel any increase of warmth during treatment. Moisture generally collected in beads on the electrodes where in contact with the skin, but beyond warmth in hands and sometimes flushing of the face little increase of heat was apparent. On no occasion did I feel warmth within."

The improvement in the circulation of the fingers was noticed by several patients, and especially in two who were being treated for Raynaud's disease. Several weeks' continuous treatment produced no beneficial effects of any sort on patients suffering from splenomedullary leukaemia, amyotrophic lateral sclerosis and progressive muscular atrophy. Three cases of asthma were treated, of whom one had only two applications. The other patients were improved as regards their general health, but the number and severity of the attacks were little, if at all, affected by treatment, and it is interesting to note that in one patient exophthalmic goitre actually developed while she was undergoing a course of diathermy. One patient, weighing 18 st. and suffering from an ulcer of the leg, was a good deal better for the treatment, apparently owing to the improvement in her general condition, and one patient who suffered from attacks of unconsciousness, said to be due to cardiac trouble, improved so much that the attacks stopped after a few weeks' treatment. Three patients suffering from diabetes had eight, eleven, and fifty applications respectively. No improvement was noticed, and the last two cases, who were patients in the hospital and were being carefully dieted, showed no diminution in the sugar output. The patient who had fifty treatments had a marked smell of acetone in her breath, and this disappeared after an
hour's treatment, only to reappear a few hours later. On several oc-
casions two hours' consecutive treatment was given without any beneficial
effects. She died a fortnight after leaving the hospital.

The conclusion arrived at is that the diathermic current has a
definite action on the movements of the intestinal canal, both in pro-
ducing peristalsis and in checking vomiting and diarrhoea, and that this
action takes place even when ordinary medicinal means have failed

Case of Acne complicated by Intestinal Stasis in which a
General Improvement was Effect ed by Diathermy.

By H. W. Barber, M.D.

The patient gave the following history: As an infant she was
exceptionally healthy, and was well, apart from attacks of chicken-pox
and whooping-cough, until the age of 12. At this time her menses
began, and she developed acne on her forehead, shoulders, and back.
She states that "when the acne first appeared it was more like small
blind boils, and, as it began to affect the shoulders and face, pustules
took the place of boils, or a combination of both would appear."

Apart from bran baths and laxatives, she had no treatment until
the age of 16, when she was given a vaccine prepared by Burroughs,
Wellcome and Co. for a period of six months. She is ignorant of the
nature of the vaccine and of the doses she received. Her condition
was rather worse than better at the end of this time. When aged 17\frac{1}{2}
she had another six months' course of vaccines, but no improvement
followed. After some unsuccessful attempts an autogenous vaccine
was prepared, and she was given injections with this when aged 18\frac{1}{2}.
There was considerable improvement in her condition after nine months'
treatment, but a severe cold apparently caused a relapse. During this
time she was also receiving local treatment by massage, ointments,
powders, and baths. She also found herself compelled to take a laxative
every day.

In June, 1913, she received treatment by small doses of X-rays, and
later by hyperaemia and ionization. Owing to the amount of scarring
caused by the pustules she was also given twelve injections of fibrolysin.
During this period, also, she was having injections of stock acne vaccines.

In December of the same year she had attacks of severe abdominal
pain in the right side, which were thought to be due to a mild attack of
appendicitis. She remembers having had similar pains when younger. From this moment she found that she was unable to tolerate certain articles of food, and she was forbidden eggs, red meat, pastries, and spiced dishes. She had always lived an athletic outdoor life.

The patient came under my care in December, 1914. The following is a brief description of her condition at the time. Her complexion was sallow, and her general appearance suggested chronic intestinal intoxication. Her face, chest, and back were covered with several disfiguring scars of varying size, and in addition to these scars there were numerous inflammatory nodules (acne indurata), pustules, and boils in different stages of evolution. Comedones were present, but not in large numbers. The patient was acutely conscious of her disfigurement, and almost in despair at the constant appearance of new lesions. Examination of the abdomen revealed marked tenderness in the right iliac fossa, but not elsewhere. The abdominal muscles were rather lax.

The opinion was formed that, seeing that all forms of local treatment had proved futile, the real source of the trouble was intestinal intoxication in a person predisposed to acne. It is to be regretted that no bacteriological examinations of the faeces were made.

It seemed probable, in view of the history obtained and the localized tenderness in the appendicular region, that the appendix might be diseased, and the possibility of ileal stasis from old inflammatory adhesions was considered. The patient was therefore advised to come into hospital for further investigation, in order that one might better decide whether an exploratory laparotomy was indicated or not. This she consented to do, and at the end of January, 1915, she was admitted into Guy's Hospital under the care of Dr. Beddard, who kindly examined her. He agreed that an exploration of the right iliac fossa was justifiable. X-ray examination of the intestinal tract after a bismuth meal showed slight delay in the lower end of the ileum, and very marked stasis in the cæcum, colon and rectum.

The position was laid before the patient, and she gave her consent to an operation. She was therefore transferred to the surgical ward under Mr. E. C. Hughes, who had kindly promised to operate. On February 23 the right iliac fossa was explored. Contrary to expectation the appendix was found to be healthy, and the terminal coil of ileum quite free. The cæcum, however, lay deep in the pelvis, and was long and flabby. The appendix was removed, and the wound closed. The patient made a rapid convalescence and was very shortly discharged.

It seemed obvious from what was seen at the operation that the
indication for future treatment was to attempt to restore tone to
the caecum and colon. I accordingly entrusted my patient to the care
of Dr. Iredell, who had achieved some striking results with abdominal
diathermy. Treatment was begun exactly a month after the operation,
and has been continued until the time of writing (July). The patient
attended the hospital for six months, on an average three times a week.

Improvement very soon became evident, and the whole appearance
of the patient gradually changed. She lost her unhealthy earthy tint,
and became much more sanguine in her attitude towards life. To quote
her own words: "Very few spots appear now, and those that come last
only a short time. I have felt ever so much better and brighter, also
I am able to eat and enjoy almost anything. My skin is much clearer
than it has been for many years." The tenderness in the right iliac
fossa has disappeared, and she has no further difficulty in getting her
bowels open. On my advice, however, she still continued to take an
occasional pill containing ext. nuc. vom. ¼ gr., ext. bellad. ¼ gr., aloes
Barb. 1 gr.

DISCUSSION.

Dr. J. Metcalfe: The results obtained by the readers of the papers are
excellent. Diathermic treatment has been applied to the most various and
diverse conditions and many of the cases appear to have been cured or much
relieved. But the very diversity of the cases treated makes one anxious to
hear more of the actual effect on the tissues involved. The generated heat was
of course the active agent, but have we any definite knowledge of the changes
produced in the tissues by this mode of applying it? One or two cases appear
to have been much relieved or even cured by a single application. Have
metabolic changes been induced or has there been direct action on nerve cells?
And may not the result in the very rapidly cured cases sometimes be attributed
to psychic influences? However, the chief object of all our treatment is to
cure the patient. Whether that be done by demonstrable changes in the body
tissues or by mental impressions is not of supreme importance.

Dr. E. P. Cumberbatch: In the treatment of glaucoma by diathermy by
the method used by Dr. Iredell, the current is conducted to the eye by way of
normal salt solution kept in contact with the orbit. Salt solution of normal
strength presents a high resistance to the diathermy current. There might be
some risk of too high a rise of temperature of the solution (and consequently
a burn) unless the current were small or applied for a short time only. Some
observations on the temperature of moist pads used to conduct the diathermy
current through the body were made in my department by Dr. Noel Burke.
If tap water was used to moisten the pads, the temperature of the pads
gradually rose till it became unbearable. The same thing happened if salt
solution, of 1 per cent. strength, was used. Stronger solutions were necessary to prevent a rise of temperature, in the pad, beyond 98° F., during a course of application of diathermy lasting twenty minutes. Dr. Iredell has obtained very good results and the treatment of similar cases by diathermy should receive a further trial. In the treatment of inoperable carcinoma of the cervix I have had experience of six cases. In four of these it is doubtful whether any benefit was derived. In these the growth had spread into the pelvic tissues and was therefore out of reach of the electrode. Haemorrhage and discharge were not allayed by the diathermic cautery. In the other cases the results were better. One patient (aged 55) was sent to me by Dr. Williamson for treatment. In the situation of the cervix was an ulcerated excavated mass with hard and everted edges. The vaginal wall was involved and the posterior left fornix was obliterated completely. Definite induration extended to the pelvic wall and to the utero-sacral folds as far as the bowel. Diathermy was applied in August, 1913, to the parts of the growth that were accessible from the vagina. The patient left hospital nine days later. The slough had not then entirely separated, but the whole of the vaginal mass previously felt had entirely gone and the vaginal wall was covered with smooth tissue. This patient came at intervals to the out-patient department and no haemorrhage, nor discharge, nor ulceration, were seen. The induration felt previously in the pelvic ligaments remained. She was seen last in June, 1914 (ten months after the diathermy) and could not afterwards be traced. Diathermy should certainly be tried as a preliminary treatment of operable carcinoma of the cervix, before the Wertheim operation, in place of an initial application of formalin or zinc chloride. I treated a case by diathermy in June, 1913. Eight days later a Wertheim operation was commenced by Dr. Barris, at St. Bartholomew's Hospital, but could not be completed on account of the extensive spread of the growth within the pelvis. The part of the cervix to which the diathermy had been applied was found to be firm, like a scar, and no haemorrhage was caused by its manipulation or incision, and there was no discharge. The patient lived till February, 1914. With regard to maladies of the abdominal and pelvic organs, my experience has been limited to a few cases of painful haemorrhoids and two cases of dysmenorrhæa. Pain and spasm are considerably allayed by diathermy applied by way of a rectal electrode. For the cases of dysmenorrhæa the diathermy was applied to the patient on the condenser couch; the active electrode was placed immediately above the pubes. The treatment was given for periods of twenty minutes, six days in succession, till the menstrual flow commenced. It had the effect of much reducing the pain. In one case the dysmenorrhæa was of the painful contraction type; in the other it was of the pre-menstrual congestion type. The treatment was not repeated for the next periods, so I am unable to say whether a further series of applications would ultimately make the menstruation bearable or painless. I am of the opinion that diathermy is specially indicated for pain accompanied by congestion or spasmodic contraction.
On the Mode of Spread of Cancer in relation to its Treatment by Radiation.

By W. Sampson Handley, M.S.

The rôle played by radiation in the treatment of malignant disease is one of increasing importance both on the preventive and curative sides. In a number of cases of cancer the efforts of the surgeon, if unsupported by those of the radiologist, would be unavailing, and of course the converse proposition is equally true. Now, I have found, as a surgeon, that a detailed study of the mode of dissemination of cancer has been of the greatest use to me in my operative work. It has enabled me to substitute carefully-planned rational operation for empirical and haphazard procedure. In the belief that a knowledge of the process of dissemination must form a necessary foundation in planning the radiological, just as much as the operative, treatment of cancer, I venture to-night to ask your attention to some facts and observations about the spread of cancer. I ask you also to consider my paper as a tribute of gratitude from a surgeon to his radiological colleagues for help received.

As might be expected the mode of dissemination of cancer was first worked out in the commonest form of external cancer—namely, breast cancer. Internal cancers are not accessible to observation until after death, and the picture then presented is so complicated and confused that it may defy analysis. Moreover, such cases are at present largely beyond the domain of radiological and still more of surgical treatment. My remarks to-night will refer chiefly to breast cancer.

1 At a meeting of the Section, held February 21, 1919.
A rare form of external cancer—namely, melanotic sarcoma—has also been accurately studied as regards its method of dissemination. Here research has been simplified by the fact that the dark coloration of the growth is practically the equivalent of a specific stain for cancer tissue, a desideratum at present lacking for other growths. The mode of dissemination in melanotic sarcoma has proved to be very similar to that found in breast cancer, and it can easily be demonstrated to the naked eye.

The spread of cancers of the mucous glands, and especially of cancer of the stomach, can sometimes be demonstrated by the use of the stain mucicarmine, which stains mucus red while leaving other tissues unstained. Wherever a degenerate cancer-cell is present and mucus is formed, a pink stain is produced. Thus in situations where mucus is normally absent the track of a cancer can be followed with certainty. When a gastric cancer reaches the parietes at the umbilicus, an event which is not very rare, it is found to spread in the abdominal wall in exactly the same fashion as a breast cancer spreads from its point of origin.

There is thus a strong presumption that all carcinomata and sarcomata which cause infection of lymphatic glands spread after the same fashion, and mainly by a process which I have termed permeation of the lymphatic vessels.

Definition of Permeation.—Permeation is the continuous tendril-like growth of lines of cancer cells, by their own proliferative power along the smaller lymphatic vessels. It is to be sharply distinguished from infiltration, which is the growth of cancer cells through the intercellular spaces.

Lymphatic Anatomy.—For the proper understanding of the process we must consider some of the facts of lymphatic anatomy. What immediately concerns us is the arrangement of the lymphatics of the body-wall, of the skin, subcutaneous tissue, fascia and the superficial muscles. Here the leading fact is the existence, just superficial to the deep fascia, of a plexus of vessels of microscopic size forming an investment for the body as complete as the skin itself. This plexus, the fascial lymphatic plexus, is the main highway for the spread of cancer in the superficial tissues. When invaded by cancer it becomes a kind of shirt of Nessus in which the victim perishes.

It is important to insist on the unity of this plexus, and on the fact that it offers no barrier anywhere to the spread of cancer through its meshes. It is drained by certain lymphatic trunks, which convey its
lymph to the cervical, axillary and inguinal glands. The imaginary lines separating the tributary areas of these three sets of glands run at the level of the clavicle and of the umbilicus. These lines, together with the middle line, divide the plexus into six areas, each of which has its own set of trunk lymphatics. No trunk lymphatics cross these lines, but the continuity of the plexus itself is not interrupted by them.

The fascial plexus is fed by little vertical tributaries which reach it on both aspects, from the skin above and from the muscles beneath. The lymphatics of origin of the skin are little blind finger-like processes, one in each papilla of the skin. A number of these unite to form one of the little vertical tributaries which run down to empty themselves in the fascial plexus.

**Summary of Conclusions on Dissemination.**

Dissemination is usually accomplished by the actual growth of cancer cells along the finer vessels of the lymphatic plexuses—"permeation." Embolic invasion of the regional lymphatic glands, though it almost invariably occurs, only leads to invasion of the blood-stream after long delay; and the work of M. B. Schmidt shows that cancer cells which reach the blood usually disappear without giving rise to metastases.

Permeation takes place almost as readily against the lymph-stream as with it. It spreads through the lymphatic vessels around the primary neoplasm in much the same way as would a thick injection fluid introduced into the tissues by a syringe. If in a late case of breast cancer one examines the region immediately around the macroscopic primary growth, no permeated lymphatics can be detected. Here and there are secondary nodules of growth entirely isolated from one another and from the primary neoplasm.

If, however, the investigation is pushed still further from the primary growth, by the examination of long radial sections of the skin and underlying tissues, we arrive at a region beyond the remotest visible naked-eye metastasis and often lying far from the primary growth. In this region the microscopic growing edge of the carcinoma will usually be detected by careful microscopic search. The microscopic growing edge is to be sharply distinguished from the infiltrating edge of the primary neoplasm where interstitial invasion of the surrounding tissues is occurring. At the peripheral microscopic growing edge there is no interstitial invasion of the tissues, but the principal
lymphatic plexus of the part—the plexus which lies upon the deep fascia—is found permeated throughout—that is to say, its vessels are obstructed by the growth of lines of cancer cells along them.

The disappearance of permeated lymphatics in the area which intervenes between the annular "microscopic growing edge" and the primary neoplasm is due to the destruction, after a time, of the cancerous permeated lymphatics by the defensive process of "perilymphatic fibrosis." The recognition of this process at once removes the difficulty that permeated lymphatics are absent in the region immediately surrounding the naked-eye primary growth.

The process of permeation follows the line of least resistance, and extends, at first exclusively, in the plane of the principal lymphatic plexus into which the lymph drainage of the cancerous organ passes. The annular microscopic growing edge of a breast cancer is therefore found in the plane of the fascial lymphatic plexus, upon, or just superficial to, the deep fascia. It is covered over by normal skin and has normal muscles lying beneath it.

If, however, the tissues are examined at points successively intermediate between the microscopic growing edge and the apparent edge of the primary growth, the cancer will be found penetrating the adjoining layers, the superjacent skin and the subjacent muscle, to a greater and greater depth, and forming nodular deposits therein, which may, however, be sporadic and few in number, and in some cases may be altogether absent.

Cancer thus spreads in the parietal tissues by permeating the lymphatic system like an invisible annular ringworm. The growing edge extends like a ripple, in a wider and wider circle, within the circumference of which healing processes take place, so that the area of permeation at any one time is not a disk but a ring. The spread of cancer in the parietal tissues is, in fact, as truly a serpiginous process as the most typical tertiary syphilide. But in the case of cancer the spreading edge is invisible; and, moreover, the advancing microscopic growing edge of a cancer, owing to the failure at isolated points of the defensive process of perilymphatic fibrosis, may leave in its track, here and there, isolated secondary foci, which give rise to macroscopic metastases. Such nodules, in spite of their apparent isolation, arise in continuity with the primary growth, but perilymphatic fibrosis has destroyed the permeated lymphatics which formed the lines of communication.

We must now turn to the question of treatment.
Treatment of Breast Cancer by Radiation without Operation.

In a number of cases, either because the disease is too advanced, or on account of some constitutional disease, surgical operation is excluded, and the treatment of breast cancer must be conducted solely by radiation.

Hitherto in many cases the only idea which has governed the exact site of application of X-rays to growths of the breast has been that the breast itself should be irradiated, with the additional proviso that attention should be devoted to the axillary glands. Such an idea belongs to the same stage of development as the surgical idea that the operation required for breast cancer is an amputation of the breast combined with removal of the axillary glands. In radiography, as in surgery, it is necessary that advances in pathological knowledge should be reflected in practice. Now that the idea of cancer of the breast as a local disease of an organ has to be replaced by the conception of it as a centrifugal invasion by permeation of the lymphatic system of the body, starting at the point of origin of the primary growth and spreading in a constantly enlarging circle which has no respect for anatomical boundaries, it is obvious that blindfold methods of applying X-rays require revision. It is especially necessary that the radiographer should know the exact site of origin of the primary growth in the breast, for otherwise he can only vaguely locate the area of his work. On the first occasion he sees the patient he should insist on making a complete inspection of the chest with the clothes removed. A chart should be made out on which the point of origin of the growth, as ascertained from the patient or the surgeon, should be marked, and a rough outline of the presumed circle of infected tissues should be traced with a flesh pencil upon the skin, and drawn upon the chart as a guide to future work.

Omission of the simple precaution of fixing the site of the primary growth may lead to an error of as much as 5 in. or 6 in. in the location of the circle of tissues to be radiated. In a case I saw a day or two ago a small carcinoma was situated in the fold below a voluminous breast. The 12-in. circle of presumed infection would extend at least down to the umbilicus. Yet if this case had been treated on general lines simply as a cancer of the breast without removal of the clothes and inspection of the growth, it is almost certain that the abdomen would have escaped radiation altogether. In view of the known facts as to
invasion of the abdomen directly through the linea alba in the epigastric angle the whole treatment might have been vitiated by the omission.

**Position of the Patient during Radiation.**—This same case raises another point. The small growth, lying in the fold below the breast, was protected in front from radiation by the whole thickness of the voluminous and pendulous mamma. It is therefore a question to be considered by the Section whether in certain cases of breast cancer the X-ray treatment should be conducted in the recumbent position and with other precautions, such as the use of a sling or bandage to the breast for exposing the growth to the full force of the radiation. Growth of the outer edge of the breast are similarly protected against radiation by the whole thickness of the breast if the organ is well developed, unless the radiologist has previously secured himself, by inspection of the growth and suitable placing of his tube, against this unnecessary addition to his difficulties. Here if the case is treated on stereotyped lines, simply as one of cancer of the breast, the whole axillary region, or at least the scapular region, will probably escape radiation, and thus again the treatment may be vitiated. These points are obvious, but they are of fundamental importance, and I believe they are sometimes ignored by radiologists. Undue restriction, and perhaps still more often, bad centring of the area of irradiation are in my opinion frequent causes of failure. The second error is inevitable unless pains are taken to investigate the peculiarities of the individual case by questioning and inspecting the patient.

**Undue Restriction of the Field of Radiation.**—It is not sufficient to apply radiation to the primary growth alone, nor even to apply it to the whole of the breast. We have seen that the most active portion of the growth is an invisible circle of permeation spreading through the lymphatics, quite inappreciable by clinical methods, and often reaching a diameter of 10 in. or 12 in. in moderately advanced cases. The most important service to be hoped for from radiation is not the destruction of massive deposits of already more or less degenerate cancer cells but the arrest of the spread of this growing edge of active cells. The achievement is unlikely to be attained unless it is deliberately aimed at. It involves the radiation of an area of the parietal tissues centred upon the primary growth and rather larger than the presumed area of the extension of permeation. The diameter of the radiation circle should, I estimate, lie between 12 in. and 16 in. It is not for me to suggest how this large area should be dealt with, but its periphery
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is perhaps even more important than its centre, because the cells at the periphery are all young and active, while the massive primary growth resembles a half-extinct volcano. On the other hand a microscopic filament like a permeated lymphatic is more easily penetrable than a naked-eye mass of cancer cells.

The Section should consider whether, and to what extent, the use of diaphragms is advisable in the X-ray treatment of breast cancer. In view of the large area to be covered it seems unadvisable to stop-down the tube too much. And in view of the facts I have adduced it is a question whether the area submitted to radiation is not frequently too small.

Prophylactic Radiation.—During the last fourteen years every case of breast cancer upon which I have operated has received, usually at the hands of your President, a prophylactic course of X-rays. I have not adopted the practice of irradiating the wound before closing it, believing that it is unnecessary and that it involves risk of sepsis, and of impairing the vitality of the flaps by chilling them. In my series of cases recurrence in the skin has been almost absent. Three of the five cases in which it took place occurred among the small percentage of cases which had escaped the usual prophylactic course of X-rays. This striking fact establishes to my mind the great prophylactic value of irradiation. In undertaking prophylactic irradiation the operator should bear in mind before beginning the irradiation:

(a) That the operation may have failed to extirpate a portion of the microscopic growing edge, and for the reasons already stated the area submitted to radiation should be a circle of 12 in. or 14 in. in diameter centred upon the primary growth. It should not be forgotten that the periphery of this circle may be its most dangerous part, and that in fixing it regard should be had to the site of the growth in the breast. Neither the radiologist nor the surgeon can afford to ignore the law of centrifugal spread from the primary growth. The isolated nodules left in the track of the process of permeation, after its spread like a smouldering fire through the lymphatics, are often very amenable to radiation. But unless the spread of permeation at the microscopic growing edge be arrested no permanent good will result. It is to the peripheral region, just beyond the remotest visible deposits, that the special attention of the radiologist should be given. Here he may hope to arrest the process of dissemination by killing the microscopic growing edge. If his radiation is confined to the region of visible deposits he can only hope for palliation. He must remember that
in the long run the naked-eye manifestations of the disease are less important than its invisible microscopic extensions by which it ultimately reaches the vital organs.

(b) That microscopic foci may lurk in the supraclavicular or the anterior mediastinal glands of the same side. Of course the whole region of the growth must be irradiated, but if a method of operation has been employed aiming at extirpation of the microscopic growing edge, recurrence, if it takes place, will usually be late and will be found at the inner edge of the second, third, or fourth intercostal spaces or in the supraclavicular triangle of the same side. It is a question, therefore, whether the dangerous regions thus indicated should not receive an extra dose of radiation.

(c) In late cases where the surgeon has found advanced infection of the axilla the lateral chest wall must receive special attention. Modern methods of operating have almost abolished axillary recurrence, but in advanced cases radiation of the axilla with the arm abducted is advisable.

It is not within the scope of this paper to consider the methods and dosage of radiation. But pathology and lymphatic anatomy must be as fundamental for the radiological as for the surgical treatment of cancer. In that belief I offer no apology for the claim upon your attention which I have made to-night.

NATURAL CURE OF CANCER.

It is not possible to form a correct estimate of the value of radiation in cancer without taking into account the natural tendency of the disease to undergo local repair. In a lecture on this subject I came to the conclusion that: "Every aggregation of cancer cells after increasing in size for a varying period and for a varying rate, tends spontaneously to undergo certain degenerative or regressive changes. These changes begin at the centre of the mass, spread centrifugally to its circumference and may terminate in the replacement of the mass of cancer cells by a fibrous scar."

These changes, which are exemplified in the ulceration of a primary growth, and in the umbilication of secondary deposits in the liver, appear to depend upon a breakdown of the improvised vascular commissariat of the growth and upon the harmful internal pressure produced by the active proliferation of the cancer cells. These degenerative changes can be seen beginning even when the collection
of cancer cells is still of microscopic size, as is a permeated lymphatic. Their onset is invariably accompanied by the advent to the neighbourhood of collections of lymphocytes. It is thus possible that in every carcinoma we have to deal with a comparatively small number of perfectly healthy and active cancer cells, and with a large mass of more or less degenerate or dying cells. The former class of cell is to be found in the permeated lymphatics of the microscopic growing edge, and at the edge of macroscopic nodules of growth which are still in the actively infiltrating stage. It seems likely that these two classes of cell differ widely in their reaction to radiation. It is probable that the crowds of degenerate cells which constitute the mass of a carcinoma may fall a ready prey to the reaction of radiation. Even the earliest attempts at the destruction of secondary nodules by X-rays were often locally successful. The shrinkage and disappearance of local masses of cancer may possibly indicate nothing more than the acceleration and completion of this natural process of destruction.

The real problem is not to dispatch the wounded but to attack the cancer cell in full vital activity, and to determine how it responds to radiation as compared with the degenerate cancer cell and what is the amount and character of the radiation necessary to destroy it. Such a research is quite practicable, but necessarily laborious. It would require the co-operation of a radiologist and a histologist. Cases of breast cancer showing a moderate and regular dissemination of subcutaneous nodules round the growth should be selected, and a sector of the microscopic growing edge just beyond the region of visible nodules should be submitted to radiation, and compared post mortem with untreated sectors of the microscopic growing edge.

So long as investigation is directed to macroscopic masses of mostly degenerate or dying cells its results must remain indeterminate and confused. Definite results can only be obtained by experiments upon the standard healthy cancer cell of the microscopic growing edge, not the edge of the primary growth or of any of the visible secondary deposits, but that of the ripple of permeation which is extending from the primary growth through the patient's lymphatic system. The detection of the microscopic growing edge of permeated lymphatics provided the key to the understanding of the process of dissemination. It is quite possible that a careful study of its behaviour under radiation may supply a master-key to the therapy of cancer.
DISCUSSION.

Dr. FINZI: We are under a debt of gratitude to Mr. Handley for his work in this subject, which I can assure him most radiologists have studied for many years. I myself have obtained the most valuable information from it, and have based my method of treatment of these cases largely upon his discoveries. I should be glad of Mr. Handley’s opinion upon a few points to which I shall refer. In the first place, it has been suggested that as X-rays cause absorption of fibrous tissue, the irradiation of fibrosing lymphatics might release the cancer cells and prevent the natural process of healing, which was occurring in these lymphatics. I think we have nothing to fear on this score, the reason being that the dose of X-rays required to produce the absorption of fibrous tissue is much greater than that which is lethal to the carcinoma cell, consequently the carcinoma cells are killed before any absorption of fibrous tissue takes place. My next point is a matter in which I am sure Mr. Handley has given a wrong impression inadvertently. He has tended rather to belittle the value of irradiating the axilla. Now when the axillary glands become invaded the growth proceeds in them, and after a time grows out of the glands and spreads by infiltration and permeation, with the axilla as a focus, in the same way in which the primary growths spread, and also at this time spreads to the supraclavicular glands. It is therefore essential in every case thoroughly to irradiate the axillary and supraclavicular regions as well as the large skin areas around the breast to which Mr. Handley has already referred. With regard to the relative value of radium and X-rays. Owing to the necessity of thoroughly irradiating a large surface, I have for many years recommended the use of X-rays in these breast cases in preference to radium, not because the X-rays have more effect on the cancer cells—I believe they have considerably less effect—but because it is possible to irradiate thoroughly a very much larger area. With the quantities of radium we use in this country this is impossible. In America a certain institution has at its disposal as much as one and a half or two grammes of radium bromide, which it can use on one patient at a time: to this institution these remarks would not apply: it can obtain results which we cannot hope for with the smaller quantities at our command. What is Mr. Handley’s opinion as to whether the lethal dose of X-rays for a carcinoma cell is the same at all stages of the disease? Personally I believe it is not. I think that as the disease progresses the lethal dose becomes larger and larger until it approximates to that which would damage the healthy cells, this of course being due to some vital reaction, stimulated by radiations, on the part of the tissues and the body against the growth. As an instance, I would cite the case we all know in which a carcinoma does well for a time and then goes ahead whether one increases or decreases the dose, or stops it altogether. I would suggest that the different behaviour of different cases of apparently the same disease is due to this cause. With respect to prophylaxis: will Mr. Handley
tell us whether he has used irradiation before the operation? It need not delay the operation in any way, as it is possible to give a sufficient dose within three-quarters of an hour with modern apparatus. The principle on which this is based is the fact that has been clearly established, that a mouse carcinoma which has been given a sufficient dose of irradiation cannot be implanted. Mr. Handley must remember that all surgeons do not employ the same operation as he does, and one has to deal with the average: there is no question that a number of cases occur in which carcinoma cells are implanted at the time of the operation. Now, as it has been found that an efficiently irradiated mouse carcinoma cannot be successfully inoculated into another mouse, an efficient radiation of the growth before the operation would render implantation impossible. The optimum time to irradiate is within a fortnight of the operation. With regard to technique. For many years I have used wooden points similar to the Kienböck points so as to keep the tissues at the correct distance, and in overlapping each area of exposure it is quite easy to calculate mathematically the amount of irradiation given as a result of the overlap, which should of course never exceed the maximum skin dose. Another thing is to have the opening of one's tube shield as large as possible: most of those made are much too small: personally I use one which is 4½ in. wide with the tube quite close to it. By this means one is enabled to treat efficiently very large areas of skin around the original growth or the recurrence.

**Mr. Handley (in reply):** I do not wish to minimize the importance of X-ray treatment of the axillary glands. It is doubtless true, as Dr. Finzi said, that an infected axillary gland in time acts as a focus of further spread, but in the past undue attention has been directed to the axillary glands to the neglect of more important avenues of spread. Regarding the question of pre-operative irradiation, I have only made use of it when for some unusual routine the operation has had to be deferred. Dr. Finzi has, however, brought forward very strong arguments in its favour as a routine procedure, and I propose in future to adopt the practice.
Section of Electro-Therapeutics.

Joint Meeting with the Institution of Electrical Engineers.¹

Mr. Cecil R. C. Lyster, and later, Mr. Wordingham, President of the Institution, in the Chair.

Mr. Lyster: Ladies and Gentlemen,—It is a peculiar pleasure to me to-night to welcome the Institution of Electrical Engineers. I think that we medical men have isolated ourselves, possibly, a little unnecessarily, and this meeting I have been looking forward to with great interest. We have a great deal to learn; we are amateurs in electricity, and we are at last coming to our senses and asking the professional electrician to tell us what we want. We all, in medicine, know, or imagine we know, that if we can get a certain result from electrical treatment, we shall be able to do the patient a great deal of good; but, as amateurs in electricity, it has not been easy—in fact, it has been a great struggle for us to attempt to do this. This is the first combined meeting, and I hope it is a meeting of the sort of which we shall have a continuance in years to come, and that we shall be able to interest the Institution of Electrical Engineers in our work as electro-therapeutists and radiologists. It is a fascinating subject, and a far-reaching one for humanity—that is, the future of the electrical and radiological treatment of disease: perhaps my optimism is enormous. I shall ask the President of the Institution of Electrical Engineers to take the chair for this evening.

(Mr. Wordingham took the Chair.)

Mr. Wordingham (President of the Institution of Electrical Engineers): Ladies and Gentlemen,—I would like to say how very cordially the Institution of Electrical Engineers appreciates the hospitality of the Royal Society of Medicine in asking us to take part in

¹ Held March 21, 1919.
this Joint Meeting in their building here. I entirely endorse every-
thing which Mr. Lyster has said in regard to the benefits that we may
expect from co-operation. We certainly, as electrical engineers, have
everything to learn in regard to the medical side, and we hope that if
we can be made to understand the conditions which have got to be
fulfilled, we shall not be found wanting, as designers and as inventors,
in meeting those needs adequately. The great thing for us to know
is what is wanted from the medical side, and then we will see what
we can do. I will not detain you with remarks of my own, because we
want to get on to the main business of the meeting. Last year we had
a Joint Meeting with the Society of Radiology and Physiotherapy, and
it was one of the most successful meetings of the whole of our Session,
and I can safely say that the members of our Institution thoroughly
enjoyed the meeting in every way. And not the least part of the
enjoyment was in connexion with the exhibition of apparatus which we
saw afterwards at the Cancer Hospital. Again, to-night, the Royal
Society of Medicine have arranged for an exhibition of apparatus
which, I am sure, will be equally interesting, and they have very
kindly arranged to keep that exhibition open until to-morrow afternoon,
so that those who are not able thoroughly to examine the apparatus
to-night, or who cannot come to-night, will have the further oppor-
tunity afforded them. We have two papers before us this evening, both
by the same author, Mr. Robert S. Whipple, a member of our own
Institution; but I propose to treat him as two separate people, and to
ask him to read first the paper entitled "Some Notes on Electrical
Methods of Measuring Body Temperatures." We will take the dis-
cussion on that, and then proceed to the second paper, which he will
then read, just as if he were a separate author.

**Electrical Methods of Measuring Body Temperatures.**

**By Robert S. Whipple.**

(ABSTRACT.)

[This paper will be published in extenso in the Journal of the Institution of Electrical
Engineers.]

Becquerel and Breschet, in 1835, were the first to determine the
temperature of animal tissues by electrical means; they used a single
pair of copper-steel couples which were thrust into the animal's tissues.
In 1908, the late Professor Arthur Gamgee commenced researches on
the subject, and in his paper on "Clinical Thermometry" pointed out
the importance of the study of body temperatures, especially in observa-
tions on the preliminary stage of phthisis, in which physical signs of
the disease are almost in abeyance, if not entirely so. Gamgee decided
to adopt thermo-electric couples in preference to resistance thermometers
for high precision work. He employed two kinds of thermometer: one
for taking the temperature of surfaces of the skin, the other for testing
the temperature of body-cavities. The couples were made of copper-
constantan. In such couples temperatures are determined by measuring
the electro-motive force in the circuit, this having a definite relation to
the difference in temperature between the two junctions of the circuit.
This demands the control of the temperature of one of the junctions,
generally the colder of the two, which is termed the "cold junction,"
the difference in temperature between it and the "hot" junction (the
thermometer in contact with the human body) being determined by
the electro-motive force recorded by the galvanometer.

Gamgee put his cold junction in a water-bath of exactly controlled
temperature. Modifications made by Sims Woodhead and Varrier-
Jones\(^1\) have enabled the cold junction to be kept for long periods of
time at a constant temperature—i.e., within 0.01° C. Both skin and
rectal thermo-couples were described in full detail, with the aid of
lantern-slides. The thermometer can be left in the rectum for
twenty-four hours or more without inconveniencing the patient. The
wires from it are led to the recording apparatus, a moving-coil galvano-
meter of the d'Arsonval type, the movements of which are recorded by
an inking device, either photographically or autographically. Later
views favour the employment of resistance thermometers, as they
require no elaborate thermostat, and a tenfold larger deflection can be
obtained with the same galvanometer. The author drew attention to
the considerable difficulty of making satisfactory surface thermometers
and attaching them to the patient. The rectum thermometer designed
by Dr. Varrier-Jones consists of a fine platinum wire wound round a
mica frame in the form of a cross, the coil on its frame being enclosed
in a silver tube, this tube being constructed in such a way as not to
irritate and therefore stimulate the sphincter. The coil is connected
with the recording instrument. In practice the instrument is simple
to operate, and its manipulation is learnt within an hour or so. It will
record the temperature of the body within five minutes of its insertion.

\(^1\) Lancet, 1916, i, pp. 173, 231.
DISCUSSION.

Professor Sims Woodhead, M.D.: I think you will realize, after hearing the admirably lucid and interesting paper Mr. Whipple has given us, what an advantage it must be to us to have had the Cambridge Scientific Instrument Company interested in Professor Gamgee’s work, and then in the continuation of that work. Without technical skilled assistance, and without the great scientific and technical knowledge available at the Cavendish works, it would have been impossible for us to obtain anything like the very satisfactory results which have been achieved, first, as regards apparatus, and secondly as to the products, the temperature records. I say this advisedly, because when we had the apparatus we often found that it was a somewhat difficult matter to bring it down to a good practical working level. Professor Gamgee’s apparatus is a really beautiful one; with it you can obtain exceedingly accurate temperature records with very little lag, and you can, as Mr. Whipple has said, obtain it to the fineness of one two-hundredth of a degree. With the resistance thermometer it is not possible to do such very delicate work, but you can obtain results which are of enormous practical value. To-night, in order to show what can be done with these resistance thermometers, there will be thrown on the screen a number of photographs of our records which Mr. Whipple has had prepared. I should like to say a few words about these, and especially to draw attention to one or two marked features in the diurnal variations of temperature of a normal individual, and then compare them with the variations in a tuberculous patient. I first turned my attention to continuous thermometry about 1879, and then, curiously enough, I learned that Professor Gamgee one or two years before, had already been engaged on the same kind of work. My old teacher, Professor Tate, told me that it was practically impossible to record thermogalvanic current as Gamgee had already found that there was no suitable galvanometer extant. When Gamgee came to Cambridge and recommended this work I, as were others, was naturally keenly interested, and I think anyone who then saw his work must have realized at once how vastly important was this or some similar method of recording temperatures. Those who have had experience of temperature work and especially of temperatures of tuberculous patients tested with tuberculin—and this is especially well known in the veterinary profession, in which tuberculin-testing has been of very great importance—recognize what a number of very difficult factors, or points very difficult of explanation, arise in connexion with these tuberculin tests: the difficulty, for example, of determining the highest and the lowest temperature of the pre-tuberculinized patient and of the tuberculinized patient. If, however, a continuous or a semi-continuous record can be obtained over three days—twenty-four hours before tuberculin is given, twenty-four hours after the tuberculin is administered, and a further twenty-four hours to determine how long the tuberculin continues to act—the tuberculin test comes to be much more reliable. It has been said to be wanting in reliability in 5 per cent. to 10 per cent. of the cases tested; that is, in this percentage of
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cases it has not given the results expected, or those that, apparently, should have been obtained, taking into consideration the conditions found after death. It is easy to determine the differences between the temperatures of the normal or pre-tuberculin and the tuberculin stages; note how the temperature varies, as also the character of the temperature curve; the point of greatest difference; the point at which a gradual fall of temperature occurs, and how, in the tuberculous or tuberculinized patient the variations are exaggerated under the influence of food, water, warm fluids, and the like. If the variations are considerable in the normal or pre-tuberculin stage, they are considerably greater—three times as great—when tuberculin has been administered. The same exaggerated variations characterize the temperatures of tuberculous patients. Variations are invariably increased when tuberculin is given, even in minute quantities, to such patients. One finds, too, that the temperature, under these conditions, is extremely unstable, and that the variations are very rapid. Instead of having a comparatively straight line with easy curves, the line becomes dentate, the "teeth" apparently very blunt in the "normal," becoming very sharp and prominent in the tuberculinized patient, or in the tuberculous patient, under the effect of exercise, hot and cold fluids, and the like. In an animal, distinctly tubercular, some of these changes are very marked.

In a record from a healthy cow which had been injected by Dr. Varrier-Jones with a full dose of tuberculin—6 c.c.—there is at one point a slight rise, to 103° F., of comparatively short duration, which falls fairly rapidly. For the remainder of the curve the temperature is practically normal. On the curve the minimum temperature marked is 100.6° F. During twenty-one hours the temperature runs between 101° and 102° F., fairly continuously after the first slight rise, which rise however does not occur in a normal animal to which no tuberculin has been given. Small depressions noted are the result of the ingestion of food and water; some of the peaks are the result of exercise: after the depressions resulting from taking food and water there usually is a slight and steady rise, apparently the result of an increase in the amount of blood in the vessels in the walls of the alimentary canal.

In a calf which had been rendered tuberculous experimentally three twenty-four-hour records were taken, as noted above. During the twenty-four hours before tuberculin was given the temperature ranged from 100.5° to 103.2° F. In the twenty-four hours following the exhibition of tuberculin there is, almost immediately after injection of tuberculin, a rise of temperature: the two curves when superposed diverging somewhat rapidly up to 105.4° F., at the end of eleven and a half hours, followed by a dentate or serrated—very characteristic—falling line to 103°, when there occurs a rapid fall of 1.2° F., which indicates the considerable change of temperature which is induced by a draught of water in a tuberculous animal. Had the animal not been tuberculous, that fall would not have been more than 0.3° or 0.4° F. The dentate line and the marked divergence for a long period of pre-tuberculin and tuberculin curves constitute very characteristic reaction manifestation; a pronounced divergence over a short
period is not nearly so characteristic, but what we call a "plateau curve," i.e., a prolonged elevation, is a sure indication of a "positive reaction."

In a doubtful case of tuberculosis the second twenty-four-hour—post-tuberculin—curve, runs considerably higher than the pre-tuberculin curve, this indicating the prolonged effect of the tuberculin. (Later the diagnosis was confirmed.) Here the "plateau" is distinct though the divergence of the two curves is not nearly so marked as in the last case.

Coming now to the human subject. In a tuberculous patient the pre-tuberculin temperature ran between 98° and 99°3° F., during the six hours between 12 noon and 6 p.m. There was then a steady fall, until 6 o'clock in the morning, apparently, the time at which the functional activity of the body is at its lowest, and perhaps, in this case, at the period of deepest sleep, when the lowest point, 97°4° F., was reached. Then comes a steady rise, characteristic also of most fairly normal temperatures. After the exhibition of a small dose of tuberculin the "plateau" is again a very characteristic feature: instead of a short maximum period, this is greatly prolonged, and the minimum temperature at a corresponding hour is at least half a degree higher than before the tuberculin was given. This is almost invariably repeated in the next twenty-four hours, but in modified degrees.

In two other tuberculous cases a tuberculin (plateau) temperature was noted. At 3 in the morning there was in both a very sudden fall, this usually corresponding to the period of the night-sweat. After that the falling curve is distinctly unstable, becoming somewhat more stable as it rises when the patient awakes rested and refreshed. In the twenty-four-hour chart the plateau, twenty-four hours after the tuberculin was given, appears at the same period as during the twenty-four hour tuberculin period, and we have a demonstration of the continuance of the effect of tuberculin for at least forty-eight hours.

From this form of record much information which, up to the present, we had not been able to obtain, may be gleaned not in tuberculosis only, but, I think, in all fevers. Some time ago I pointed out that in regard to trench fever we might be able to obtain very considerable information by the application of this method.

With the apparatus as finally devised a range of 18° in three series, 88°-98° F., 96°-106° F., and 102°-112° F., may be determined with extreme accuracy, whether the temperature range be high or low.

I can only say, in conclusion, that I hope other diseases will be explored by this method. This can be done very easily, as with the apparatus in your laboratory the patient on whom the observation is being made may remain in bed in the hospital. Much of our work was done on patients in Addenbrooke's Hospital, with the apparatus in the basement floor of the laboratory. I regret that I did not realize earlier how easily this distance recording may be done, as during the earlier stages of the investigation I spent many weary nights in that basement; and I might have spent them comfortably in the hospital 600 yards away. We have carried out most
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satisfactory and convincing tests on animals on which control and verification are more easy than in the human subject. Before the war, however, Dr. Varrier-Jones and I had sketched out a plan of work on the temperature of tuberculous patients and many records have already been obtained, though not enough. We hope before long, however, to complete our series and to study them carefully. As regards the apparatus we are now satisfied that it has the following advantages: wide range; it is not too delicate and is easily worked; it has little "lag," the records are quasi-continuous (every half minute); are not under the eye of the patient and may be recorded at a distance and so centralized.

Dr. P. C. Varrier-Jones: I have not much to add to what Professor Sims Woodhead has told you about the methods of recording temperature by means of the continuous and quasi-continuous apparatus described this evening. For some years we worked at temperature recording with thermo-couples, but when we turned our attention to taking temperatures of cattle over long periods in association with the tuberculin test, we found that the thermo-couples were rather too delicate for the work we had in hand. The resistance thermometer was, however, found very suitable for taking the temperature of cattle, and the work, whether on animals which were suffering from tuberculosis or on those which were in normal health, was very instructive. The instrument was not so liable to break as were the thermo-couples designed by us, and was admirably suited for work on the delicate tuberculin test. In the case of cattle we could take very long records and observe the pre-tuberculin curve, the post-tuberculin curve and the late post-tuberculin curve. With the thermo-couple thermometer it was a simple matter to take records of patients over half an hour or two hours, but we wished to have much longer records over long periods of time, and the difficulty was to retain the thermometer in situ for the longer period, especially in cattle. With the resistance thermometer we were fortunate in so arranging it that we could take records for three or four days. We could thus see for exactly how long a drug acted upon the animal. With regard to patients, we find the apparatus as now designed simple to use, easily fixed in position, is accurate and reliable, does not easily get out of order, and the patient does not object to it. We are now working at the question of determining the early diagnosis of phthisis in human subjects. With regard to the temperature of persons suffering from phthisis, we find we can reproduce in the patient the exact condition we have in calves which have been experimentally tuberculinized: the records are practically identical. So from the very small rise in an early case of phthisis, if we give a small dose of tuberculin we can produce the "plateau" of temperature, and by means of exercise in a person with well-advanced disease, produce the inverted curve which is characteristic of an animal that has got a great amount of tuberculous disease. Patients do not object to these records being taken over considerable periods of time, and the little rectal thermometer can be used for twenty-four or forty-eight hours
with but one remission in the latter case. The skin thermometer can only be used for very short periods of time, after which the instrument becomes unreliable owing to the great difficulty in maintaining it in close proximity to the body-wall. But the rectal resistance thermometer can be used with absolute certainty at the present time and for long periods. The apparatus is simple and is easily standardized. By the simple means of a switch the record is started: we can then, if we wish, leave it alone and come next day and take out the record.

Professor PORTER: The difficulty there is in the determination of a surface temperature such as the temperature of the skin is extraordinary; the determining of rectal temperatures is a different matter. The determination of the actual temperature of a point on the surface is, as every physicist knows, a matter of great difficulty, and I am not quite sure to what extent that difficulty has been overcome in the case of the simple thermo-couple arrangement which has been described. There is probably no serious defect in it so far as the determination of variations of temperature is concerned, and that measurement, I suppose, is what especially concerns the medical man. The actual temperature is probably quite uncertain as so determined. The difficulty is that no matter in what way you apply the thermo-couple, you must modify the conditions of radiation, and so on, in the neighbourhood of the point of application, and especially when the thermo-couple itself is surrounded by an india-rubber container, as I understand it is. That container, when placed on the skin, must of course tend to prevent not only radiation but also evaporation from the surface, upon which largely the temperature of that surface depends. I would like to know to what extent experiments have been made in order to determine the magnitude of this disturbance. It might be better if the actual extent of the surface pad were reduced. I observed in the diagram that the thermo-couple itself is placed near one end of the pad. Why should the pad be more extensive than the area represented by a square of 1 cm. area of which the thermo-couple is the centre? The rest of it, if it is intended as a support—as no doubt it is so intended—for the leading-in wires, might be placed at right angles to the surface. In such case there would be less tendency to disturbance in the surface conditions than in the actual arrangement. You must remember that the pad plays the part of a small overcoat in its immediate neighbourhood, and it is not like taking the temperature of an inorganic body: in the living object you have reactions setting in, no doubt at once, as soon as the coat is put on, and you are taking the temperature under those modified conditions. But it is an extraordinarily difficult thing to determine in any surface. In every other respect the make of the instrument as Mr. Whipple has described it shows a tremendous amount of ingenuity.

Dr. RANKINE: To a physicist I should say that the use of the thermo-couple as a means of determining the quickly changing temperature of a subject would appeal very strongly. It will have very little thermal capacity,
and should therefore record the temperature very quickly after being placed in position. I was very interested in seeing the diagrams which Mr. Whipple showed in connexion with the thermostat which was used to keep the second juncture of the couple at constant temperature. The difficulty was to understand why it was necessary in practice to have such an elaborate arrangement. The obvious thing would seem to be to put the second junction, not into the thermostat, but into a suitable mixture of ice and water, the temperature of which would be constant without elaborate precautions. If you did that, it is true you would be measuring a larger difference of temperature, but it should be possible by external means to control the movements of the galvanometer suspension, so as to keep the pointer on the scale, or the spot of light on the photographic paper. A suggestion has just occurred to me. I suppose it is admitted that, from the medical point of view, it is the difference of temperature between that of the patient and the normal body temperature which is the important thing. It is clear, from the diagrams which have been shown us, that there are ordinarily variations to be expected, quite apart from illness, and I wonder if it is out of the question to think of the possibility of having a sort of standard man who is kept always in good health and to whom the second junction of the thermo-couple is kept permanently attached.

Mr. E. H. Rayner: I do not think Mr. Whipple has done justice to the apparatus by leaving out certain technical electrical data which should be included. He, and other speakers, mentioned the fact that something of the order of one-hundredth of a degree centigrade can be measured in practical working with an apparatus of this nature, both in the resistance method and in the thermo-couple method. To show what that actually means one may take it that ordinary pure metals change three or four parts in a thousand for each degree centigrade, so when you are reading to one-hundredth of a degree you are measuring, with reasonable accuracy, changes of three parts per hundred thousand. To do that with an electrical laboratory apparatus is not difficult; to do it by a self-recording apparatus not in highly skilled but only intelligent hands, to do it in a hospital by the bedside, is a very great achievement. When dealing with the thermo-couple for measuring temperature one naturally chooses the most convenient pairs of metals or alloys which will give a large thermo-electrical voltage per unit difference of temperature. For many purposes copper and iron ore are used, or a nickel-copper alloy against iron, and for each degree difference of temperature of your two junctions, the best you can get in practice is one twenty-thousandth of a volt, except by the use of metals which are unsuitable for general purposes; so that indications to one-hundredth of a degree mean that a two-millionth of a volt is just appreciable. This can be done in a special laboratory, but to do it under hospital conditions is again a great achievement. Perhaps Professor Sims Woodhead will tell us whether tuberculin does produce any reaction on quite healthy people.
Professor Sims Woodhead (in reply): Many control observations were made with the ordinary clinical thermometer carefully graduated; these, carefully timed and recorded, were compared with continuous temperature records. By this method of trial and error we obtained and controlled the actual temperatures being recorded. As to the thermo-couple thermometer with which I experimented in 1879: we tried—and I may say Dr. Varrier-Jones spent much time, ingenuity, and trouble in devising—a thermometer of small mass and limited surface. We worked with fine metal wires, to which were fused thicker and thicker wires. The couple wires were so thin that it was very difficult to support them on anything but a flat surface of considerable area. We hope in time to diminish the surface area, but as we are working now with the "resistance" rectal thermometer, we have not been able to give very much attention to the surface thermometer. Tuberculin has very little effect on the temperature of a normal individual. I think the first slide showed that. There is but a short period and small range of elevation.

The Chairman: I understand there are a number of American gentlemen present, and perhaps they will be good enough to tell us what they do on the other side on this question. I cannot believe they have done nothing.

Dr. E. P. Cumberbatch: I understand from Mr. Whipple that his apparatus will record such small variations as one-hundredth of a degree centigrade. If that is the case, it ought to be of considerable use in determining the passage through the tissues of electrical currents of high-frequency oscillation. During the past few years I have been using these currents for raising the temperature of different parts of the body, especially the interior parts. From the inquiries which I have made amongst physicists, I have been unable to discover exactly what is the path of these currents through the tissues. I have used them in attempting to raise the temperature of interior organs of the body, such as the uterus, the liver, the spleen. I have connected a plate electrode to a diathermy or sustained high-frequency oscillation apparatus, and connected the current to electrodes on different sides of the body, so as to include between them, in the depths of the tissues, the organs the temperature of which I wished to raise. Recently, I have conducted experiments to determine the temperature of the interior of the body when passing the diathermy or high-frequency current through. In one patient there was a deep sinus passing down to the kidney region, and I placed a clinical thermometer in the depth of the sinus, and an electrode on the front of the trunk, and another electrode on the back of the trunk. After the current had been passing twenty minutes, the clinical thermometer showed no elevation of temperature, so it appeared that the current travelled along the peripheral region of the body. If, as I say, we have here an apparatus which can detect one hundredth of a degree centigrade, it should be of great use in determining, more exactly, what is the path of a current through the body as shown by its power of raising the temperature of the deeper tissues.
Mr. F. C. Raphael: It is of very great advantage that these instruments should have been given to medical men a method of accurate quantitative measure-
ment they have not had until comparatively recently. The instruments are
very wonderful and beautifully worked out, but in considering their delicacy
we must not base the conception of their accuracy on the fact that they
measure to one-hundredth of a degree in a measurement of about 100
degrees. In both the thermo-junction and the Wheatstone bridge method,
it is a measurement of a difference in temperature of a few degrees only that
is being measured to an accuracy of one-hundredth of a degree. Consequently
Dr. Cumberbatch need not despair of being able to apply the apparatus, or
similar apparatus, to the measurement which he has indicated. What I have
said also answers the remark as to why the so-called cold junction should not
be put in melting ice. The reason is, that it is expedient to keep the cold
junction at a temperature very nearly equal to the temperature which one is
measuring, because what is being measured is a difference, and one wants that
difference to be as small as possible, so that the percentage inaccuracy allow-
able in the measurement to correspond to a definite permissible absolute error
in the result may be as large as possible.

The Chairman: If there are no other speakers, I will close this discussion,
as time is getting on, and I think it will be well to ask Mr. Whipple to reply
to the discussion in writing. We can proceed at once, in that case, to the
second paper: “Some Notes on the Electrocardiograph.” If we begin at
once we shall not have to curtail discussion on the second paper.

Mr. R. S. Whipple (reply communicated): Professor Porter touched on
the difficulties to be overcome when the skin thermometer is used. In
conjunction with Professor Gamgee a great many experiments were made to
find the best type of mounting for the sensitive element. Gamgee thought
that the mounting over the couple tended by conduction to lower the tempera-
ture of the skin at the point of contact, so that he aimed at reducing heat
losses rather than preventing evaporation from the surface of the skin. To do
this to the maximum extent, a couple was mounted on the face of a small flat,
exhausted and silvered glass vessel. This should have almost entirely prevented
radiation losses. It was found to give no more satisfactory results than the
cloth pad. The great difficulty is to obtain intimate contact between the
thermometer, whether it is of the resistance or thermo-couple type, and the
skin. If a thin film of grease is introduced between the thermometer and the
skin, the result is better than if only the moisture of the skin is relied upon.
A great many forms of mounting were tried with pads of various areas and yet
no absolutely satisfactory method was found for keeping the axilla thermometer
in contact with the skin. On the other hand the rectum thermometer is
surrounded by moisture, and hence its more satisfactory behaviour. As Mr.
Raphael pointed out, it is not possible to use ice and water as a method of
keeping the cold junction at a constant temperature as suggested by Dr.
Rankine. Temperature differences are being measured, and the difference
between body temperature and the temperature of molten ice is far too great to be readily covered by the sensitive galvanometer used in these experiments. The "standard man" suggested by Dr. Rankine has already been designed and used in the form of a very small electrically heated space, the temperature of which is controlled by a bi-metallic thermostat. It works well, but as the water bath is more satisfactory in practice I did not mention it. I agree with the statement that an accuracy of one-tenth of a degree centigrade is sufficient for nearly all medical work; Professor Gamgee undoubtedly aimed for an accuracy of one one-hundredth of a degree centigrade. With regard to Dr. Cumberbatch's remarks, I think that it may be possible to measure the rise in temperature of the surface of the skin when a patient is undergoing high frequency treatment. There is the risk that edy-currents in the thermometer may vitiate the records, and there will undoubtedly be other difficulties, all of which can only be determined, and perhaps solved, experimentally.

Some Notes on the Electrocardiograph.

By Robert S. Whipple.

(ABSTRACT.)

[This paper will be published in extenso in the Journal of the Institution of Electrical Engineers.]

Professor A. D. Waller was the first physiologist to demonstrate that the electric potentials developed in the heart at each contraction were sufficient to deflect a sensitive galvanometer connected to the limbs of the human subject. Little progress in the development of this discovery was made until Professor Einthoven, of Leyden, invented the string galvanometer in 1901. This had a powerful electromagnet and a comparatively short silvered quartz fibre as the moving system. He used a single stretched fibre. The cardiograph now in use preserves these main features, the "string" being a quartz or glass thread 0·003 mm. in diameter. The following apparatus is required to obtain an electro-cardiogram: (1) The galvanometer, to receive the electric impulses of the heart; (2) the camera to record the movement of the fibre in the galvanometer; (3) a time-marker, or mechanism for marking time intervals on the photographic plates; (4) a source of illumination to enable photographs to be obtained; (5) the electrical equipment for making the necessary standardization and various other adjustments; and (6) the electrodes for making electrical connexion to the patient.

1 Lancet, 1912, i, p. 853; 1913, ii, p. 380.
DISCUSSION.

The Chairman: Ladies and Gentlemen,—We have had superstations very much in our minds of late, but I suppose it is rather a revelation to many of us that we have been carrying a micro-station about in our hearts. Much of our time has gone, but we have a few minutes left for the discussion, and I will ask Dr. Thomas Lewis if he will kindly open the discussion.

Dr. Thomas Lewis, F.R.S.: In the first place I should like to congratulate Mr Whipple very much upon his lucid demonstration, for which I am sure we all feel much indebted to him. I heartily endorse the remarks Professor Sims Woodhead made as to the co-operation between physiologists, clinical investigators and those who are engaged in the invention and the production of scientific instruments. Mr. Whipple gave us in his first demonstration an example of the effects of such co-operation. His second demonstration provides an equally good illustration. It was my privilege, ten years ago, to introduce to clinical medicine in this country the electrocardiograph. The first instruments I used were the Edelmann instruments. After I had been a long while working with these instruments, and wasting much time with them, I had the good fortune to meet Mr. Whipple, and during the last eight years my laboratory at University College Hospital Medical School has worked in active co-operation with his firm; I cannot speak too strongly as to the advantage which has come to us through that co-operation: it has been a very great advantage. If we, as clinical workers, wished for a particular adjustment which would render our work easier and give us speed—and speed is of very considerable consequence to us in our hospital work—we had only to mention the need of such adjustment to the firm’s representatives, and they quickly put that adjustment on for us, in a form effective to save us much time and a great deal of labour. The apparatus, as Mr. Whipple shows it this evening, is essentially a clinical installation: it is set up for clinical purposes, and many of the adjustments on the galvanometer and much in the general arrangement of the switchboard, have come as a direct result of the co-operation between the two laboratories. The instrument, as it now stands, is meant for speed in the taking of accurate records, and when I tell you that three curves may be taken of a patient’s heart in a few minutes, whereas in the old days it would take, perhaps, half an hour to secure the same curves, you will see at once what an extraordinary gain of time there has been through such adjustments having been adopted. Now, if I may do so, I will show you a few clinical records, the interpretation of a few curves from patients, in order to illustrate the value of such an apparatus as Mr. Whipple has demonstrated to you.

This first slide, a reproduction of the actual curves, represents the normal electrocardiogram, and is very similar to those Mr. Whipple has shown you. I take one cycle of the heart-beat. You have, with each single heart-beat a
complex series of deflections of the string. These deflections may be divided into two sections or parts. The heart consists of two parts: the auricular portion and the ventricular portion. Each of those portions gives rise to an electric current in the body, which is registered by means of this apparatus. The auricular portion of the heart gives rise to this small deflection, which is labelled "P." The auricle starts to contract in the neighbourhood of this point, and pumps blood into the ventricle. The contraction of the auricle ceases at approximately this point, and then comes the contraction of the now fully-engorged ventricle, and with that contraction of the ventricle you get this series of larger deflections. The first point I would make in regard to the machine and its value for clinical purposes, is, that it gives a separate record of the auricular and of the ventricular movements. In normal hearts, there is an orderly sequence between the contraction of those two chambers: there is the contraction of the auricle first, pumping its blood into the ventricle, then contraction of the ventricle, pumping its blood into the arteries of the body. You see this orderly sequence expressed in this curve. But it is not always maintained in conditions of disease: frequently there is a disorder in the sequence of action of those two chambers.

Mr. Whipple referred to the standardization of electrocardiograms, and I have put in this second slide to show you the purpose of that. It is necessary to standardize curves, so that we can take a record of the same patient by means of different machines, and yet obtain similar records. In that way, workers in different laboratories can compare their separate results very accurately. Here is a record taken of a patient in Professor Einthoven's laboratory, and with it is a record from my own laboratory of the same patient. You see how closely they correspond in their amplitudes and in their general shapes.

I have said that there is sometimes a lack of harmony between the beating of the two chambers of the heart. One of the chief forms of lack of harmony is what is spoken of as "heart block." The ventricle depends for its impulse to contract on the contraction of the auricle. It waits to receive the impulse from the auricle, and contracts a little later. In disease, that period of delay between the contraction of the auricle and ventricle is frequently greater: instead of the auricular contraction being immediately in front of the ventricular, it stands back at double the distance, and from time to time, as you see in the present slide, there is an auricular contraction in the absence of a corresponding response from the ventricles, that is to say, the ventricle fails to contract. This record shows clearly the nature of a disturbance which leads to irregularity of the heart's action and of the pulse. To go a step farther, the difficulty of conduction may be very much greater, and may be absolute in some cases. There are patients in whom the auricles and ventricles beat at separate rates, the auricle beating much faster than the ventricle. I show you a record of this kind. The sharp deflections are the contractions of the ventricle, the smaller deflections are the independent contractions of the auricle. It is a very perfect demonstration of what is spoken of as dis-
association between the two chambers, a very important demonstration because it throws very considerable light on the pathology of a serious disease.

I will show you another method in which this machine is used. In attempting to analyse irregularities of the heart's action in the human subject, it is frequently necessary to attempt to create in animals precisely similar disturbances: then one is oftentimes able to understand the meaning of the human irregularity. These two curves I show you for comparison, because the one is a record from a patient whose ventricle is beating irregularly, and the other is a record from an animal in which there is a precisely similar disturbance of the heart's action produced experimentally. By taking records in that way from the human subject and by taking animals and creating in them similar disturbances by artificial interferences, one is very often able to elucidate the irregularity as met with in the human subject. This galvanometer of Einthoven's is an apparatus particularly suited to that purpose.

Here is another condition, a rather rare one, which you find in the human subject. Note the sharp deflections marked "R." The contractions of the ventricle in this subject were occurring at the normal rate of 75 per minute, and here are the corresponding pulse beats recorded. You see that each beat of the ventricle corresponds with a beat in the pulse. That is a patient who, on examination with the stethoscope or by any other means, revealed nothing but this normal beating of the ventricle: one could not, by this means, find any trace of disturbance. But the electrical curve shows it is anything but a normal heart. Instead of there being in front of each ventricular beat a single beat of the auricle, there are three, and here there is a fourth, so that the auricle is beating at four times the rate of the ventricle, not at 75, but at 300 per minute. The detection of an auricular rate of that type is of very great importance, for this reason, that at any moment the responses of the ventricle may double or quadruple their rate. In other words, it is an unstable condition: instead of the ventricle responding in this way, it will from time to time respond to each second auricular beat, or even to each, so that the rate of the ventricular beat may spring to 150 or even to 300 per minute; the last will create a considerable disturbance and an attack of unconsciousness will supervene. The galvanometer, in a case of this kind, gives us, practically speaking, the only means of determining with any degree of confidence the auricular rate, or the presence of this curious mechanism.

Lastly, I put on the screen a few records of heart sounds, obtained with this same machine. Connecting the string galvanometer with a microphonic system, you can obtain a record of heart sounds. In the first curve you see the first and second sounds, due in the main to closure of the heart valves. The third curve is one taken of an abnormal sound in the chest, a musical murmur due to rupture of one of the cardiac valves. And you will notice how rapid and how regular the oscillations of the string are when the musical murmur occurs. You will see a similar heart-sound curve in this bottom record: again the musical murmur is displaying itself by the regularity of
the oscillations; the curve is taken with a simultaneous electrocardiographic curve, so that you may place the murmur accurately relative to the systole and the diastole.

I have introduced those sound records chiefly for this purpose. The microphone which has been available, so far, in taking these heart sound records is not entirely satisfactory. It will produce the records which are shown here, but we are badly in need of a microphonic system which will do more for us: we want one with a higher natural frequency, one which will respond to oscillations of 300, 400 or 500 per second. If we could obtain such a microphonic system and work it in conjunction with a galvanometer we should be able to record many sounds, such as murmurs of the heart, which we cannot at present record, and that is because the natural frequency of our instruments is insufficient, because in consequence of the rate of oscillation of the sound waves they are damped out by our present apparatus. It occurred to me this might be a good opportunity of expressing these difficulties, because possibly some member of the two Societies present to-night may be able to help us.

Dr. Alexander Morison: I rise to express the gratification we all must feel at the demonstration which has been given to us by Dr. Thomas Lewis on a subject on which he is par excellence in this country, and, I think I may say, in more than this country, the acknowledged master. He knows me as, to a certain extent, a conservative in these matters, and if our expressed views do not always coincide, I hope we feel alike towards each other, for I have a high admiration of his work. I endorse what he said in his concluding sentences as to the desirability of an invention by electrical experts which will enable us to detect, better than we can at present, the sounds which so often elude our ears. We must not forget we have in our heads rather sensitive organs for registering sounds, and that organ, unless dulled by disuse or disease, is still capable of recording very important events and coming to important and valid conclusions with regard to the condition of the heart being dealt with without cardiographic aid. There is no question, however, that there are conditions of heart which escape us, and which electrical methods have undoubtedly revealed and may still further elucidate in the future. In the condition about which Dr. Lewis last spoke, in which the audibility of sounds would be an advantage—namely, a rhythmical contraction or flutter of the auricle, the sounds are too fine to be heard as a rule, and indeed are very rarely heard, although I have heard them, and believe I was the first to publish an account of such a case under the designation of "Jugular Embryocardia." With the help of experts, if some method of increased electrical auscultation could be introduced, as Dr. Lewis suggested, we should make an advance in the detection of the audible phenomena of some of these auricular conditions which at present usually escape us.

Dr. Sidney Russ: If the Cambridge Scientific Instrument Company cannot claim to have invented this instrument, they can at least claim to
have popularized it. On first acquaintance it appears rather complicated, but in this case I think it is only complicated so that it can be much simpler for people's hands, and it does perform in the most wonderful manner. There are one or two questions I would like to put. I do not know whether they are more appropriately addressed to Mr. Whipple or to Dr. Lewis. Looking at some of the tracings obtained by means of the electrocardiograph, one notices, very often, a series of vibrations. They appear to have different periodicities, and sometimes give much trouble. Are these periodicities due entirely to mechanical vibration, or is there a physiological basis for them? Secondly, in the apparently quiescent part of the curve you occasionally notice that the curve rises, and sometimes it will fall back to its previous level. It seems unlikely that it is an effect due to incorrect compensation. It is a very small thing, and the present sensitiveness of the instrument would, perhaps, not allow interpretations to be made from it. Looking at the facts which have been stated, it is a question whether it would be advisable to cut down the skin resistance. In the paper, various estimates of the skin resistance are given from hand to hand, and there is a rather wide margin allowed for it. I have no doubt that Mr. Whipple knows the experiments by Professor Gee on the resistance of the human body, and how it can be considerably lessened by preparation of the surface of the skin. Does he think any purpose would be served by elaborating on those lines?

Dr. G. E. S. Ward: It may interest some of the members to hear some views from a clinical and practical standpoint. I cannot praise too highly the apparatus which has been described by Mr. Whipple this evening. I think I was present at University College Hospital when this type of apparatus was first installed, and I took a great deal of trouble to see that at the hospital to which I am attached we secured the same kind of apparatus. It is extremely easy to work: in fact, the various parts are so carefully put together that one might almost say they are fool-proof. At the hospital where I work, every ward is wired to the apparatus, so that it is possible to take tracings of any of the patients in the hospital without moving them from their beds. Yet for several years we have never had any radical defect in the machine itself. When we have been hung up, it has been for some trifle connected with the wiring. With regard to the galvanometer and the rotatory time-marker and camera, I cannot praise them too highly. Ours have never caused any inconvenience. With regard to the other parts of the apparatus, there are one or two little points about which I would like to ask questions. First, with regard to the control board compensating wire and sliding adjustment, when one realizes the consummate care which has been taken in regard to other parts of the machine, one cannot help feeling that if there were some means adopted of covering in that portion of the control-board, small difficulties, such as may be caused by microscopic pieces of dirt, would not occur. With regard to the electrodes, one sees the type of electrodes used here, but there are certain types of patient who cannot sit in a chair: they may be very ill, and must have the

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tracings taken from the bed, and often it is difficult to get the electrode to their limbs. If something could be bound round the limb, it would be more comfortable, and a more perfect record might be obtained. For some years the hospital to which I am attached, has used a different type of electrode. It is a piece of matting of copper filings and string: it is flexible, and it is sewn into a flannel jacket long enough to encircle the limb. It is kept in position by tapes, and is attached by a screw adjustment to the wire of the machine. It is kept soaked in saline solution, and rubber sheeting is wrapped round it, so that it keeps moist. We find by experience we can get very good tracings with that, better than with those shown at this meeting. Finally, there is one point of which Mr. Whipple makes a note, namely, with regard to the light. It is most important to have as steady a light as possible, and I think the light provided on the apparatus is as steady as an arc lamp can be. This week I asked Dr. Sidney Russ if a "point-o'lite" would be possible to work with. We have not tried it, but we feel inclined to do so, because it is steady. Dr. Lewis has so ably demonstrated the clinical value of the electrocardiograph, that there is nothing for me to add, and I thank Mr. Whipple and Dr. Lewis for their addresses.

Dr. Alexander Morison: Is there a possibility of the time-marker being made absolutely automatic? With the instrument I use, one by the Cambridge Scientific Instrument Company, one has to revolve the rotor to catch the time of the tuning-fork. The light I use is the one used here, and I find it is not as satisfactory as it might be always, though one takes the trouble to get the electrodes into a proper position. If the automaticity of the rotor could be secured, and a better illumination, the instrument would be perfect.

Mr. P. M. Baker: The use of the "point-o'lite" lamp has been mentioned. The lamp has many advantages, and is free from some of the disadvantages of the arc, but in some work, comparable with that described in the paper, namely, on optical indicators for aéro engines, we found considerable difficulty in obtaining reasonably good photographs with this lamp. Possibly this may be due to the actinic value of the light, but that is a point on which I have had no opportunity of satisfying myself.

Professor Porter: I would like to add a point on the historical summary at the beginning of the paper. It does not seem to be generally known that an instrument for indicating currents by means of the movement of a metal strip between poles was devised by Cumming in 1827. And a similar instrument, that is to say, one depending on the same principle, was devised by Highton, known as the gold-leaf telegraph, in 1846: it was shown at one of the International Exhibitions, at South Kensington. Those two instruments preceded Einthoven's by many years. In stating this, I do not mean to detract from the great advances which were made by Einthoven in designing his instruments. There is considerable difference in detail between the earlier forms and the latter, but, in the matter of priority, it is worth remembering the pioneers in devices of this kind.
The CHAIRMAN: I propose a very hearty vote of thanks to Mr. Whipple for the two papers he has given us. The papers themselves have been intensely interesting to every one here, and the discussions have been no less interesting.

Mr. WHIPPLE: I should add that the apparatus has been lent by Dr. French, and it is entirely owing to his kindness that it is exhibited here today. And I forgot to mention that the lantern slide was taken by Professor Coker. Dr. Lewis is kind enough to say nice things about us, but I assure you the debt is the other way, absolutely: he has helped us all along the line.

Mr. WHIPPLE (reply communicated): I agree with Dr. Lewis that the microphone at present used for the measurement of heart sounds is unsatisfactory. There is undoubtedly an interesting and useful field of research open to the experimenter who obtains a satisfactory microphone for this purpose. The movements in the electrocardiogram to which Dr. Russ refers are not improbably due to muscular tremors. It has been found advisable to pack the electrodes into which the hands are placed with cotton wool soaked in the salt solution. The wool steadies and supports the hands and greatly reduces the muscular contractions. The string galvanometer is undoubtedly sensitive to mechanical vibration, but as a rule the vibrations met with in an ordinary pathological laboratory or hospital are not sufficient to affect the records seriously. No real advantage would be gained by cutting down the skin resistance—as a whole better results are given when the skin resistance is high, as the vibration in resistance with slight variation in the depth of immersion of the hands in the electrodes is then almost negligible. I am grateful to Dr. Russ for his reference to Professor Gee's work on the resistance of the human body. I agree with Dr. Ward as to the advisability of enclosing the moving parts of the control board to protect them from dirt. In all the modern instruments this course is adopted. It is impossible to use the electrodes exhibited at the bedside of a patient, and electrodes of various types with varying degrees of success have been used for this purpose. The electrode designed and shown by Dr. Ward should be of considerable service. It has not been found possible to make the rotary time-marker start automatically, although as a rule they start well. Dr. Russ will find that with a little practice it will be possible to start the rotor at the second, if not the first attempt. I have known a rotor run continuously without any attention for thirty hours. I am interested to learn from Professor Porter of the instruments designed by Cumming and Paton. The references are of interest owing to the controversy there has been as to the priority of invention of the string galvanometer. I do not know if the capillary electrometer has been used as suggested by Mr. Trotter for recording heart murmur. The use of the instrument had been revived considerably by the late Dr. Keith Lucas.
Section of Electro-Therapeutics.

President—Mr. Cecil R. C. Lyster.

Stereoscopic Radiography in the Treatment of Fractures of the Femur.

By James Metcalfe, M.D.

I desire to bring before you the great importance of frequent stereoscopic radiographic examinations in fractures of the femur. In fact, I consider these examinations so influence the procedure adopted that they almost become an integral part of the treatment. The methods I am about to describe were only adopted at a late stage of the war. Any one who saw the results of the treatment of these fractures in the earlier stages of the war and compares them with the results of more recent date will bear witness to the enormous improvement during the latter period. The limbs have in most cases shown very little shortening; the knee has not been stiff and walking has been quite easy. As the prints show, the bones were usually very comminuted and often septic. The economic result too, has been a great saving of expense to the country.

I do not wish to enter in detail on the actual surgical methods used in treating these fractures. The whole scheme has been largely a process of evolution, and the honour of initiating and carrying it out belongs to Major Maurice G. Pearson, of the South African Medical Corps, whom I had hoped might have been present tonight. Major Pearson developed his methods in France, and the results were of such an eminently satisfactory character that the War Office brought him and some members of his staff to England and gave them a considerable number of beds specifically for fractures of

1 At a meeting of the Section, held April 25, 1919.
the femur at the Edmonton Special Military Surgical Hospital. I should rather say that bed space was given to Major Pearson and his staff as they brought their own beds from France. When the scheme was fully developed there were 300 of them. The beds have an ordinary tubular frame, but instead of spring mattresses, have tight canvas slings, 16 in. wide, fastened by straps and buckles on one side and by metal hooks or a quick release contrivance to the other. The mattresses lie on the slings in several sections, and one section lies below the fracture or wound. This can be easily removed for dressing the wound or for radiographic examination.

The fracture is treated by a Thomas's splint and extension, and with the use of callipers when necessary, inserted into the femur just above the knee. The leg is bare below the knee, and massage, electricity, &c., can be effectively used without moving the limb.

One great point in the method is that there is no after stiffness of the knee-joint. The leg is measured periodically, and the amount of extension required carefully regulated.

The radiographic examinations are made with a portable apparatus worked from a set of 48-volt accumulators. The patient is undisturbed, and the examinations are all made from below the bed. Lateral examinations are impracticable from the position of the parts, the splints, and the proximity of the bedding. Stereoscopic pictures are made about every ten days or a fortnight. Wellington and Ward 6 by 12 plates are used. The tube used has been a Macalaster-Wiggin 7 in. tungsten target tube. This is placed in a covered holder, which is approximated to the position and centred under the wound from which the portion of mattress has been removed. I had a special cassette made for 6 by 12 plates, but as a matter of convenience it is found better to simply place the plate over the centre of the part to be radiographed and mark its corners on the skin or clothing with a blue pencil. The first exposure is made with the tube displaced 3 cm. to the patient's right: another plate is substituted and the tube displaced for a similar distance to the left. The tube has a spark gap equivalent of 5 in. to 6 in.; 2 ma. to 3 ma. of current are passed through it; the exposure is about thirty-five seconds, and the distance of the anticathode from the plate is about 40 cm. The plates have to be placed at various angles, and the tube holder has of course to be centred for the necessary position. Major Phillips, of the Government X-ray laboratory, designed a special holder with a centring device. We found, how-
ever, the centring mechanism inconvenient, and our own adjustment accurate, more readily adapted, and for all practical purposes just as efficient.

After the plates are developed and dried they are reduced in a dark room by a photographic reducer directly on to half-inch plate size Ilford glossy bromide papers. An arc lamp is used to illuminate the negative, as it allows the print to be made much more quickly than when electric lamps are used. The time of exposure is a little over a minute. The prints after development are examined at leisure with a hand stereoscope by the surgeon in charge and then attached to the case boards.

The necessary rectifications are made in the limb to improve the position steadily. Increased or diminished extension, weights and pressure pads or other devices are used. Massage or electricity is also utilized when required.

It has been found that the frequent stereoscopic examination in this way has led to an enormous improvement in the results obtained. The fragments are kept in much better apposition: shortening is infrequent, and the resulting limb is of an infinitely more useful character. I think that the examination of reduced stereoscopic prints in a hand stereoscope shows the appearance and contour of the bone much better than is seen on the large plates in a Wheatstone or similar stereoscopic device. And of course the small prints are very convenient and readily kept in series.

(Some hundreds of reduced stereoscopic prints were shown for examination by hand stereoscopes, and many were demonstrated by the epidiascope.)

Some of the prints go through a series of nine to twelve examinations, and show the gradual evolution and progress of the fracture from the earliest date at which the case was received at hospital up to that when firm union took place, resulting in a useful limb.

**DISCUSSION.**

The President: If we enter upon a discussion of fractured femurs from the surgical point of view we shall not finish to-night, so we had better limit our remarks to the radiographical point of view, pure and simple.

Dr. Florence Stoney: Are the results which we have been shown those of picked specimens, or do they represent the average which Dr. Metcalfe gets in hospital? I ask this because they seem to me to be remarkably good
results. I would point out the importance of taking a lateral view in these fracture cases. Many cases look very nice from the front view, but on taking a lateral view you find one or other fragment (generally the lower) behind the other, and very much out of place, which does not show so accurately on the stereoscopic view, though it could be ascertained by taking localizing measurements.

Dr. Hernaman-Johnson: From the purely radiographic point of view, I appreciate the remarks made by Dr. Stoney in regard to the importance of the lateral view. And unless there be something peculiar about the treatment which is adopted at Edmonton as regards splinting, there should not be any difficulty about getting a lateral view in a bad case. In 1914, when I first went to Aldershot, the portable apparatus there took seven minutes at least to skiagraph a thigh. Nevertheless, we got fractured femurs in early days, and always took a lateral view with the patient in bed as well as an antero-posterior one. As Miss Stoney says, the bones often appeared to be in good position from the front, but when seen laterally much overlapping and distortion became evident. I agree with her that the stereoscopic view cannot wholly take the place of the lateral in these cases, and the lateral should be always taken when possible, so that the results can be seen from both aspects.

The President: Most of us know the difficulties there are in bedside radiography, and I think Dr. Metcalfe has overcome those difficulties extremely well.

Dr. Metcalfe (in reply): I quite agree with the remarks of Dr. Stoney and Dr. Hernaman-Johnson. I think that very often the lateral view does show some deformity which the stereoscopic picture does not show, but you may take it from me that the condition of most of these men was so serious that it was impossible to get the lateral view with the splints on; there were so many appliances used. But when, later in the case, we could get the lateral view, we did so. When men are in the precarious stage they should be disturbed as little as possible. We were able to get stereoscopic views, and, judging by results—and, after all, the results are the test—they were better than by any other method. The great point is that frequent examinations should be made. I think this method will come to be the common one in civil practice and in civil hospitals, where, probably, conditions like fractured femurs will be all aggregated in one large ward, and the apparatus will be there available for frequent examinations.
DISCUSSION ON THE RADIOGRAPHY OF GALL-STONES.¹

Dr. Robert Knox.

The value of a correct diagnosis of the presence of gall-stones in the gall-bladder or bile ducts is very great. The differential diagnosis between symptoms caused by gall-stones and conditions which give rise to similar symptoms is very difficult. In the consideration of lesions on the right side of the abdomen we have to remember that there are so many structures situated in the same region which may give rise to perplexing symptoms, that any method of examination which is likely to aid in the differential diagnosis is worth any trouble its execution may entail. The X-ray examination of the liver and structures in its vicinity is extremely useful, and, if the percentage of accurate diagnoses can be increased, the value of the method will, of course, rise proportionately to this increased percentage of accuracy. It is, therefore, essential that all steps should be taken to ensure the proper carrying out of the technique. It is also imperative that the radiologist should be conversant with the anatomy of the region, and that he should have a sound working knowledge of clinical medicine. With these should be coupled a familiarity with the radiogram and its interpretation. A review of the literature of the radiography of the liver, gall-bladder and bile ducts, is instructive: it reveals a gradual conversion of radiologists from an attitude of almost sceptical indifference to one of overweening confidence in the belief that gall-stones may be diagnosed in a very large percentage of the cases examined. This percentage rises as high as between 80 and 90 with a number of workers, and falls as low as 5 to 10 with others. A diversity so great requires careful consideration before a decision can be arrived at regarding the value of radiography in the diagnosis of the presence of gall-stones. It is obvious that there must be an explanation of this great difference of opinion. It may be largely explained in a difference in the technique employed by various workers, and the importance they attach to the exhibition of doubtful shadows. The

¹ At a meeting of the Section, held April 25, 1919.
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Technique is readily standardized, so this should offer no serious obstacle to an understanding of the value in the future. Critical workers refuse to admit a doubtful shadow as having any value in diagnosis. This is too strong a position to assume, since the value of a doubtful shadow is very difficult to estimate. It should not be taken as an indication for operation when clinical signs and symptoms do not support the suggested diagnosis. A negative radiographic report is of no value, because it is not possible to demonstrate all cases of gallstones, and we have the authoritative statement of C. H. Mayo, who says that "to depend upon radiographic evidence as now developed alone would be to step back twelve years in the advance of gall-bladder and bile-duct surgery." A careful study of the doubtful shadow is of value, if for no other reason than that it encourages research and stimulates the observer to obtain better results, in order that a more positive opinion may be expressed. The observation and recording of all doubtful shadows must be of value when operative measures are afterwards employed, because then the findings of radiography may be compared with those conditions which are found at the operation. In this way valuable aids to diagnosis may be acquired. Until quite recently, in company with the majority of workers, I was sceptical with regard to the value of radiography in the diagnosis of gall-stones. During many years of observation I had met with only a few cases which could be positively diagnosed as gall-stones. The recorded observations of workers in various parts of the world—notably Thurstan Holland, in England; Ledoux-Lebard, in France; Carman and Miller, Lennard and George, and Case, in America; and Macleod, of Shanghai—induced me to make a careful study of the literature of the subject, the technique employed, and a critical examination of the published radiograms. This led to experimental work with calculi, comparisons of density of tissues, absorption of radiations, and particularly to an inquiry into the photographic processes employed.

The investigations were carried out under the following heads: (1) Anatomical considerations; (2) pathology of gall-stones—classification; (3) experimental investigation on absorption coefficients of gall-stones and surrounding tissues; (4) radiographic appearance of gall-stones; (5) technique of the examination; (6) situations in which gall-stones may be found; (7) differential diagnosis; (8) the pathological gall-bladder; (9) record of cases.

I do not propose to burden you with the results of all these investigations, but I wish to show you a number of explanatory slides.
The first slide, and the three which follow, are taken from
"Cunningham's Anatomy." They demonstrate the position of the
gall-bladder and its anatomical relationships. The portion of the
gall-bladder which is in relation to the anterior abdominal wall is
situated at the level of the ninth costal cartilage. Note how little
of the gall-bladder is visible from the front.

Here is a photograph of the liver viewed from the front, showing
the portion of gall-bladder which comes below the inferior border of the
liver in the normal subject. The next slide shows the liver viewed
from behind, and here we have a fairly good picture of the gall-bladder,
its position, and its relation to the common bile duct. You will note
the depth to which the gall-bladder goes beneath the liver.

The next picture shows the structures between the layers of the
lesser omentum: it shows the gall-bladder, hepatic ducts, the common
duct, and the cystic duct, down to the duodenum. In it are seen the
relations of the gall-bladder and ducts to the surrounding structures.
It is useful to remember the position of the common duct in relation to
the gall-bladder.

The next picture is from "Symington's Sectional Atlas of Anatomy."
It is extremely important when you have to consider the position of a
shadow on the photographic plate, because if you realize that the
gall-bladder and the common duct lie in different planes, and that a
shadow of a stone in the common bile duct might come in front of the
body of the vertebra and be lost in the picture.

To facilitate the examination of the patient a simple table-top was
constructed with a hinged part in the middle which permits the ends
to be raised so that the patient may be placed on the slope. I show
you the position for the gall-bladder from the front, and the plate is
seen beneath. In the kidney position there is complete relaxation of
the parts, and a close approximation of the patient's back to the plate.
This table-top is simply constructed: it is made of three-ply wood and
has a rack arrangement at each end.

The next stage of the investigations led to a large number of experi-
mental exposures of stones of various kinds, with the intention of
finding their approximate density. I show you pictures of some small
gall-stones and some larger ones. You also see a stone of greater
density with the peripheral margin well shown.

Next, you see a collection of gall-stones put into a finger-stall and
radiographed: also one of the stones which was split open to show the
structure. These were radiographed many times under varying condi-
tions of current, conditions of tube and duration of exposure. The prints exhibited on the wall give the results of two experiments. There is also a photographic enlargement of a stone cut in two to show the internal structure.

Here is another taken with a penetration scale of the same stones to find out the best quality of radiation with which to radiograph those calculi. I show you the scale with the number of steps of aluminium of known thickness, 32, 16, 8, 4, 2, and 1 mm. The penetration scale is extremely useful in experimental work. The next slide is one of the first pictures I took of a gall-stone, now many years ago. It is piece of liver, with the gall-bladder projecting below, and in the end of the gall-bladder is a well-defined shadow of a stone. What struck me at that time was the fact that liver substance cast a denser shadow than did the gall-stone, so that if a gall-stone were behind the liver there would be no shadow cast on the plate. That left me for a long time in a very uncomfortable frame of mind, because I had the idea that it was impossible to demonstrate a gall-stone if it happened to be in or behind the liver. That appears to be wrong; perhaps we shall hear some views on that in the discussion.

The next slide shows a kidney split open, with calculi and various chemical deposits of varying density in its interior. I show this because a shadow of similar density in the region of the kidney might easily give a similar shadow to that of a gall-stone viewed from the front. Renal calculi may simulate the appearances of gall-stones when the internal structure of the stone is shown in the radiogram. In these cases very fine detail may be shown. Here, for example, is a calculus in the urinary bladder, in which the detail is extremely well shown, from the nucleus to the periphery.

The next is somewhat apart from gall-bladder work, but it shows the density which you can obtain, even in a radiogram in the human subject. Here is the same calculus after removal from the urinary bladder showing a definite nucleus: you can see the various layers and a central nucleus.

I now show a calculus of another kind, a salivary calculus, situated behind the jaw. It was taken with the plate in the mouth, and the rays were projected from under the chin. The detail of the calculus is well seen.

The next stage of the investigation took us to the absorption of radiations by the tissues. The slide illustrates an experiment which was carried out on beef. Each of these layers is 1 in. thick, and various
radiations were projected through those on to photographic papers at the different levels. These were carefully collected, and the percentage of radiations passing through and reaching each level was estimated at the end of the exposures. Filtered and unfiltered rays were used. There is a rapid drop in the penetration after the first inch, in the second inch it is less rapid, while at the fourth inch very little gets through. The same was seen with the filtered ray. Curiously enough, both the filtered and the unfiltered rays drop to practically the same level at the end of the second inch. I show that to indicate that in radiography of the gall-bladder or of the kidney there is a very great absorption by the tissues in the first two or three inches, so that if a so-called "soft tube" is used for these exposures you can realize that the bulk of the radiations are absorbed by the tissues before they get near the plate on which it is desired to obtain an impression.

Here is another experiment on penetration. A dried femur was radiographed along with a penetrometer scale, with spark lengths of 2, 4, 6, 8, 10 in. The tube was a Coolidge, and the exposure was the so-called instantaneous, or single flash. Note the gain in penetration with the increase of the spark length and the detail becoming progressively greater with the increase in the spark gap.

Now we come to a consideration of the demonstration of gall-stones in the patient. The particular patient from whom the plates were obtained was sent with the definite history of a tumour felt under the liver. I examined the case very thoroughly, and was certain I could feel, under the liver, a definite resistance, and I went farther and concluded I could almost feel the outline of the gall-bladder and gall-stones. Accordingly, I exposed a plate in the kidney position for four seconds. During the middle of the exposure the patient must have moved, because a blurred shadow was obtained, and one could not arrive at a diagnosis from such a plate. I felt so convinced that I had to deal with a gall-stone case that I persevered. The patient was placed in the prone position, and the shoulders elevated, with the plate beneath; a somewhat shorter exposure was made. A much more definite picture was obtained, but nothing so clearly defined as the final one taken with an intensifying screen and an exposure of half a second. In this you can see the outline of the thickened bladder wall. The patient was admitted to hospital three weeks afterwards, and again examined. The swelling had entirely changed in character; it was smaller, but could just be felt. The explanation of the difference in the two pictures is interesting: In the first picture, the gall-bladder was, presumably, dis-
tended with some fluid, probably bile. The patient at the first examination complained of a very distinct sense of distension in the region, also pain, but at the second examination it appeared that the pain and feeling of distension had gone, and she felt much more comfortable. In the interval the gall-bladder had probably emptied some of its liquid contents, had contracted on the stones, giving the sausage-shaped appearance. The case was operated upon, and the next slide shows the radiogram of the gall-bladder after removal.

The next slide is from a case of Dr. Thurstan Holland's, showing gall-stones in the gall-bladder in a case which had had an opaque meal. The shadows are characteristic of gall-stones.

The next slide is also from Dr. Thurstan Holland. He demonstrated a group of shadows which are not very sharp, and he explained that the reason of the lack of sharpness was that the plate was taken in the kidney position, and that the stones being a considerable distance from the plates are not so sharply defined.

The next is probably the first definite case of gall-stones which I came across. There was a definite shadow under the liver, and you see a number of opaque shadows with the transparent centres. This case was not confirmed by operation. A diagnosis of gall-stones was made in this case.

A few days after seeing the case of multiple gall-stones, I had a case at hospital which, on routine examination, showed a definite and very dense shadow in the renal region, and the question arose as to whether it was a renal calculus, a gall-stone, or a foreign body. This case received a good deal of investigation, and we succeeded in getting a fairly good lateral plate, which shows a definite shadow of the stone lying a little in front of the anterior border of the vertebrae. That, to my mind, is the diagnostic point between renal and gall-bladder calculi. If you can succeed in demonstrating the lateral picture of one or the other, you can fairly well say whether a stone is in the kidney or in the gall-bladder. It is not easy to get a satisfactory plate, but it is worth while making a good trial. This case was operated upon, and the stone was found to be in the common bile duct, and it was removed.

A few days afterwards, I had another case, in which I got a very definite shadow between the eleventh and twelfth ribs, though not so dense as the one I have just shown you. In both there was that curious appearance in the middle of the shadow which suggested the appearance of a button: in fact in both the cases I carefully inquired
as to the possibility of a button being there. The lateral view clears up that point, because a button would not show in both the antero- position and the lateral position. The following slides are shown to illustrate points in the differential diagnosis.

Here is an ordinary renal calculus, showing the characteristic appearances of these shadows. This should be compared with the round stone in the common bile duct.

The next shows four shadows in a kidney. Here you have the shadow of the kidney to guide you. You may have a shadow in the kidney and another in the gall-bladder in front of it, and that is where a lateral view would settle the question at once. There are other methods of doing that. Stereoscopic plates might be taken, or you could shift the tube, so as to throw one shadow clear of the other. Here is a print of Dr. Thurstan Holland's which shows the relationship between the kidney and gall-stones. The kidney shadow is beautifully shown. Just below the kidney shadow are two gall-stones, and clearly separated from the kidney shadow. This picture is one of the best diagnostic radiograms it is possible to obtain.

The differential diagnosis of gall-stones is complicated by shadows, which might come in the region of the gall-bladder or kidney, and give rise to much doubt as to their nature.

The next slide is from a case of calcified mesenteric glands: you see a calcaeous nodule lying between the two ribs, which would be very difficult to distinguish when met with on the right side: this happened to be on the left side. The patient had calcified glands removed from the peritoneal cavity on two occasions before being examined for gall-stones.

A faecal accumulation might give rise to a difficulty in diagnosis. The radiogram shows a large faecal mass in the kidney region: its nature was settled by free purgation and another radiographic examination.

The question of differential diagnosis is often complicated when a shadow is seen of the character I now show you situated on the under surface of the liver. The patient was sent for examination to eliminate gall-stones: there was a large shadow in the gall-bladder region, but no evidence of a calculus could be found. It turned out to be an empyema, and pus was coming down from behind, giving the appearance seen in the radiogram.

Malignant disease of the gall-bladder will give a shadow if the growth is at all extensive.
There is a great deal more which might be said on the diagnosis of gall-stones, particularly in regard to technique, but there is not time to enter fully into that. I may say, however, that much depends on the type of the plate. The usual mistake is to get negatives which are much too dense, so that it is difficult to differentiate shadows.

Dr. R. W. A. Salmond.

This Section is deeply indebted to Dr. Knox for having opened this discussion on the diagnosis of gall-stones. It must be only too evident, judging from our published results and our contributions to the literature, that we, in the past, have been somewhat behind our friends in the States. It is hoped that to-night’s discussion will tend to level up this difference.

First, as to technique. The patient has to be properly prepared as for a kidney examination, and if you are going to depend on one position, the plate should be placed on the front, because both gall-bladder and duct are nearer to the front of the patient than to the back. So, taken from above, the patient would be lying prone, and the upper part of the chest, as Dr. Knox mentioned, should be raised. There are very sound reasons for this attitude being adopted. First, when the patient is prone, the liver gravitates towards the front; and, secondly, with the upper part of the chest raised, the costal cartilages are kept clear of the anterior margin of the liver and gall-bladder. In fact, it is the exact reverse of what takes place when the patient is supine, for then the liver gravitates towards the back and also recedes up behind the costal cartilages. Also, a lateral view is of great help, especially in differential diagnosis. The possibility or ease or difficulty of showing up gall-stones depends very largely on the thickness of the patient and on the composition of the stones. To put it in a nutshell, the chemical composition of gall-stones may be said to vary from nearly pure cholesterol—it is impossible to get it quite pure—to nearly pure lime salts, with every intermediate stage. Unfortunately for radiologists, the tendency is towards the cholesterol side of the scale. Naturally, it is much easier to show up a stone containing a fair amount of lime salt than one of nearly pure cholesterol which is composed of elements of low atomic weight.

The essentials for the radiography of gall-stones are, it is considered:
(1) Correct exposure; it does not matter very much whether one's tube is hard or soft, provided, of course, that it is sufficiently penetrative to pass through the thick organs of this region. (2) The exposure must be quick enough to exclude the factor of respiratory movement. There are accessory or indirect aids to diagnosis. We may be able to show up a thickened or distended gall-bladder. It has been said that if there is cholecystitis there is always also choledolithiasis. This may be true, or it may not, but it is obviously open to misconception, because a gall-stone may pass through the patient, be completely away from him, and yet may leave a thickened gall-bladder. Again, we may see a defect after an opaque meal examination, at the pylorus or duodenum. In the differential diagnosis in difficult cases it seems much more rational to depend rather on the clinical symptoms and the laboratory tests than upon the question of the uniformity, density, and shape of the shadows. A lateral view in these cases is of great help.

I would like to show you, in conclusion, four experimental plates, which may be of some interest. The first shows radiograms of four typical kinds of stones. The upper left one is practically pure cholesterol; the central nucleus is a little denser than the rest, because it contains more lime salts. The upper right one is practically pure cholesterol stained with biliverdin, and in the centre is a cavity which is so common in gall-stones. The lower left one is one of those rare forms composed of nearly pure lime salts, and in its centre there is a tendency to a cavity nucleus. The lower right one is one of the combinations of cholesterol with a lime salt nucleus. The next slide shows the relative densities of liver tissue and a cholesterol gall-stone. I took a piece of fresh human liver and cut it into a portion as near the size of a selected gall-stone as I could, and put it at the side of the selected cholesterol gall-stone. On the left you see the sketch of the piece of human liver, and it is as dense as, or even denser than, the stone. The next slide shows three relatively pure cholesterol stones. They were laid on the plate, and above them was a slice of fresh human liver. It varies from \( \frac{3}{4} \) in. on the left to \( 1\frac{1}{2} \) in. about the centre, and you see that in the gall-stone nearer the right side the shadow is less distinct, this being due to the increased thickness of the liver in this position. The last slide shows a relatively free cholesterol stone through 16 mm. of aluminium, which corresponds, approximately, to 16 cm. of human tissue, that is, over 6 in.
Captain Backman (U.S.A. Army).

In 1913 I attended a meeting of the American Society for this specialty in Cleveland, and I was present when Dr. Leonard, of Boston, brought forward his paper on the radiological examination of the liver and the gall-bladder and gall-stones. At that time he demonstrated the gall-bladder very rapidly, and he was laughed at by some and cheered by others. His demonstration evoked quite a discussion. Since that date the technique has been wonderfully well thought out. Dr. Leonard demonstrated the best position for taking gall-stones, in which the patient is quite prone; the head, shoulders, and extremities being extended towards the left in order to get the greater angle of the gall-bladder. Dr. Knox's position is, I think, a great improvement upon that, and I think he has shown, by his results, that he can get much clearer and better pictures of the gall-stones.

With regard to Dr. Salmond's remarks as to hard rays and soft rays, my experience is that the hard rays do not show gall-stones; I prefer a softer ray. And that, too, is the experience of some of my colleagues.

Dr. Richardson.

I agree with Captain Backman in preferring the soft ray, a short exposure, and using the intensifying screen. I believe Dr. Knox is experimenting with double intensifying screens and films, and I think he might be able to tell us something about technique which would be valuable.

Dr. Hernaman-Johnson.

Have any members had any experience of inflating the bowel with air for the purpose of defining the gall-bladder, as was described last year in the Archives of Radiology?

Dr. Robert Knox (in reply).

I think that the tube of medium hardness will produce the best results. I carried out a number of experiments with the Coolidge tube: I radiographed gall-stones with a ½-in. spark, and went up to one as AU—10α
large as 9\(\frac{1}{2}\) in. I gave almost all lengths of exposure, and got as good results with the 9\(\frac{1}{2}\)-in. spark as with the \(\frac{1}{2}\)-in. with shorter exposures. In none of the experiments did I succeed in going right through the stone—in other words, every stone showed on the plate. It is not a question of the radiation which is being used for your exposure, but what type of radiation gets through the patient. Are not all the softer rays absorbed before they get there? And is not the ray which is doing the work the harder ray in the bundle? If you can get your result by hardening the tube and shortening the exposure, your risk is not increased and you are gaining something.

Captain Backman flatters me when he speaks of “my position”: it is not mine, its application originated with Carl Beck, of New York, who first described the position obtained by raising the chest and throwing the gall-bladder down. All that I have added which can be called new is the hinging of the table in the middle so as to raise the two ends.

I have never intentionally inflated a bowel for the examination of cases of suspected gall-stones, but I have repeatedly seen cases in which the bowel has been distended with gas, and certainly the detail then procured is very much better: there is a sharper distinction of shadows if the gall-bladder is anywhere near it.
Section of Epidemiology and State Medicine.

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The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

An Outbreak of Typhoid Fever in Inoculated Soldiers.
Clinical Study.¹

By CLINTON B. HAWN, Captain M.C., U.S.A.

(Chief of Medical Service, Base Hospital 33.)

BEING on temporary duty at U.S. Military Hospital, Hursley, England, I was asked on July 18, 1918, by the Commanding Officer, Major John B. Fattic, to supervise the care of this group of typhoid and suspected typhoid cases. This work has been carried on with the hearty co-operation of Captain Lee F. Hunt, Chief of Medical Service, and Lieutenant Clarence L. Bock, Ward Surgeon. Prompt segregation of these cases was effected by screening their ward. Disinfection of excreta was accomplished by chemical means: all dishes, bed pans, and urinals were scalded immediately after use, and up to the present time no case of typhoid has developed here secondary to this group. The diet has been largely liquid. A plentiful supply of excellent milk and eggs was available, and was supplemented by broths, cocoa, and soft foods as much as possible, especially in the milder cases. Facilities for the preparation of articles of soft diet were limited, and consequently additions of this nature to the diet were not as extensive as might have been desired. However, the size of the intestinal ulcerations found at autopsy in the fatal cases was such as to cause extreme hesitation in the matter of more liberal feeding for the rest of the cases.

¹ At a meeting of the Section, held November 8, 1918.
A short discussion and explanation of the appended chart may aid in promoting a clear understanding of the cases. Of the forty cases in this group, only two (Nos. 7 and 38) were clinically not typhoid.

(1) Under the heading "onset," the date and initial symptoms are shown. The establishment of the exact date was very difficult, and in many cases was determined by the clinical record during course of illness in hospital, verified as far as possible by a more or less indefinite statement of the patient. The striking feature shown under "initial symptoms," which are mentioned in the order of their occurrence, is the frequency of chill as the first abnormality noted by the patient. This occurred in approximately 16 per cent. of the cases, and, according to the patients' statement, was a definite rigor. Diarrhoea early in the onset occurred in approximately 58 per cent. of the cases, constipation in approximately 21 per cent., abdominal pain in approximately 6 per cent., nose bleeding in but one case, approximately 2·6 per cent.

(2) The figures in the column, "duration on admission," are based on the dates of onset, and are therefore not exact, but closely approximate the truth. The shortest duration of disease on admission was three days, the longest was thirty-one days. From this column the average duration of disease on admission is determined as 13·5 days.

(3) Diarrhoea was troublesome in but seven cases, constipation in six cases, abdominal distension in nine cases, marked persistent abdominal distress in three cases.

(4) Rose spots were observed in nineteen cases. Probable enlargement of the spleen as determined by percussion was noted in eighteen cases, and is designated by a single plus under the column marked "spleen." Palpable spleen was made out in fifteen cases, and is shown in this column by a double plus. Tender toes, designated by a plus sign, were demonstrated in fifteen cases. In five cases this condition was so marked as to cause considerable discomfort.

(5) The leucocyte counts are quite typical of typhoid. A count of 7,000 or less occurred in eighteen cases. The second count in Case 29 shows the leucocytosis of a complicating lobar pneumonia. The second and third counts in Case 33 show the leucocytosis of perforation and commencing peritonitis. The second count in Case 36 was considered as a strong indication of perforation, occurring in coincidence with severe abdominal pain. However, there was not enough evidence to

1 Chart omitted.
warrant operation, and we were later forced to conclude that the perforation, if any had occurred, had been sealed immediately by the omentum.

(6) "Atropine test." The results obtained by this test are of extreme interest and worthy of a more extensive discussion than can be given in this report. In one column appears the day of disease on which the test was performed, in the next column is noted the pulse reaction to the test. If an acceleration in pulse-rate occurred it is shown by a plus sign and a number representing the amount of increase due to the atropine over the normal rate immediately before the administration of atropine. Unfortunately, it was impossible to repeat tests in any of the cases because of lack of time. Two of the forty cases in this group, Nos. 34 and 37, died before the series of tests was started. Two of the remaining thirty-eight cases, Nos. 7 and 38, clinically not typhoid, showed accelerations of twenty-four and thirty-four beats respectively, and should be considered negative reactions (acceleration of over fifteen beats), leaving tests on thirty-six cases of clinical typhoid for consideration.

An acceleration of pulse-rate occurred in thirty-three cases, a decrease of rate occurred in one case, No. 33. No alteration in rate occurred in two cases, Nos. 10 and 28. Twenty-three of the thirty-six cases showed a positive reaction (either a decrease, no alteration, or an increase of less than fifteen beats). The earliest day on which the test was performed in this group of twenty-three was the seventeenth day, the latest the thirty-first day. The results in this group of thirty-six show a positive reaction in 63·8 per cent. of the cases. Of the eleven cases showing an acceleration of fifteen or more beats, the test was performed later than the thirtieth day of the disease on seven cases, and on none was the test done earlier than the twenty-first day. It is only fair to the originators of this test to state that accuracy is not claimed for tests done later than the thirtieth day, and to point out that if we eliminate the seven cases just mentioned the percentage of positive results on the whole series of thirty-six cases would be 79·3 per cent.

These tests were carried out according to the technique described by Captain H. F. Marris, R.A.M.C., in the British Medical Journal, 1916, ii, p. 717, and Special Report Series, No. 9, Medical Research Committee, to which article, and articles on the same subject by A. Friedlander and C. P. M'Cord, in the Journal of the American Medical Association, May 18, 1918, p. 1435, and E. H. Mason in JA—8a
Hawn: *Typhoid Fever in Inoculated Soldiers*

*Archives of Internal Medicine*, January, 1918, p. 1, the reader is referred for further information.

(7) Typhoid bacilli were isolated from the blood in thirteen cases, from the stools in ten cases. In one case, No. 34, the blood culture was positive as late as the twentieth day of the disease. In the column marked "Widals" are placed the results as determined by a series of tests done on each case. These results will be discussed at greater length in the accompanying report on laboratory work by Captain J. G. Hopkins, M.C.

(8) The clinical course in these cases is briefly shown in the latter part of the chart. Average temperature and pulse-rates are shown in their proper columns. The average duration of fever in thirty-two cases, now convalescent, is over thirty-five days. The shortest case, No. 32, is nineteen days, the longest case, No. 40, is fifty-eight days. Of the fatal cases, Case 11 died on the thirty-third day (terminal pneumonia), Case 30 died on the thirty-ninth day (toxæmia), Case 33 died on the thirty-eighth day (surgical shock, following operation for perforation), Case 34 died on the twenty-seventh day (toxæmia), Case 37 died on the ninth day (toxæmia). Out of the thirty-eight cases, twenty-seven were high grade typhoids, much more severe than should be expected in vaccinated men. This is undoubtedly due to the average lateness in the course on admission to the hospital. The remaining eleven cases were mild, low grade typhoids. Complications occurring in this series were: Lobar pneumonia, one, Case 29; terminal pneumonia, one, Case 30; broncho-pneumonia, two, Cases 11 and 30; severe bronchitis, six, Cases 1, 3, 8, 12, 27 and 34; hemorrhage, three, Cases 10, 12 and 13; perforation, one, Case 33; phlebitis, one, Case 36; bed sore, two, Cases 5 and 10. One case, No. 15, is still febrile in the fifty-first day of the disease.

**Conclusions.**

(1) Owing to the lateness in the course on admission to hospital, cultural confirmation of clinical diagnosis was impossible in a considerable number of cases. The fact that all of these men had received routine immunizing vaccine during the past eleven months made the ordinary Widal reaction unreliable. Careful clinical study, however, justifies a positive diagnosis in thirty-eight of the cases.

(2) That severe typhoid may occur in vaccinated men is proved by this series of cases.
(3) The occurrence of uniformly low, white blood counts in this series again illustrates the diagnostic value of routine blood examination in all febrile conditions.

(4) The atropine test may be of great value in the diagnosis of typhoid fever, although the smallness of this series, impossibility of laboratory confirmation of diagnosis, and lack of control cases, make authoritative conclusions in this matter impossible.

(5) Explanation of the lamentably high mortality rate (13.15 per cent.) may be found in the average lateness in the course of disease on admission to the hospital.

Epidemiology of the Outbreak of Typhoid Fever among the Fourth Casual Company, J.A.R.D., Camp —.

By Fred. M. Meader, Major M.C., U.S.A.

(Sanitary Officer for Winchester Area.)

On July 11, 1918, there appeared at an American rest camp the Fourth Casual Company J.A.R.D., Camp —. Soon after entrance to the camp several cases of fever were brought to the camp hospital. At the time many cases of influenza were present among the soldiers who were passing through this camp, so that no special significance was attributed to the symptoms. After a few days' observation the Commanding Officer, Captain Logan, suspected that the cases might be typhoid fever. At about the same time a few patients had been sent to a base hospital. Suspicion of typhoid fever was soon aroused, and blood cultures were ordered by Captain Hawn. The District Sanitary Officer inspected the cases and took specimens of blood from each of the new cases for a culture. *Bacillus typhosus* was isolated from eight out of ten cases two days later. During the next month *Bacillus typhosus* was isolated from the blood or feces of ten more cases. Altogether thirty-eight soldiers developed typhoid fever at this camp. *Bacillus typhosus* was isolated from eighteen of these cases.

This organization left a cantonment on June 15, 1918, for Camp Merritt, New Jersey. While en route, on June 18, the organization
stopped at Meridan, Mississippi, and was taken for a swim in a lake. The organization arrived at Camp Merritt on June 21, and embarked on June 28, and arrived at Liverpool, July 10. After disembarkation the organization proceeded to ——. At Liverpool three cases were left at the Red Cross Hospital, and it is reported that two other cases were left at some station en route. At the American rest camp forty soldiers were found ill and transferred to a base hospital, where a diagnosis of typhoid fever was made on thirty-eight of them.

From a study of the history of each case it was noted that the date of onset varied over a period of about a month. The first case was ill on leaving the original cantonment. The second developed on June 19, the third on June 21, the fourth on June 24, three on June 26, four on June 29, five on July 6; after which a case developed every day up to the time the organization arrived at the American rest camp.

The organization left the American rest camp before the diagnosis of typhoid fever had been established, and it has since been learned that fifty-seven other cases developed; this makes a total of ninety-eight cases of typhoid fever out of a company of 248 men.

In seeking the sources of the infection of this outbreak we naturally turn to the patient who was first ill, namely W. F. The following notes were made of the case:—

F., 1416955 W., Private Fourth Casual Company, J.A.R.D., Camp ——. Onset of illness of this patient seems very obscure. He states that he had rheumatism at Camp —— all the time he was there. He states that he was at the base hospital for two months. Patient states that he did not feel well when he left the original cantonment in the United States, and did not feel any different en route. Patient proceeded with his company to the American rest camp at ——, England, and was admitted to the hospital on July 11 with a doubtful diagnosis. He was transferred to the base hospital on July 16. The patient’s temperature and pulse curves would indicate that his illness had continued for a considerable period. It was quite typical of a convalescent period of typhoid fever. This patient went with the company to bathe in the lake at Meridan, Mississippi.

Service record shows that inoculation with triple typhoid vaccine was completed on October 23, 1917, and paratyphoid vaccine was given on October 28, 1917. Patient states that he received at Columbus, Nebraska, five doses, and at Camp —— two doses of typhoid vaccine.
The following history is also of interest in connexion with the source of this outbreak.

B., 1415881 W. W., Private Fourth Casual Company J.A.R.D., Camp—. Patient states that he did not feel well when he left the original cantonment on June 15, and that he had stomach trouble and severe diarrhoea en route to Camp Merritt. At Camp Merritt he states that the doctor gave him some pills to relieve his trouble. Patient’s illness continued across the ocean on the steamer. He continued with his company to the American rest camp and was admitted to the hospital on July 11 with no diagnosis and was transferred to the base hospital on July 14 as a suspected case of typhoid fever. The clinical record of this case shows that when the patient first entered the hospital his fever and pulse-rate were lower than they were three or four days later, so that probably the real date of the onset of his typhoid fever was about July 10. It should, however, be noted that this increase in fever beginning about July 10 may have been a recrudescence of typhoid fever which began at the time he left the original cantonment. Patient’s service record states that inoculation with triple typhoid vaccine was completed on October 30, 1917. Patient states that he received three doses of typhoid vaccine at Camp Dodge.

**Chart showing Cases of Typhoid Fever by Date of Onset.**

### June.

<table>
<thead>
<tr>
<th>Cody</th>
<th>En route Camp Merritt</th>
<th>Camp Merritt</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30</td>
<td></td>
</tr>
<tr>
<td>B A</td>
<td>A B A A B A B A B A B</td>
<td></td>
</tr>
</tbody>
</table>

### July.

<table>
<thead>
<tr>
<th>En route to Liverpool</th>
<th>Camp Ramsey</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 2 3 4 5 6 7 8 9 10 11 12 13 14 15</td>
<td></td>
</tr>
<tr>
<td>A A A A A B B A B B</td>
<td></td>
</tr>
<tr>
<td>A A B B A B A B</td>
<td></td>
</tr>
<tr>
<td>A A A</td>
<td></td>
</tr>
<tr>
<td>B A</td>
<td></td>
</tr>
<tr>
<td>B B</td>
<td></td>
</tr>
</tbody>
</table>

**Note.**—(A) Cases from whom *Bacillus typhosus* has been isolated. (B) Cases clinical but no *Bacillus typhosus* isolated to date.
From the above chart it will be noted that a considerable group of cases developed during the latter part of the month of June and the early part of July. This suggests a common source of infection; there are two possibilities:

(1) That these cases became infected in the train early in the journey.

(2) That they became infected bathing in the water at Meridan, Mississippi, on June 18. The water may easily have been infected by one or two cases of typhoid fever which were present in the organization at the time.

The remaining cases of typhoid fever undoubtedly received their infection by contact with the patients who were ill in the organization. This could be easily done since the men were crowded together on the train or on shipboard.

There were about 1,800 men on board the steamer, this number being composed of Casual Companies 4, 10, 11 and 15 and Supply Company 112. Most of these organizations went on to France and among the members of Casual Company 4, fifty-seven other cases developed, as mentioned above.

All of the above mentioned cases had been inoculated with either typhoid vaccine and paratyphoid vaccine or triple typhoid vaccine, or both. Many of them have received several doses in excess of the required number. Also, the men were inoculated at widely different posts and at different times. One patient states that he received but one dose, otherwise it would seem that the whole organization was thoroughly immunized.
An Outbreak of Typhoid Fever in Inoculated Soldiers.

By J. G. Hopkins, Captain M.C., U.S.A.

(abstract.)

LABORATORY FINDINGS.

Of the forty patients in this series, admitted to Hursley Hospital as typhoid suspects, the laboratory findings were positive in twenty-five. The results are summarized in the following table:—

<table>
<thead>
<tr>
<th>Description</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacillus typhosus isolated from blood and from stool</td>
<td>4</td>
</tr>
<tr>
<td>Bacillus typhosus isolated from blood only</td>
<td>8</td>
</tr>
<tr>
<td>Bacillus typhosus isolated from stool only</td>
<td>5</td>
</tr>
<tr>
<td>Typical typhoid lesions at autopsy in cases from which the bacillus was not isolated during life</td>
<td>3</td>
</tr>
<tr>
<td>(Bacillus typhosus isolated post mortem from two of the above.)</td>
<td></td>
</tr>
<tr>
<td>Typical shift in typhoid agglutinins; blood and stool cultures negative</td>
<td>6</td>
</tr>
</tbody>
</table>

The remaining fourteen cases, all laboratory tests, were negative, although thirteen were clinically cases of typhoid.

BLOOD CULTURES.

Of ten blood cultures taken within a few hours of admission to the hospital, eight were positive irrespective of the stages of the disease. Of twenty-nine cultures taken after patients had been in bed twenty-four hours, only four were positive. This suggests that the physical strain the men had undergone may have caused a persistence of their bacteræmia.

POST-MORTEM FINDINGS.

Five fatal cases all showed typical typhoid ulceration of the lower ileum; one case showed evidence of recrudescence. Extraordinary enlargement of the spleen was found in two cases. The complications noted at autopsy were as follows:—

<table>
<thead>
<tr>
<th>Description</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intestinal haemorrhage</td>
<td>1</td>
</tr>
<tr>
<td>Perforation</td>
<td>1</td>
</tr>
<tr>
<td>Thrombosis and infarction of lung</td>
<td>2</td>
</tr>
<tr>
<td>Pneumonia (pneumococcus type 1)</td>
<td>1</td>
</tr>
<tr>
<td>Intramuscular haematoma</td>
<td>1</td>
</tr>
</tbody>
</table>
DISCUSSION.

Sir W. LEISHMAN, K.C.M.G.: I am sure that everyone here, like myself, must have been greatly interested in the very clear report on this outbreak given by our three American colleagues. From several points of view it is a very exceptional one as well as mysterious in several aspects. As regards the type of disease it is clear that this was the old severe form of typhoid with which I was very familiar in old days in India. This type, during the war, has been very rare, and, in my experience, only seen in individual cases and not in groups of men. The very high incidence in the unit attacked is remarkable, and it is difficult to account for this. Of the various possibilities suggested by the speakers it appears to me that the most probable was the fact that the men were very closely aggregated on board ship, and that there were, therefore, abundant opportunities for both massive and repeated infections to occur. It is possible also that the virulence of the infecting organism may have been exalted by rapid passage. The chief interest of the outbreak to me, however, naturally, centres in the fact that it took place in inoculated men. I may say at once in this connexion that we have no experience in our Armies of any outbreak among inoculated men at all comparable to that described. It is true that odd cases of a severe type are occasionally encountered, even after recent and apparently effective inoculation, but when severe cases are seen it is usually found on inquiry that the men have either never been inoculated or that their inoculation dates back to a period of two or more years. As to the apparent failure of vaccine in this instance, Captain Hopkins has very clearly discussed the various possibilities. Although men may frequently avoid inoculation by various subterfuges this cannot be responsible here, since there is clear evidence from the finding of paratyphoid agglutinins in their serum that they had received T.A.B. vaccine. The fact also that these inoculations were given at different times and in different places makes it impossible that a particular batch of vaccine may have been at fault through some error in
preparation or application. There is, in my opinion, no evidence to suggest that the strain of typhoid bacillus isolated from these cases was in any important respect different from the ordinary strains. It is well known that individual strains of *Bacillus typhosus* may differ in respect of their antigenic properties, but there is no evidence that we have in enteric fever to deal with more than three classical organisms of typhoid, paratyphoid A and B. Altogether the unusual features of this outbreak appear to me to remain without satisfactory explanation, and I can only say that our experience in the British Army, with the T.A.B. vaccine prepared at our Army Medical College, shows nothing at all comparable nor anything which can suggest the reason for the apparent failure of vaccine in this instance.

**Dr. Foord Caiger**: Quite apart from the clinical value of the record of these cases there are two questions of special interest which emerge from these reports: First, how did these men become infected? and secondly, why in such a large proportion of inoculated men did preventive inoculation prove a failure? It seems to me quite likely that the causes leading to these two results may be somewhat closely connected. Having regard to the close and intimate association of the men which was inevitable during their training, ship and camp life, quite apart from the fact of their having bathed together on one occasion, I do not think we need look much further for a reasonable explanation of the high percentage of infection. To refer in detail to the multiple opportunities of transmitting infection under these conditions would be but to labour the subject, and I will not occupy your time in doing so. The suggested explanation, that the men had bathed together, apparently on a single occasion, on June 19, even though the water is described as shallow, I confess does not impress me. It is perfectly true that the large bulk of the thirty-eight attacks developed during the course of the second and third week after the bathing, but that one or more of the unrecognized, but inevitable, opportunities of transmission incidental to the close conditions under which the men were living, was responsible for the spread of infection appears to me more probable, especially in view of the fact that fifty-seven other cases arose among the organization subsequently. Now, why it was that in such a large proportion of these men preventive inoculation failed to protect, is to my mind more difficult of explanation. The result is certainly disappointing, and to those who, like Sir William Leishman and myself, were members of the Committee appointed by the Army Council to investigate and report on antityphoid inoculation, the fact that in 35 per cent. of 248 men inoculation failed to protect, comes as somewhat of a shock, after the years of strenuous work which Sir William devoted to the subject during the course of that inquiry. That the vaccine was not at fault is probable, since it came from many and various sources. That the diagnosis of the cases was beyond question is attested both by the excellent clinical records and the careful bacteriological and serological data presented by Captain Hawn and Captain Hopkins.
Discussion on Typhoid Fever in Inoculated Soldiers

respectively. A point which occurs to me as possibly worthy of some consideration is the length of the interval following inoculation. In this series the interval appears to have been in most cases about eight or nine months. Now, although there is a general belief that the protection conferred by inoculation may be expected to last for a period of about two years in gradually diminishing degree in the human subject, I believe, if my memory serves me right, that specific antibodies cannot be detected in the blood after about five months from the time of inoculation. May not this have some bearing on the failure of protection in this series, especially if in the presence of what Sir William Leishman is inclined to regard as the most likely explanation—viz., a mass infection? To this view I incline, though not necessarily committing myself to the bathing hypothesis. No vaccine is capable of affording protection against a virus if present in overwhelming quantity. As regards the clinical aspect of the attacks, they were, as Sir William Leishman has rightly said, characteristic of typhoid as we knew it before the days of inoculation: 70 per cent. are returned as severe, with a mortality of 13.15 per cent., a death roll consistent with failure of inoculation. The diarrhoea rate, 58 per cent., is evidence in the same direction, diarrhoea being admittedly more frequent in severe cases of typhoid than in mild ones, which are more apt to be attended with constipation. Abdominal distension is recorded as having occurred in nine instances—i.e., almost 25 per cent. For this, I think, the treatment must have been responsible. With judicious dieting, coupled with the administration of a reliable antiseptic in efficient doses, distension should not occur at all in uncomplicated typhoid. Except as a result of perforation, I have not seen meteorism arise in a case of typhoid treated in my wards for the last twelve or fifteen years. Nose bleeding is given as occurring in as many as 26 per cent., a proportion which must be quite exceptional. The atropine test is recorded as positive in 79 per cent. of the cases. Since reading Captain Marris's paper, in which I was much interested, I have only had the opportunity of trying the test in some twelve or thirteen cases of typhoid fever, and in every instance it was positive. The number is, of course, small, but the test was in each case applied before the end of the second week, whereas I gathered from Captain Hawn that in few, if any, of his cases was it tried before the twenty-first day, which may account for the difference. As an early test I regard it as valuable, though controls have shown that it occurs not infrequently in other febrile conditions. Like the diazo reaction, its chief claim would seem to be its negative value in the earlier stage of the disease, before the agglutination test can be relied on.

Lieutenant-Colonel D. Harvey, C.M.G., R.A.M.C.: As officer-in-charge of the Vaccine Department of the Royal Army Medical College for the last five years the news that an outbreak of unmodified typhoid fever had occurred among inoculated American troops came as a severe shock; and this for two
Section of Epidemiology and State Medicine

reasons: first, that the vaccine as used in our Army is practically identical with that used by the American Army, the same strains being used, although the basis of our vaccine is still a broth culture of typhoid. Secondly, that previous to the war the American Army had got most excellent results with their vaccine, and had practically wiped out enteric fever in their old regular army, and also up to the present their results had been satisfactory. We were, of course, familiar, both before the war and during the war, with cases of typhoid occurring among inoculated men, but these cases were isolated instances and were modified by the inoculation. With only one exception we had no outbreaks of unmodified enteric among inoculated men, and in this one exceptional case in one body of men it was found that the vaccine used was not efficacious. I would suggest that possibly the fact that the men were inoculated frequently and at short intervals of time with varying doses of different strains of bacilli may have resulted not in an increased immunity for the typhoid element of the vaccine—as would have been the result if an interval of some months had been allowed to elapse between the various series of inoculations—but rather in a decrease of the immunity. For instance, I notice that it is stated on the protocols that one man completed his typhoid inoculations on October 23, and his paratyphoid on October 28, 1917. The question has been raised whether the strain which infected these men may not be a new race of typhoid bacillus against which our vaccine is not protective. Through the kindness of Captain Glynn, R.A.M.C., of Liverpool, I have been able to test this theory, as he supplied me with three strains of Bacillus typhosus isolated from the blood of cases of fever among men who had landed with this party. Unfortunately, owing to pressure of other work, I was not able to investigate the matter thoroughly, but so far as my experiments go they afford evidence that the strain isolated from these men is not a special and distinct race of Bacillus typhosus. I found that the serum of a rabbit immunized by our vaccine strain agglutinated this bacillus to the same extent as it did the homologous strain, and also that the serum of inoculated men who had received two doses of our vaccine, when tested against this U.S.A. strain, agglutinated it, and when tested by the opsonic method it was phagocyted to the same extent as our own strains. I was unable to carry out any tests as to the virulence of the strain, but possibly before subculture it may have been possessed of a high virulence. The question whether we should include this strain in our vaccine was discussed, but this has not been done, and our results continue to be satisfactory. In spite of the introduction of this fresh source of infection, cases of enteric fever have not occurred among troops associated with this body of men either on board ship, at home, or in France.

Captain Fred. M. Meader, M.C., U.S.A.: In reply to questions which have been raised in the discussion, I would state that I am not sure that bathing in the shallow lake had anything to do with the spread of the infection. However, the appearance of several cases during the period of ten to fifteen

JA—8b
days after this incident suggests that it may have been a factor. In regard to the inoculation with paratyphoid vaccine on October 28 in the given instance, I would state that the record is such that it is not possible to know whether the series of injections was begun or ended on that date. It is quite possible that only one dose of paratyphoid was given, and that on the 28th.
Section of Epidemiology and State Medicine.

President—Dr. E. W. Goodall.

PRESIDENT'S ADDRESS.¹

Enteric Fever in Flanders, 1914 and 1915.

By E. W. Goodall, M.D., Lieutenant-Colonel (Temp.), R.A.M.C.

The great War brought its cohort of fevers in its train, just as in the past international conflicts of less magnitude and ferocity almost invariably brought theirs. Of these fevers some were old acquaintances, whose reappearance was not unexpected; others, known to our forefathers, were strange to us, who christened them with new names and for their very novelty paid more attention to them than to their better-known allies. But as the mere waging of this war has involved the energies of so large a portion of mankind as to preclude up to now the setting forth to those engaged in it of any clear view of its purely military events and circumstances in their interwoven relations, so it is, I must suppose, that not yet has there been presented to us a comprehensive picture of the course and effects of the aforementioned diseases, which are part and parcel of the circumstances of the War. A snapshot here, a sketch there, rarely anywhere a finished picture, are all that have been produced. The clinician and the pathologist, especially the bacteriologist, have so far been the principal artists. Not that the epidemiologist has left his canvas untouched, as was shown by my predecessor in this chair a year ago. Moreover, as I have said, it is the more unfamiliar diseases that have attracted most notice, and it is partly, I believe for this reason, that so little has been heard,

¹ At a meeting of the Section, held December 13, 1918.
in this country at any rate, of the enteric epidemic in Flanders, and so much, comparatively, of epidemics of other diseases in various theatres of the War. Not that I can claim to furnish the finished picture. I did not see enough of the epidemic; I caught but glimpses here and there. It engaged the attention of several different authorities, those of the Belgian, French and British Armies and of the Belgian Civil Government (not to mention those of the German Army), so that only a writer who has access to the several official documents and has studied its phases continuously and on the spot, can hope to present a complete and accurate account of the epidemic as a whole. I saw and learnt enough, however, of its main features to justify me in bringing some account of it, however imperfect, to the notice of this Section.

It is certain that enteric fever was prevalent, though perhaps not widely prevalent, in Belgium at or about the time the War broke out, and soon after that date indications of an epidemic began to appear. Cases of enteric began to crop up in England during the autumn of 1914 among Belgian refugees who must have become infected in the country from which they had fled. I had a few cases of this kind under my own observation at the Eastern Hospital, Homerton. For instance, a certain family left their home in Werchter, a few miles from Louvain, on September 12 and went to Baal the same day. There they stayed till September 26, when they left for Antwerp, which they reached on the 27th. On October 4 they sailed from Antwerp for London and they arrived at the Alexandra Palace (then used as a refuge) on October 6. Two of the family, a boy and a girl, began to fall ill on the steamer between Antwerp and London. Allowing for an incubation period of ten days these children must have been infected about September 25, while they were at Baal. Similar cases continued to be met with in London up to January, 1915 [1].

You will remember that the German Army, after the first few days of the War, advanced rapidly through Belgium and many of the inhabitants fled hastily before it. Some of the advanced patrols of the German Army were reported at the time to have pushed forward so far ahead as to have got out of touch with the main body and to have been temporarily short of provisions, so that a silly rumour arose that the German Army was already getting into a bad way as regards its food supplies. The flight of the inhabitants and the rapid advance of the Germans were eminently calculated to spread enteric fever amongst the latter as well as the former, if the disease was already at all prevalent. Besides these factors the nature of the invaded country has
to be considered. The whole country west of a line drawn through Antwerp and Brussels is a plain, intersected with rivers, mostly canalized, canals and ditches, the water in which can flow but very slowly. This plain is in places thickly populated and highly cultivated, and human excreta are largely used for the purpose of fertilization. The water supply is derived, at any rate in the villages and small towns, from wells, mostly shallow. It will be remembered, further, that the Belgians as a defensive measure flooded the country to the north-east of Dixmude, and the scheme of inundation was completed by November 18. The effect of this was to accentuate the natural stagnation and to swamp large areas in that part of the country. About the third week in November the German Army was held up at a line, which, starting from near Nieuport on the coast, ran southwards east of Ypres and west of Lille; trench warfare began, and the trenches, largely in consequence of the inundations above mentioned, became flooded. Further, those who, like myself, are convinced that antityphoid inoculation is a valuable prophylactic against typhoid fever would adduce as another favourable condition the fact that of the four armies engaged in Flanders, only the British was at all adequately protected by that measure. From what was said at a meeting of the Strasbourg Society of Military Surgeons [2] towards the end of 1914, it appears that antityphoid inoculation was not practised in any thoroughness in the German Army during the first months of the War. This statement is corroborated by Goldscheider and Kroner [3] who, in September, 1915, recorded the fact that antityphoid inoculation was not begun in that army till late in October, 1914; and again later, by Henke [4], who in an analysis of autopsies of German soldiers who died of enteric in Flanders at that time (1914-15) states that few of them had undergone inoculation. As regards the French Army Vincent and Muratet [5] write that "the circumstances of the present war did not at first permit of the universal employment of preventive vaccination in the Army"; and Blum and Voisin [6], in drawing attention to the modification which enteric fever in the French Army underwent soon after the beginning of 1915, concluded that the change was due to the fact that by that time the troops were inoculated, whereas before that date they had not been so protected. With all these favouring conditions it was not surprising to find Major Stedman, R.A.M.C. (T.), writing to the Times on December 1 to say that there was a good deal of typhoid amongst the Belgian troops and that an epidemic was to be feared in Flanders. He suggested that a hospital
for 600 beds should be established at La Panne and another for 300 at Calais. According to a statement in the British Medical Journal a few days later (December 5, 1914) [7], cases from the Belgian Army began first to arrive at Calais on November 20, and came in at the rate of twenty to thirty a day. On December 2 the Hon., now Sir Arthur Stanley, and Sir H. C. Perrott, of the Joint Committee of the British Red Cross Society and of the Order of St. John of Jerusalem, wrote to the Times stating that the Committee was making a grant of £10,000 for hospitals for typhoid amongst the Belgians, and on the same date a leading article in that newspaper drew attention to the seriousness of the position. On December 5 there was published, also in the Times, an anonymous letter, signed "W. A. F.," in which the writer stated that a week previously he had been in a town in South-East Belgium, occupied by the Germans, that there were 1,500 cases of typhoid in the town, and that the authorities were providing accommodation for 4,000 additional cases. In another leading article the Times suggested that there was a good deal of typhoid in the German Army and that the disease might spread. On December 7 two letters appeared, one from Colonel Fairholme, the other from Dr. Melis, who were both at Dunkirk, accusing Major Stedman of exaggeration. Dr. Melis, who was officially connected with the Belgian Army, stated that there had been only 126 cases amongst the Belgian troops. The Times returned to the subject the next day with a special article in which it repeated the belief that the German Army was infected, and added the suggestion that the disease was likely to spread through floods, a view which was supported by a writer in the Lancet for December 12, 1914. There the subject ended, so far as the Times was concerned. But the 126 cases in the Belgian Army, admitted by Dr. Melis on December 7, were quite sufficient to point to what was taking place there and was likely to happen later. The epidemic must have been well on its way about that time; not only was the Belgian Army affected, but also the British, French and German Armies, and Belgian civilians. Of the British and French military cases I shall make mention later. As regards the German Army there is evidence, besides that to which I have already alluded, that the military authorities had considerable anxiety on account of typhoid during the last quarter of 1914. There were meetings of German medical men at the end of 1914 and beginning of 1915, and the British Medical Journal [8] of May 29, 1915, stated that "in the accounts appearing in the German medical press figures are not given, but enough is published to show that these outbreaks
have given great trouble and that they have in some respects differed from the classical picture of typhoid fever." According to Bruns [9] there were 20,000 cases in the German Army during 1914-15. The German writers Goldscheider and Kroner, whom I have already quoted, state that the disease appeared in the German Army during the second week in September, 1914, and reached its height in November. I do not mean to suggest that the typhoid in either the French or German Armies was confined to the Flanders area. It was certainly prevalent elsewhere, for in March, 1915, we find Professor Grober [10] discussing its prevalence on both German fronts, and as regards the French, according to Bousquet [11], there was an epidemic during the first fortnight of September, 1914, in the military territory of Belfort. Factors which helped both to propagate and foster the epidemic were overcrowding and difficulties with the supply of water and the removal of excreta. For instance the search parties of the Friends' Ambulance Unit found later on, in March and April, 1915, that at Brielen, with a normal population of 800 to 900 persons, there were 500 refugees; in the commune of Proven, besides the usual population of 1,600, there were 600 refugees; in that of Watou, population 3,500, there had been as many refugees, and even in the middle of April there were still 2,500. In Vlamertinghe there were 3,000 refugees. It must further be remembered that many of these towns and villages also contained large numbers of soldiers, billeted and encamped. Inquiry elicited that in some of these places the water from the wells had never been satisfactory in quantity or quality even before the War, and was usually "troublée" after heavy rain. The overcrowding increased the difficulty as to quantity.

It would appear that the outbreak began to assume epidemic proportions soon after the armies settled down to trench warfare in November, 1914. By the end of the year the epidemic was assuming a very serious aspect, and was compelling the attention of the various authorities. Many civilian cases of enteric were daily being brought to the notice of the medical staff of the Friends' Ambulance Unit, who were stationed at Ypres, and it is through my membership of this organization that I learnt most of the facts which I am about to relate.

The Unit had posted a party in the affected area, Ypres and its neighbourhood, as early as October, 1914. After several adventurous attempts to find a suitable refuge for the sick and wounded inhabitants who still remained in and about the town, the Unit obtained possession
of part of a large asylum for lunatics, L'Asile du Sacré Coeur, the other part of which was occupied by a military ambulance. This was a fine building, erected only a few years previously just outside Ypres on the Poperinghe road. At that time, December 1, 1914, it had escaped damage. On December 21 and 22, however, it was shelled, and the inmates had to be evacuated. But-a return was made a few days later, on December 27, and the building, most of the wards of which remained intact, was used as a hospital by the Unit till the second bombardment of Ypres in April, 1915. The Unit's local headquarters were also set up in this institution.

Besides the treatment of patients in hospital the medical officers of the Unit carried on an extensive outdoor visiting practice amongst the remaining inhabitants of Ypres and the surrounding villages not occupied by the enemy. During the latter part of December cases of febrile diarrhoea, which roused a suspicion of enteric, began to occur in considerable numbers. The medical officers seem at first to have had some doubt as to the diagnosis, because the cases did not correspond to enteric as they had known it in times of peace in England and other countries, and there was no means then available to them of supplementing clinical by bacteriological methods of diagnosis. It was no very long period, however, before the true nature of the cases was recognized.

From the middle of November, 1914, to the middle of January, 1915, Ypres had been evacuated by the armies. Then both the British and French Armies returned, and the sanitary conditions necessarily occupied the earnest attention of the medical departments. The Friends' Ambulance Unit was now able to undertake a very important rôle. I have been informed that in January, 1915, there were upwards of 8,000 civilians still in the town of Ypres, and large numbers in the neighbouring villages. But few Belgian medical practitioners remained. I suppose there had been a large call on the profession in that part of Belgium for service in the army and amongst refugees elsewhere. Moreover, those doctors who stayed had not such facilities for getting about as were at the disposal of members of the Unit. Consequently the services rendered by the Unit in succouring the sick and distressed had greatly endeared it to the inhabitants, with whom, therefore, it had considerable influence. Indeed, the hospital in the asylum had been recognized by the Ypres Committee of Safety as the Ypres Civil Hospital. The authorities of the British Army, therefore, acted wisely when they
determined to enlist the services of the Unit as a friendly link between themselves and the Belgian Civil Authorities, for while it was absolutely necessary to enforce certain measures in order to ensure the safety of the health of the armies, it was desirable to do so with as little friction as possible between the British Army on the one hand and the Belgian Civil Government and population on the other. Not only did the Unit carry out this delicate and difficult duty with tact and success, but its share in the execution of a number of sanitary measures which it was found necessary to put into action was large and important. The heads of the Unit at that time were: Mr. Philip Baker, the officer in command, Mr. Geoffrey W. Young, the officer in charge of the work in the Ypres area, Dr. Humphrey Nockolds (now Captain Nockolds, D.S.O., R.A.M.C.), the principal medical officer, and Dr. G. R. Fox, officer in charge of the medical arrangements at Ypres. The representative of the Medical Service of the British Army was Captain M. Coplans (now Major Coplans, D.S.O., R.A.M.C.), Sanitary Officer of the British Army, Ypres-Poperinghe zone, to whom, as also to Captain Henry, I am indebted for much information.

Area Affected by the Epidemic.—Whilst we know that there was a considerable amount of enteric in the German Army in Flanders towards the latter part of 1914, we know nothing as to the extent to which the civil population in the territory occupied by the enemy was affected. The same uncertainty exists in respect of the area over which the epidemic spread itself. I can speak only of the country occupied by the Allies. The patients, civil and military, who came under the cognizance of the Friends’ Ambulance Unit came almost entirely from that small corner of Belgium which the Germans had not invaded, from Nienport on the north to Ploegsteert on the south. This is an area about thirty-one miles long by twelve broad. The patients were Belgian civilians and French soldiers. A considerable number of the Belgians were refugees from places within the German lines; a few of them, at the beginning of the epidemic, may have been infected in these places. Most of them, however, became attacked in the towns and villages in which they had found a temporary home. A few French soldiers under my care had fallen ill in places at a considerable distance from the frontier on the French side; but they may have been, and most probably were, infected at the Front. As the medical officers of the Unit were brought into contact only with patients who were either French soldiers who had been on active service
at the Front when they fell ill, or with Belgian civilian inhabitants and refugees, I cannot say with absolute certainty whether or not the epidemic flowed over the Franco-Belgian frontier. I have no evidence that it did. No Belgian refugees stayed in that part of France, and French civilians were not allowed to cross over the frontier into Belgium. As it was the refugees who brought the disease into the Belgian villages away from the Front, in their absence from French territory the presence of the disease was hardly to be expected. The authorities of Dunkirk were naturally apprehensive of an outbreak on account of its proximity to the epidemic area. But so far as I could ascertain no cases arose in that town or its suburbs during the three and a half months I was there (January to May, 1915).

Nor am I certain whether the epidemic suddenly and quickly spread over the area mentioned above, or gradually invaded it. The cases first dealt with by the Unit naturally came from its centres of work, Ypres and Poperinghe, and their immediate vicinity; but there are reasons for believing that the epidemic spread rapidly towards the end of December, 1914.

Number of Cases.—As with regard to the extent of country affected so in respect of the number of persons attacked my information is limited; but so far as it goes it indicates that the number was very considerable. During my stay in Dunkirk I visited three large military hospitals in that port and its immediate neighbourhood which contained between them about 3,000 beds, nearly all of which were occupied by enteric cases; and there were other hospitals concerning which I received information, but which I did not visit, containing some hundreds of similar cases. So anxious were the French authorities for increased accommodation for enteric fever cases amongst their troops in the Ypres sector that they appealed to the Friends' Ambulance Unit for assistance in providing them. Even if it is admitted that not all the cases received into these hospitals were enteric yet there must have been somewhere about 4,000 cases under treatment daily during January and February, 1915, in Dunkirk and its neighbourhood. How many cases there were in the Belgian Army I do not know, but from the statements already quoted from the Times and the British Medical Journal the number must have been considerable. The British Army furnished comparatively few cases, only 827 up to May 22, 1915. As for the German Army I have already stated the reasons for concluding that there were in it a very large number of cases. Of Belgian civilians in round numbers 1,000 passed
through the wards of the hospitals of the Friends' Ambulance Unit at Ypres and Poperinghe, all of them suffering from enteric; most of the admissions were during the three months January to March. It has been estimated by one of the medical officers of the Unit who is thoroughly conversant with that branch of the Unit's work, that about 1,000 other cases came to the cognizance of the officers of the Unit, patients who remained in their homes, or were admitted to other hospitals, Belgian and British. Doubtless there were other cases of which the Unit never heard.

Character of the Cases.—As I have already stated, there was at first some doubt as to the true nature of the disease; but before I joined the Unit the medical officers had come to the conclusion that most of the cases were enteric. On February 1 I went to Poperinghe and stayed there for two or three weeks, so that I had ample opportunity of clinical observation both there and at Ypres. I confirmed the opinion already expressed by the medical officer in charge, that the disease was enteric, though the type was a little different from what I had been accustomed to meet with in London. I quote from a report which I made to the Committee of the Unit at the time:—

"The patients were Flemish civilians. The majority of them were refugees. . . . A few of them showed signs of deprivation of food, and many were very dirty, and were the subjects of bed-sores and other cutaneous lesions when they were admitted. Women were in a decided majority, which was, I suppose, to be accounted for by the fact that a large number of the men were serving in the army. The majority were of the ages at which enteric is most common, 15 to 25. The type of disease was severe, especially amongst the Ypres cases. . . . The patients were very ill on admission; it was rare for a patient to be admitted who had been ill less than a week, and most of them had been ill for two or three weeks or longer.

The disease as I saw it at Poperinghe and Ypres differed in several points from the types I had been accustomed to see in England during the previous twenty-five years.

(1) The fever, as shown by the temperature charts, ran a very irregular course, the oscillations being frequent and extreme, so that the curve on the chart at the height of the disease resembled in many cases that of a case of pyæmia or phthisis rather than of enteric fever. High temperatures, 104° to 105° F., were by no means uncommon, though the temperature seldom remained at this height for any length of time.

(2) A considerable number of the cases exhibited pronounced nervous symptoms, coma, or semi-coma, or on the other hand restlessness and delirium. An interesting point was that in the cases which recovered the mental symptoms did not quickly pass off as the general condition improved
and as the temperature fell. The comatose cases did worse than the others; in such cases death occurred without recovery of consciousness, even though the temperature fell. I am inclined to attribute this mental state to the harassing conditions under which the patients had been living for some time before they were taken ill.

(3) The clinical evidence went to show that probably the intestinal lesions were by no means extensive or severe. Death was due either to cardiac failure, probably caused by myocarditis, or to hypostatic pneumonia. The number of cases of intestinal haemorrhage and perforation were distinctly below the average. While I was at Poperinge there was only one case in which there was reason for supposing that perforation had occurred (no autopsy was made), and two of haemorrhage; both the latter cases were slight so far as the haemorrhage was concerned. Including the cases in the wards of which I had charge and those under the care of Dr. Rees, there must have been nearly 120 cases through the hospital while I was there. I was informed by Dr. Fox and Dr. de Wulf that the same scarcity of perforation cases was noticed at the Sacré Cœur Hospital at Ypres and the civil hospital at Poperinge. The clinical symptoms were borne out by such post-mortem evidence as I was able to obtain. An autopsy was performed on eight cases at Poperinge. The conditions under which the examinations were made were such as allowed us to inspect only the abdominal organs. In every case the intestinal ulceration which is present in the vast majority of cases fatal from enteric fever, was found, but in only one of the eight cases was it recent. In the remaining seven cases it was comparatively old, and in two or three it was in process of repair. In only one of these seven cases had the ulceration been extensive or deep." [This discrepancy between symptoms and intestinal lesions was found also by Henke [4] in the case of German soldiers who died of enteric in this epidemic.]

Later on, at Dunkirk, during the months of March and April, I had under my observation upwards of 100 French soldiers who were suffering from enteric fever. On the whole these cases were certainly not so severe as those I had seen at Poperinge and Ypres, even though they were cases in which in most instances the infection had been received at or in the neighbourhood of those places. They conformed, also, to the type of the disease I had been used to seeing in England. Several reasons may be given to account for this difference. In the first place the patients, who were soldiers of the French Army, had been well-cared for before they were attacked by the disease; and especially they had evidently all been well fed. In the second place a considerable number of them had been inoculated against typhoid fever. The civilian patients I saw at Ypres and Poperinge had not been inoculated. Thirdly, a large proportion were not cases of typhoid, but of para-typhoid, and these diseases are well-known to be usually less severe
than typhoid. Doubtless some of the cases I had seen at Poperinghe were cases of paratyphoid, but not till after I left that place was it possible to apply serum tests or make bacteriological examinations. Even if there had been a large proportion of paratyphoids amongst the Poperinghe and Ypres cases which I saw in February, 1915, the fact remains that that group of enteric cases was of a severe type. It is possible that paratyphoid might under certain circumstances assume a severe character, and Marcel Labbé [12] has described a small epidemic in which the fatality was as high as 11 per cent.

Early in March, 1915, the laboratory at the Queen Alexandra Hospital, Dunkirk, the temporary hut hospital erected by the Unit in response to the request of the French military authorities, came to our assistance. The laboratory was under the charge of Dr. H. T. Gillett, who was responsible for the blood cultures and serum tests of the patients admitted to the hospital. He also investigated a number of the cases in the hospital at Poperinghe during March; later the examination of these cases was undertaken by bacteriologists of the R.A.M.C.

As soon as laboratory methods of diagnosis were employed it was found that a considerable number of the enteric cases in both these hospitals were paratyphoid. In forty-three cases at the Queen Alexandra Hospital in which the blood cultures were positive, sixteen were typhoid, twenty-two were paratyphoid A, three were paratyphoid B, and two were paratyphoid, though whether A or B was not ascertained. If we add to these cases those in which the results of the blood serum tests were taken as clinching the diagnosis, then there were forty-three cases of typhoid, thirty-two of paratyphoid A, twelve of paratyphoid B, and two of paratyphoid, A or B; so that rather more than half, 51·6 per cent., were paratyphoid. This proportion is vastly different from that found amongst the Belgian civilians admitted to the Hôpital Elisabeth at Poperinghe. Out of 308 cases in which the clinical diagnosis was supplemented by positive blood cultures or serum tests, 245 were typhoid, sixty-two were paratyphoid B, and only one paratyphoid A; that is 20·4 per cent. were paratyphoid. Probably several causes account for this difference. In the first place the Dunkirk cases were fewer in number than those at Poperinghe; secondly, the Dunkirk cases occurred during the two months from the middle of February to the middle of April, while the Poperinghe cases were spread over the period from the beginning of January to the middle of September. I am referring to the dates at which the patients must have been infected. Thirdly, the
proportion of patients in the two groups who were inoculated against typhoid is different. Of the 102 cases in the Queen Alexandra Hospital at Dunkirk which were diagnosed as enteric, thirty, or 29.4 per cent., had not been inoculated; while of the 454 admitted to the Hôpital Elisabeth at Poperinghe, 418, or 92.0 per cent., had not been inoculated. In the fourth place, local conditions must be taken into account, such, I mean, as the presence in any particular area of more or fewer cases of the three varieties of enteric. At the Italian front, for instance, during the period December, 1915, to February, 1917, it was found that the proportions of the three varieties varied from time to time. According to Crossonini [13], during the period December, 1915, to July, 1916, the percentages were as follows: Typhoid, 38; paratyphoid A, 6; paratyphoid B, 56; while during the period August, 1916, to February, 1917, they were: typhoid, 9; paratyphoid A, 73; paratyphoid B, 18. Crossonini attributed these variations to the variations in the proportions of inoculated men: but the factor I have just mentioned must not be forgotten. In their book, "Typhoid Fevers and Paratyphoid Fevers," Vincent and Muratet, referring to certain statistics derived from the French Army since the outbreak of this war, write as follows: "These statistics . . . show that in the Army in campaign, paratyphoid A fevers are much more frequent than paratyphoid B. In certain regions, however, paratyphoid B has been predominant" (loc. cit., p. 91). In the British Army in Flanders during 1914-15, 50 to 60 per cent. of the enteric cases were paratyphoid, while, according to Sir William Leishman, there was amongst the paratyphoid cases an excess of paratyphoid B varying from three-fourths to two-thirds. In the British Army in the East, Dr. Buchanan told us last year that, with some local exceptions, paratyphoid A predominated. Fifthly, the Dunkirk patients were French soldiers, the Poperinghe patients were Belgian civilians. The occurrence of cases of paratyphoid A in considerable numbers was one of the surprises of the War so far as enteric fever is concerned. It is generally believed that it was introduced by carriers in troops from the East. Paratyphoid A was very rare in Europe before the War, and paratyphoid B was not common. Not more than 3 per cent. of all enteric cases were paratyphoid and they were paratyphoid B. Vincent and Muratet, however, state that before the War Germany was the most important seat of paratyphoid, of both A and B variety. They place the incidence of paratyphoid in the enteric class in that country.

as high as 10 per cent. Before the War, in France, especially in the army, paratyphoid B occurred in small local epidemics, and paratyphoid A was rare. It is clear, therefore, that in given numbers of enteric cases in this War, the proportion of the three varieties has varied according to time and place. The figures given above indicate that the Belgian civilians were not exposed to the infection of paratyphoid A, but that the French soldiers were. This was probably the case, on account of the presence amongst them of troops from the East, native as well as European. In this epidemic, as in others in this War, the three varieties of enteric were mingled one with the other, and there were cases of double and triple infection, which I have not differentiated. Last, and perhaps not least, the warning must be repeated which was sounded by Dr. Buchanan in his president's address of a year ago, that different laboratory methods in different hands will yield different results.

As to the severity of the epidemic, as gauged by the fatality, I am able to give the figures only for the cases treated in the two hospitals at Poperinghe and Dunkirk. Of the 435 cases of undoubted enteric fever admitted to the former hospital during the febrile stage of the disease, ninety-three died, a fatality of 21·3 per cent.; while of 102 such cases admitted to the latter, ten died, a rate of 9·8 per cent. The Poperinghe cases were of all ages and both sexes, with a majority of females; the Dunkirk cases were all males, mostly from 20 to 40 years of age. The Poperinghe rate is somewhat high, the Dunkirk rate is certainly low. I have already given the reasons for the difference in the severity of the two groups of cases, reasons which apply to a certain extent to the differences in the fatality rates. I am bound, however, to add that there is a curious difference in the fatality rates of the typhoid and paratyphoid cases in the two hospitals. For the Hôpital Elisabeth they are as follows: Typhoid, 9·7 per cent.; paratyphoid, 19·2 per cent. For the Queen Alexandra Hospital: Typhoid, 20·9 per cent.; paratyphoid, 21 per cent. In the absence of other details respecting the Poperinghe cases, I am quite unable to explain these differences. The Dunkirk figures conform with the fatalities of typhoid and paratyphoid as met with by other observers in various countries and at different times, but they are too scanty for trustworthy conclusions.

So much for the general character of the epidemic in so far as it came within my knowledge. I will now turn to the measures employed against it. As is the case in any epidemic they were (1) those for providing for the sick; and (2) those for protecting the healthy.
First, as regards the provision for the sick. The medical departments of the several armies dealt with the soldiers, and the Friends' Ambulance Unit chiefly with the civilians. Even before the regulations as to the removal of enteric cases were issued by the Belgian authorities the Unit began to receive such cases into the Hôpital du Sacré Cœur at Ypres towards the end of December, and 100 beds were set aside for enteric in that institution. In consequence of the increase in the number of cases the Hôpital Elisabeth was opened at Poperinghe a month later. This was a large private house on the outskirts of the town which happened to be empty at the time. Originally, there were seventy beds, but in the course of a few weeks huts were erected to accommodate eighty more patients. At first Dr. Rees was in charge, afterwards Dr., now Captain, Henry, R.A.M.C. It was, however, even as early as January, clear to the Unit that it would not be able to cope with all the cases that were certain to require removal to hospital. The authorities of the British Army, therefore, in collaboration with the Belgian Government, established a large hospital at Malasisse, near St. Omer, about twenty-five miles from Poperinghe and thirty-two from Ypres. Belgian doctors and orderlies were employed in this hospital, so that the patients should not feel that they were entirely amongst strangers. As this hospital was too far from the Ypres district for the patients to be visited by their friends, it will be readily understood that some apprehension arose, to allay which the Belgian civil authorities issued handbills explanatory of the position, and pointing out how information as to the progress of the sick could be obtained. The patients were removed from the hospitals at Ypres and Poperinghe by ambulance convoys of the R.A.M.C. At first only convalescent or nearly convalescent patients were removed, and the first convoy ran on February 7. Later, I understand, cases in earlier stages of the disease were transported, and the two hospitals near the Front were used as casualty clearing stations for enteric patients. The convoys continued to run till the hospital ceased to be used for enteric cases, and so far as I can learn without any mishap, so admirably were the arrangements designed and carried out by the A.M.S.

After the German attack in April, 1915, many patients were removed straight from their homes to the hospital at Malasisse. I have already mentioned that other hospitals were established not only for soldiers, but for civilians: at Coxyde and Bailleul, for instance, but of these I have no details, except of the Queen Alexandra Hospital at Dunkirk, which was erected by the Friends' Ambulance Unit at the request of
the French military authorities. There were 200 beds in the hospital, but its use for enteric cases continued for only about three months, as the shelling of Dunkirk at long range towards the end of April led to the removal of the enteric cases elsewhere.

(2) As regards the measures adopted for preventing the spread of the epidemic by protecting the healthy, the authorities acted quickly and energetically. They were such as are usually taken in epidemics of this nature—viz.: (a) The removal and isolation of the sick in the hospitals already mentioned; (b) the cleansing and disinfection of premises; and (c) the provision of a safe water supply. Besides these there was an unusual measure—namely (d) the inoculation of the civil inhabitants of the infected areas with antityphoid vaccine.

The difficulties in carrying out these measures were considerable. The affected country reached right up to the very trenches, and embraced a large tract which was at any time liable to enemy attack. Troops of three armies were billeted in the towns and villages. It was necessary to enlist the co-operation of the authorities of three armies and of a civilian population. But these obstacles formed little, if any, hindrance to the execution of a comprehensive plan. In order to remove the infectious sick to hospitals, the sick must first be found; to cleanse insanitary premises, they must be known and inspected. The sick soldiers could be and were dealt with by the Army authorities. The difficult problem was the civilian population. In consequence very largely of conferences between representatives of the Belgian Government and local civil authorities, of the British Army, and of the Friends' Ambulance Unit, a concerted and drastic action, on the lines indicated, was agreed upon. In the first place the Belgian authorities issued an order by which (i) antityphoid inoculation was made obligatory for the civil population in the zone occupied by the allied armies; and it was ordered that persons refusing to be inoculated should be expelled from the zone and treated as refugees; (ii) enteric fever was made notifiable by the medical practitioners to the local authorities and the inspector of the Service du Santé; wilful omission to notify was punishable by a fine or imprisonment. Persons ill of enteric were to be moved to hospital if necessary. A notice stating the nature of the illness in French and Flemish was to be placed on the doors of houses from which a case of typhoid was removed; and the notice was to remain up till the house was disinfected. Inoculation and disinfection were to be performed gratuitously.

These measures are sufficiently drastic. I am not aware of another
instance in which inoculation against typhoid has been made compulsory upon a civil population, with a punishment of deportation for refusal. Maurange states that upwards of 5,100 civilians were inoculated in and about Paris in September, 1914, but in this instance inoculation was submitted to voluntarily.

Even before the Government order was promulgated inoculation against typhoid had been urged upon the civilians in Ypres by the Friends' Ambulance Unit, and the first inoculations were performed on February 5 by its medical officer. Bills and posters were prepared and distributed, in which inoculation was urged. Inoculation stations were distributed in and about Ypres; and a large number of persons submitted themselves to this prophylactic. Through the kindness of Sir George Newman, K.C.B., the Chairman of the Committee of the Unit, I am able to give the number of civilians inoculated by the medical officers from February 5, 1915, to the end of May, 1916. The table has been compiled by Captain H. Nockolds, D.S.O., the principal medical officer.


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<th>1915</th>
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<td></td>
<td>First</td>
<td>Second</td>
<td>Per cent.</td>
<td>Total</td>
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<td></td>
<td>1915</td>
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<tr>
<td>February 5 to 28</td>
<td>4,890</td>
<td>2,649</td>
<td>54.1</td>
<td>7,539</td>
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<tr>
<td>March 1 to 31</td>
<td>4,942</td>
<td>4,576</td>
<td>92.5</td>
<td>9,518</td>
<td></td>
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<tr>
<td>April 1 to 30</td>
<td>1,010</td>
<td>972</td>
<td>96.2</td>
<td>1,982</td>
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<td></td>
<td>10,842</td>
<td>8,197</td>
<td>75.6</td>
<td>19,039</td>
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<tr>
<td>Up to end of July, 1915</td>
<td>14,886</td>
<td>11,375</td>
<td>76.6</td>
<td>26,211</td>
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<td>&quot; , May, 1916</td>
<td></td>
<td></td>
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<td>26,700</td>
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(totals inoculations)

Per cent. = percentage of first inoculations inoculated a second time.

I have dealt first with the inoculation of the civilian population because it is the subject of the first article in the Belgian Order; but it was by no means the only nor even the principal of the prophylactic measures adopted. Not even the most ardent advocate of inoculation claims that it will protect everyone. A very urgent question was the water supply. I have said that this was mostly derived from surface wells. I was informed that the supply of Ypres was good before the War. The water-tower remained undamaged up to April, 1915; but the bombardment of the town in November, 1914, had broken many of the mains, and the supply could not be maintained. The inhabitants had, therefore, to fall back on water from wells and the moat. In
January, 1915, the Burgomaster had ordered the boiling of all water used for drinking and for cleaning food utensils. But it was deemed advisable to sterilize the water as far as possible before it was delivered. Under the direction of Captain Coplans the swimming bath situated at the north-east corner of the town and capable of holding 1½ million gallons, was cleaned and made use of as a storage-tank. Water from the adjacent moat was treated with chloride of lime and turned into the bath, from which it was pumped into barrels and distributed to seven fixed stations. Two or three times a day the water at the stations was tested by one of the members of the Unit, in order to ensure that no untreated water had been added surreptitiously. From 45,000 to 75,000 litres were distributed daily. At Dickebusch a similar system was installed. In other towns and villages the inhabitants were warned of the danger of drinking unboiled or untreated water. Chloride of lime was supplied to householders, together with a little spoon-measure and a printed card of instructions. The work of distribution was carried out almost entirely by the Unit, and up to the end of June, 1915, over 1,800 householders were supplied in Poperinghe and the villages near that town and Ypres.

The duties of finding the cases of enteric and investigating the sanitary conditions of the towns and villages in the area just mentioned were assigned to the Unit. Search parties were formed; each party consisted of a doctor, four voluntary orderlies, four interpreters (certain nuns who spoke French and Flemish), and two motor ambulances with drivers. The search was conducted systematically, street by street, and house by house. Cases of enteric were removed to hospital, unless they could be safely left. The sanitary condition was examined and investigation made as to the water and food supply. Inoculation was brought to the notice of the inmates and urged upon them. For each house a card was filled up giving the results of these inquiries, which was handed in to the local headquarters of the Unit the same evening so that a card index could be kept. A distinctive mark was placed on the door of the house, for the benefit of the sanitary squad, R.A.M.C., who followed the visits of the search parties. The search parties were working under the supervision of the sanitary officer, Captain Coplans, and reports as to the discovery of cases of enteric, the sanitary state of the premises and so forth were regularly furnished to him. The sanitary squad undertook the disinfection and cleansing. In Ypres, at any rate, a local contractor emptied the cesspits, disposed of the contents by burning or burial in fields away from the house, and executed repairs.
The search parties began their work on February 14 and continued it till June 28, 1915. The area which they thoroughly explored consisted of the quadrilateral Ypres, Elverdinghe, Poperinghe, La Clytte, and the triangle, Poperinghe, Proven and Watou. A few villages outside these areas were also dealt with on reports made to the Unit that their insanitary condition rendered a visit necessary. A list of infected houses was drawn up which covered a much larger area than that traversed by the search parties. The compilation of this list, indeed, was commenced before these parties got to work. Spot maps showing infected houses were also prepared. During the period mentioned above, four months, nearly 6,500 houses were inspected, of which 700 (about 10 per cent.) were found to be in a very insanitary condition. The bulk of this work was accomplished before the second bombardment of Ypres and neighbouring towns and the renewed German attack in April, and these acts of warfare brought most of this work to a standstill.

These are the facts—I regret they are so meagre—so far as they have come to my knowledge concerning this epidemic. The epidemiologist and the health administrator will ask, "What were the results of the measures I have mentioned upon the course of the epidemic?" The question is difficult to answer in respect of any epidemic; it is particularly so of this. All epidemics sooner or later die a death which is natural and without the interference of a human executioner. More than one instrument is usually employed to bring them to an end; each at its best is seldom perfectly efficacious, and, therefore, when apparently they have been effective, the share of each in the consummation is difficult to estimate. In the Flanders epidemic there was present at least one other than the usual factors—namely, the flight of large numbers of the civil population from the slough of infection before the renewed attack of the enemy. I was informed about the middle of March by certain French army medical officers in Dunkirk that so far as they were able to judge by the enteric admissions to the hospitals in that town, the epidemic was then on the wane in the French Army. By the beginning of the summer it was at an end, not only in the armies but also amongst the civilians. If I were to attribute this result to any human agency, I should attach more importance to the effects produced by the usual sanitary measures (the disinfecting of the water, the removal of the sick to hospital, the cleansing of houses, and so forth), than to the inoculations; and for the following reasons. The inoculations were against infection by Bacillus typhosus, and not against
infection by *Bacillus paratyphosus* A and B. Now we know that both amongst the Belgian civilians and the troops of all the armies engaged paratyphoid was frequent. It has been shown that antityphoid inoculation is not protective against paratyphoid infection. It cannot, therefore, be claimed that an inoculation which admittedly protected against only one portion of the cases which made up the epidemic, and that far from a negligible portion, was responsible for the cessation of the mixed epidemic. The Belgian civilians received only the antityphoid vaccine; and I believe I am right when I say that the triple vaccine was not employed in the French and British Armies till after the epidemic was virtually over. Inasmuch as antityphoid vaccine was not extensively used in the French Army and not at all amongst the civil population till the epidemic had attained a considerable height, I should expect to find as a beneficial result of that vaccine an increase in the proportion of paratyphoid as compared with typhoid cases towards the end of the epidemic. This relation between these two varieties of enteric fever appears, indeed, to have been reached in the British Army; for according to Sir William Leishman, of the 112 cases of enteric in that Army which were under treatment in the middle of November, 1915, thirty were cases of typhoid, and ninety-two of paratyphoid fever. The clinical records of the cases admitted to the hospitals of the Friends' Ambulance Unit at Ypres and Poperinghe are in existence, and are, I understand, being tabulated. It would be most interesting to ascertain whether amongst the Belgian civilians paratyphoid cases also predominated towards the end of the period during which admissions to those hospitals continued. Of the Unit itself only two members were attacked by enteric fever, and in both cases the variety was paratyphoid. All the members had been inoculated against typhoid; a large number of them worked at one time or another in the Ypres-Poperinghe area, and not a few were brought into intimate association with sufferers from the disease.

With the knowledge that the civilians had not been protected against paratyphoid, I should not have been surprised and indeed was expecting to hear that another epidemic had arisen in the following autumn. But there was no epidemic. Sir William Leishman stated on November 23, 1915, that there were practically no cases in the Ypres-Poperinghe area on November 15. Nor was there in the autumn and winter, 1916-17. Major Coplans, in a letter to me dated September 28, 1916, stated that there were on that date signs of a recrudescence towards the Belgian coast; but it came to nothing.
I expected an epidemic in the winter of 1915-16 at any rate, in spite of the care which had been taken as regards water supplies and sanitary precautions generally, because if ever a soil was saturated with the poison of enteric, it was that of Flanders in the winter of 1914-15 and the following spring; but so far as my knowledge goes, there has been no enteric worth mentioning in that region since the epidemic which has been the subject of this address. I suppose that now the enemy has been driven from that country, the civilian inhabitants will soon be returning in large numbers. In those who were inoculated in 1915-16 the protective effects will by this time have disappeared. Every effort should and doubtless will be made to prevent a fresh outbreak. For even if the virus does not persist in the polluted soil, and according to Vincent and Muratet the paratyphoid A and B bacilli are much more resistant to external causes of destruction and also much longer-lived in water than are the bacilli of typhoid, yet the sanitary conditions can hardly have been so much improved as to render its reintroduction by cases or carriers at all unlikely.

REFERENCES.

Section of Epidemiology and State Medicine.

President—Dr. E. W. Goodall.

Some Simple Tests of Physical Efficiency.

By Martin Flack, Lieutenant-Colonel R.A.F.

(ABSTRACT.)

[The full paper will appear in a Report to be issued by the Medical Research Committee.]

I am bringing to your notice this evening some quite simple tests which I believe will be of use in determining the physical efficiency of an individual. I am approaching this problem from the point of view of a physiologist, and the tests have been devised to show the physical condition, not particularly of any one system, but of the individual as a whole.

The procedure has been to select as far as possible healthy controls. Officers of the R.A.F. who have made good and who have been selected by their commanding officers for their efficiency in flying and in fighting, have been examined and standards have been set, provisionally of course, as they may have to be altered. On the other hand, a number of officers who have broken down for some reason or other have also been examined and the results obtained compared with these healthy controls.

These tests are not designed to supplant the work of the clinician in any way, and when a man is reported as physically unfit on these tests, it does not mean that the work of the physician is finished, but that it is beginning. If the subject does not come up to the

1 At a meeting of the Section, held January 10, 1919.
standards on being overhauled by the physiologist, then the psychologist, neurologist, cardiologist, or general physician will find something is wrong with him. These tests give indications for such overhaul.

The chief point in connexion with these tests is the technique. This is important because, if adopted, it is essential that all the tests be carried out in the same way on all occasions, as various medical officers may apply them to the same individual at different times. It has been suggested in regard to the treatment of officers in the R.A.F.: that preventive treatment is best, and if officers were periodically subjected to these tests by medical officers at different stations, they would carry with them a certain definite amount of information.

The first test is the response of the pulse to exercise. The way this is done is important. I would point out that there is no limit to the number of times a man may be asked to stand upon a chair, but this technique is suitable to the examination of a large number of subjects. The test is that a man shall lift his body-weight through a definite height five times in fifteen seconds. The rate of increase in the pulse as a result of the exercise is noted and the rate of return to the normal, the pulse having been taken standing immediately before the exercise. In this way uniformity of result is obtained. Hitherto if ten medical officers were examining candidates, some might order the candidates to touch their toes four times; the enthusiastic man would do so in five seconds, the lethargic man in twenty seconds; thereby achieving no uniformity of result. The test as devised is an effort to set a level basis for all candidates. Preferably the sitting rate of the pulse is first taken. The pulse-rate is then taken standing. If the rate is unsteady it should be counted in periods of five seconds until a steady rate is obtained. The candidate is then put through a regulated exercise, which should be carried out as follows: Standing before a chair, he places one foot upon the seat of the chair and steadily raises his whole body to the height of the seat five times in fifteen seconds, one foot being retained on the chair throughout. The examiner should regulate the speed and rhythm in the following way: Standing beside the candidate, holding the wrist, with his fingers on the pulse, the examiner swings the arm forward and backward to indicate the time of raising and lowering the body. The subject still standing, the examiner then counts the pulse in five-second intervals, and notes the acceleration and the time taken to return to the previous rate. In a good subject the increase of rate is about twenty and the time of return to normal
fifteen to twenty-five seconds. If the time of return exceeds thirty seconds it is suggestive of cardio-vascular inefficiency.

The second test consists in getting the subject to hold his breath without any preliminary deep breaths. I would insist upon the actual lines on which the test is laid down being followed: A deep expiration followed by the filling of the lungs, clipping the nose, holding the breath as long as possible. The significance of the test should not be mentioned to the subject. He should just be told to breathe out and breathe in as far as possible and then to hold the breath. At the end of the test the question should be asked as to what is the sensation experienced by the subject.

The test was originally designed to show whether there was oxygen want, and I still believe the test does show the subject who would suffer from oxygen want. From my experience I found that people who were likely to suffer from oxygen want would give up after a very short time in holding the breath, and would almost invariably return an abnormal answer. A normal answer would be that the subject "had to give up," "felt he would burst," an abnormal answer that the "blood rushed to the head," "things became blurred," &c. The test, however, has other significance. The man without resolution, for example, will give up early.

As originally shown by Dr. Leonard Hill and myself, if a man who has held his breath in this manner then takes a lungful of oxygen instead of a lungful of air, the time of holding the breath will be increased from one and a half to two and a half times the time before. Therefore what one breaks down from is, in the first case, discomfort due to lack of oxygen, because when one breaks down in holding the breath on oxygen, the symptoms are quite different, and are those due to CO₂ excess, headache, sweating, &c.

Another interesting point in this connexion is this: It is known that the power to hold the breath is greatly diminished at altitudes. Therefore a man who can hold his breath a long time at ground level without discomfort will have greater room for diminution in his power to hold his breath than a man who can hold his breath but a short time at ground level before discomfort occurs. An efficient man at altitudes is a deep breather, whereas the man who is inefficient is a panter. The figures shown on the screen in regard to holding the breath were worked out in the first instance on forty successful pilots, all picked out by squadron commanders or by the Admiralty or the R.F.C. as being quite able. The time the breath is held averages about sixty-nine
seconds. You will also see from this table that the vital capacity averages at 3,800 c.c., with a minimum of 3,400, in efficient fliers.

The next table shows the results obtained from a number who were sent up for medical boarding, in which case the breath was held for not more than forty-five seconds, and in most cases they gave answers which showed they were not comfortable.

The deduction then is that the breath-holding test on an individual would be an idea as to whether he was likely to do well in the air. As the results appeared to show that poor breath-holders could not last in the air, the breath-holding test was adopted at the R.A.F. Commissions Board. In my opinion it is preventing people going into the Air Force who would not do well.

The question of "oxygen want" is a matter for serious future research, and in peace time one will be able to do such research on a more scientific basis.

The third test is a combination of the first two tests. Having got the pulse response and breath-holding, then the time the breath can be held after the exercise can be taken. In the unfit, the breath-holding power comes right down, probably thirty seconds. The fit man may possibly hold his breath almost as long as before, but will not have a fall of more than twenty seconds. The man out of condition gives a big fall in time after exercise.

The minimum standard for admission for the ordinary breath-holding test is forty-five seconds. Under forty-five seconds should be looked upon with suspicion, and probably the candidate graded in regard to the height to which he should go.

The Vital Capacity of Pilots.—The minimum in the table of successful flying officers is 3,400 c.c. I suggest that the use of a modified gas-meter is the best way of measuring vital capacity, and preferably one made by an English firm, which has the great advantage over the German model from which it was copied that its capacity cannot easily be overshot. Among officers who had broken down a great number of those tested were under the minimum of 3,400 c.c., but it was subsequently found that this was due to flying stress, some having a vital capacity of only 2,800 c.c.

Captain Bazett, M.C., R.A.F.M.S., has shown that in addition to these tests, if the respiration-rate is multiplied by the ventilation per minute and divided by the vital capacity, it is a very good indication of the power of a pilot to fly. A figure below thirty is good, a figure above thirty is poor. A test like this will be of value for the selection of the high flier.
The apparatus for the next test is a U-tube manometer, filled with mercury, with the scale movable. The test is a measure of the tone of the abdominal wall. The subject is asked to blow up steadily the mercury column as high as possible. The number of mm. Hg. blown is recorded. If for any reason it is suspected that the subject is not trying, he is asked to try again with the scale of the manometer turned away. There should be but little difference from the previous reading, and in such a case encouragement may cause the subject easily to surpass his previous effort. He is then asked to try again while looking at the column. If he is not trying he will not surpass his first effort, which he saw.

The sixth test is another test with the U-tube manometer. This test is performed as follows: The subject is asked to empty the lungs, fill up, blow the mercury to the height of 40 mm. and hold it there, without breathing, for as long as possible. The nose should be clipped. A valuable adjunct to this test is the behaviour of the pulse during the time the mercury is being sustained. It is counted during each period of five seconds that the mercury is sustained. Starting at the fifth second in the normal individual there is generally a slow steady rise in the rate of the pulse, or a fairly marked rise which is sustained most of the time. For example, the pulse-rate may rise gradually from 72 to 96 or 108, according to the time the breath is held, or it may rise at once from 72 to 96 or 108 and be sustained there. A large rise in rate—e.g., from 72 to 132 or 144 is unsatisfactory. In cases of flying stress a characteristic response is for the pulse to jump up to a quick rate during the fifth to the tenth or fifteenth second, and then to fall away in rate to normal or even below. Such a response is as follows: Normal at start, 84; fifth to tenth second, 144 (sometimes almost impalpable); falling away (say twenty to twenty-five seconds) to 72 or even 60. Such cardiomotor instability is frequently associated with flying stress and is indicative of a need of rest. In any case the subject is generally not in a condition to be allowed to continue to fly. Other points in the examination should, however, be taken into consideration.

The averages obtained for these tests from some selected flying officers were:

<table>
<thead>
<tr>
<th>Expiratory force</th>
<th>Mercury held</th>
</tr>
</thead>
<tbody>
<tr>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>112 mm. Hg.</td>
<td>52 seconds</td>
</tr>
</tbody>
</table>

It is suggested that candidates for the Air Force should all reach the minimum standard, and preferably the average standard.
The table gives a synopsis of results obtained from various sources:—

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Number examined</th>
<th>Breath held in seconds</th>
<th>Vital capacity in cubic centimetres</th>
<th>Supplemental air in cubic centimetres</th>
<th>Expiratory force in millimetres Hg.</th>
<th>Sustaining 60 millimetres Hg.</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fit instructors...</td>
<td>22</td>
<td>67</td>
<td>4,062</td>
<td>1,620</td>
<td>112</td>
<td>52</td>
<td>* Average</td>
</tr>
<tr>
<td>Ditto</td>
<td>—</td>
<td>46</td>
<td>3,300</td>
<td>1,000</td>
<td>80</td>
<td>43</td>
<td>+ Minimum</td>
</tr>
<tr>
<td>Home Defence pilots</td>
<td>24</td>
<td>72</td>
<td>3,940</td>
<td>1,496</td>
<td>119</td>
<td>50</td>
<td>One or two suffering from stress included in the table</td>
</tr>
<tr>
<td>British candidates</td>
<td>23</td>
<td>69</td>
<td>3,823</td>
<td>1,590</td>
<td>106</td>
<td>52</td>
<td></td>
</tr>
<tr>
<td>U.S. candidates</td>
<td>7</td>
<td>66</td>
<td>3,814</td>
<td>1,386</td>
<td>116.4</td>
<td>53.5</td>
<td></td>
</tr>
<tr>
<td>Delivery and test pilots</td>
<td>10</td>
<td>57</td>
<td>3,620</td>
<td>1,050</td>
<td>108</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Pilots returned for rest</td>
<td>17</td>
<td>57</td>
<td>3,897</td>
<td>1,423</td>
<td>95</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>Pilots training for scouts</td>
<td>15</td>
<td>62</td>
<td>3,820</td>
<td>1,433</td>
<td>96</td>
<td>49</td>
<td></td>
</tr>
<tr>
<td>Pilots taken off flying through stress</td>
<td>27</td>
<td>49</td>
<td>3,480</td>
<td>1,134</td>
<td>74</td>
<td>25</td>
<td></td>
</tr>
</tbody>
</table>

Practically all the cases examined in hospital fell below the standard. There was one very interesting case of a man who came with a letter to the hospital and was examined. I told him he seemed very fit, and on reading the letter afterwards found he was still doing very good service in France, and had not come as a patient to the hospital but for special examination on account of his fitness and meritorious flying service. Another was a pilot, whose only disability was that he was nearly blind in one eye. I could find nothing wrong by these tests.

Having found standards from the examination of successful pilots, I went to the Commissions Board and examined a number of rejects (see Table II, next page).

It will be seen that except one man who was unfit on the vital capacity test, every man was rejected by the fatigue test. Some of these were people about whom the medical officer was in doubt, and had sent them for examination, saying he had to pass them fit, but did not like the look of them, though he could see nothing wrong with them.
I do not suggest that any one test should be taken in examining a candidate, but that they should be used in combination, and the instructions are that they should be used by the assessor for his guidance.

Table II.

<table>
<thead>
<tr>
<th>No.</th>
<th>Initials of name</th>
<th>Age</th>
<th>Time to breath held in seconds</th>
<th>Vital capacity in cubic centimetres</th>
<th>Supplemental air in cubic centimetres</th>
<th>Expiratory force in millimetres Hg.</th>
<th>Time in seconds of sustaining 40 milligrams Hz. by blowing</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>G. E. B. ...</td>
<td>17 11</td>
<td>55</td>
<td>4,200</td>
<td>1,300</td>
<td>80</td>
<td>42</td>
<td>Rejected</td>
</tr>
<tr>
<td>2</td>
<td>W. D. H. ...</td>
<td>17 11</td>
<td>84</td>
<td>4,300</td>
<td>1,800</td>
<td>60</td>
<td>25</td>
<td>Rejected</td>
</tr>
<tr>
<td>3</td>
<td>J. McK. ...</td>
<td>18</td>
<td>53</td>
<td>3,700</td>
<td>1,700</td>
<td>55</td>
<td>32</td>
<td>Rejected</td>
</tr>
<tr>
<td>4</td>
<td>H. G. R. ...</td>
<td>18</td>
<td>66</td>
<td>3,800</td>
<td>1,650</td>
<td>130</td>
<td>30</td>
<td>Rejected</td>
</tr>
<tr>
<td>5</td>
<td>H. B.</td>
<td>17 11</td>
<td>53</td>
<td>2,800</td>
<td>1,000</td>
<td>60</td>
<td>25</td>
<td>Rejected</td>
</tr>
<tr>
<td>6</td>
<td>R. R. S. A ...</td>
<td>18</td>
<td>48</td>
<td>3,600</td>
<td>1,650</td>
<td>70</td>
<td>27</td>
<td>Rejected</td>
</tr>
<tr>
<td>7</td>
<td>W. M.</td>
<td>17 11</td>
<td>44</td>
<td>3,400</td>
<td>1,000</td>
<td>120</td>
<td>35</td>
<td>Rejected</td>
</tr>
<tr>
<td>8</td>
<td>R. C. T. ...</td>
<td>18</td>
<td>85</td>
<td>2,750</td>
<td>900</td>
<td>100</td>
<td>28</td>
<td>Rejected</td>
</tr>
<tr>
<td>9</td>
<td>H. E. S. ...</td>
<td>17 11</td>
<td>71</td>
<td>2,400</td>
<td>1,050</td>
<td>100</td>
<td>50</td>
<td>Unfit by V.C. Standard</td>
</tr>
<tr>
<td>10</td>
<td>F. R.</td>
<td>19</td>
<td>50</td>
<td>3,100</td>
<td>1,000</td>
<td>60</td>
<td>20</td>
<td>Rejected</td>
</tr>
<tr>
<td>11</td>
<td>D. H.</td>
<td>19</td>
<td>63</td>
<td>--</td>
<td>--</td>
<td>60</td>
<td>40</td>
<td>Rejected</td>
</tr>
<tr>
<td>12</td>
<td>C. T. M. H.</td>
<td>19</td>
<td>42</td>
<td>3,800</td>
<td>--</td>
<td>40</td>
<td>33</td>
<td>Rejected</td>
</tr>
<tr>
<td>13</td>
<td>2/Lt. C. H. S.</td>
<td>23</td>
<td>42</td>
<td>4,200</td>
<td>--</td>
<td>60</td>
<td>25</td>
<td>Rejected</td>
</tr>
<tr>
<td>14</td>
<td>W. A. B. ...</td>
<td>18 1 1</td>
<td>64</td>
<td>--</td>
<td>--</td>
<td>60</td>
<td>33</td>
<td>M. O. says fit but does not like him; referred by Assessor</td>
</tr>
<tr>
<td>15</td>
<td>2/Lt. F. W. C.</td>
<td>22</td>
<td>61</td>
<td>4,100</td>
<td>--</td>
<td>100</td>
<td>30</td>
<td>Assessor did not like the look of him</td>
</tr>
<tr>
<td>16</td>
<td>F. C. B. ...</td>
<td>23 2</td>
<td>63</td>
<td>4,300</td>
<td>1,800</td>
<td>60</td>
<td>35</td>
<td>Assessor did not like the look of him</td>
</tr>
<tr>
<td>17</td>
<td>F. R. B. W.</td>
<td>21 6</td>
<td>55</td>
<td>4,100</td>
<td>1,700</td>
<td>80</td>
<td>37</td>
<td>History of migraine referred by Assessor</td>
</tr>
<tr>
<td>18</td>
<td>C. T. Y. ...</td>
<td>18 2</td>
<td>48</td>
<td>3,800</td>
<td>1,700</td>
<td>100</td>
<td>35</td>
<td>History of migraine referred by Assessor</td>
</tr>
</tbody>
</table>

Average ... 58 3,650 1,450 77 32

The results obtained by Lieutenant-Colonel Birley from pilots who had been fighting for several months and were being sent home for a rest, support the results obtained by the U-tube test. These pilots were fairly up to the average standard. On the other hand, cases who had been concussed were below average. One has here, therefore, a valuable test for the effect of crashes.
Lieutenant-Colonel Birley obtained for those found permanently unfit for flying the following results:—

<table>
<thead>
<tr>
<th>Average expiratory force</th>
<th>...</th>
<th>...</th>
<th>...</th>
<th>76 mm. Hg.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sustaining 40 mm. Hg.</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>28 seconds</td>
</tr>
</tbody>
</table>

As I have stated, it is the combination of the tests, however, that is important. The average and minimum standards are given in the following table:—

<table>
<thead>
<tr>
<th>Breath-holding</th>
<th>...</th>
<th>...</th>
<th>Average standard</th>
<th>69 seconds</th>
<th>Minimum standard</th>
<th>45 seconds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital capacity</td>
<td>...</td>
<td>...</td>
<td>3,900 c.c.</td>
<td>...</td>
<td>3,400 c.c.</td>
<td>...</td>
</tr>
<tr>
<td>Expiratory force</td>
<td>...</td>
<td>...</td>
<td>110 mm. Hg.</td>
<td>...</td>
<td>80 mm. Hg.</td>
<td>...</td>
</tr>
<tr>
<td>Fatigue test (U-tube)</td>
<td>...</td>
<td>...</td>
<td>52 seconds</td>
<td>...</td>
<td>40 seconds</td>
<td>...</td>
</tr>
</tbody>
</table>

*Pulse response to exercise—*

| Increase as result of exercise | ... | 12-24 beats per min. | 36 beats per min. |
| Return to normal              | ... | 10-20 seconds        | 30 seconds        |

I believe that every pilot could be overhauled by the breath-holding, expiratory force and U-tube tests and a station graded according to its efficiency. As a matter of fact, this has been done. At a certain fighting station the medical officer found that the average for all the pilots by these tests were:—

<table>
<thead>
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<td>Expiratory force</td>
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He picked out one officer as being badly off colour, a pilot with two hundred and fifty hours' experience. The pilot soon after went for a flight: there was nothing wrong with the machine, but the pilot lost control, crashed, and was killed. His results for three tests just previously were:—

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The medical officer had advised that this officer should not be allowed to fly. This so impressed the commanding officer that the medical officer was asked every week to grade the pilots. It soon became evident that the officers who were picked out by the commanding officer or squadron flight officer for special duty were practically always those graded by the tests as extra fit. It would seem from this that the selection of pilots for special work by these tests, if adopted,
would be of great value. The commanding officer eventually made a rule that if an officer did not come up to the standard of the tests, he must not be placed in charge of a machine. The medical officer then gave him instructions for making himself fit and he was told if he was still unfit by the tests in a fortnight, he would go up for a board, and possibly be found unfit for flying. This was actually done in one or two cases. By this means the efficiency of the station was greatly increased.

I suggest that these tests would also be of value for measuring trench fatigue, industrial fatigue, and fatigue in women workers; also in the grading of people for positions of trust, such as special motor drivers and members of mine rescue teams. With special standards set according to age, they would possibly also be of value to educational authorities in measuring how children were maintaining their physical efficiency.

DISCUSSION.

Sir R. DOUGLAS POWELL, Bt.: The very definite tests which Colonel Flack is able to indicate as the result of his prolonged and able researches will prove of vast importance, not only for the Royal Air Force but in the selection of pilots for the extensive air navigation which must prevail in the near future; for such pilots must, of course, be most searchingly examined before they can be entrusted with the care of the public.

Dr. HENRY HEAD, F.R.S.: At first candidates for the Flying Services had to pass a more or less strict medical examination of the ordinary kind. But with the Military Service Act volunteers greatly increased in number. An attempt was, therefore, made to invent tests which should explore the various functions on which depended the power of flying a machine heavier than air. It was hoped that by these methods of examination it would be possible not only to exclude men physically abnormal but to choose those with a capacity to fly. Meanwhile, Colonel Flack had devised his series of respiratory and cardiovascular tests aimed at eliminating those who would suffer at high altitudes from oxygen want. But he soon discovered that some of these methods revealed men who were suffering from flying-stress. This was particularly the case with the U-tube test. His tests were applied as a routine to the large majority of the medical patients at the Central R.A.F. Hospital, Hampstead. We then found that these methods stood in some relation to other tests, such as balancing on one foot with the eyes closed for fifteen seconds. This was thought to be a test for "muscle sense," but careful examination showed that the patient
might be entirely unable to balance himself in this way, and yet his "muscle-sense" might be perfect. The question of the correlation between tests so different as the U-tube and balancing on one foot is a matter for statistical examination. But before any such research can bear fruit it is necessary that the results obtained must be recorded in terms of the tests themselves. All such terms as "cardio-vascular" or "nervous" instability, "vestibular stability," "muscle-sense" must be avoided in the protocol and the actual method employed must be indicated. I should like to suggest that in the responses to all these tests there are two factors. First, the behaviour of the peripheral sense-organs and of the lower effector mechanism; secondly, a central factor, physiological or mental, which for want of a better name may be called "higher control." This may be disturbed by any condition which lowers the vital activity of the nervous system. The cause may be states as diverse as influenza and fear. All conditions, physiological or psychical, which cause lack of control, lead to want of resolution and even to diminished effectiveness of those automatic actions, dominated by the will. This is a factor apart from the specific appeal of each of these tests, and is, I believe, responsible for failure in such different tasks as those set by the U-tube and balancing on one foot.

Brigadier-General J. G. Hearson: Any method which reduces the medical examination to an exact science which can be recorded in figures capable of direct comparison with similar figures obtained on another date by another medical officer (thus eliminating the "matter of opinion") should be of the greatest value and will be especially welcomed in the Air Force. I know that this is the goal of Colonel Flack's endeavours rather than the perfection or restriction of the actual tests themselves.

Sir Walter Fletcher, F.R.S.: In looking back to the beginning of our attempts to bring physiological help to the practical needs of the Air Board, now the Air Ministry, some memories of that time are perhaps significant. I think that Colonel Flack found, like others working elsewhere, that if there was any reluctance to welcome the establishment and introduction of physiological tests for flying men, it was not on the whole shown by the men themselves or by any others accustomed to the study and use of planes and engines. To these it was a natural idea that the human machine, like other machines, should be subjected to tests and trial performances before final use for particular tasks, and that these tests should be based upon a study of the functions of the machine and its parts, together and in detail. But there did, I think, betray itself at some stages a feeling of suspicion, on the part of medical men, that novel tests by novel apparatus worked out in the laboratory could not replace or do much to supplement the results of what is ordinarily called "clinical examination." In so far as this feeling arose it was due, I fancy, to a vague and unexpressed tradition that the use of physiological apparatus in the daily work of the physician is to be strictly limited, and that
the line is to be drawn at a point in historic time: everything later than the introduction of the stethoscope by Laennec is to be ruled out. I suppose one speaker had something of this kind in his mind just now, when he hoped that physiological tests might come into use "some day even in the consulting room"; but why "even" there? We must all cordially share Colonel Flack's hope that pragmatic tests of this kind, or like those already introduced by Dr. Thomas Lewis for grading military "heart" disorders by test exercises, will make their way in other directions. Putting square men into square holes is really, if we take a large view, a primary aim of all civilization as such, and yet this has never been attained in any age, or nearly approached. We can hardly imagine the results in efficiency and happiness that would come from a scientific, that is a successful, selection and grading of workers of all kinds for their tasks, or again of children for varieties of educational method.

Mr. M. GREENWOOD: I wish to emphasize the importance of some considerations adduced by Dr. Head which have necessarily engaged my attention as a member of the Air Medical Investigation Committee. A long series of researches, with which the name of Professor Spearman will always be associated, has demonstrated not only that ostensibly distinct psychological tests are correlated one with another but that the form of the association finds its readiest explanation on the hypothesis that each test depends not only upon a specific factor but also upon a generic factor, common in greater or less degree to all, and that, further, the importance of the part played by this common general factor is often overwhelming. Hence it follows that, in practice, when we are forced to select a limited number from a variety of possible tests we should give the preference to such as are most highly correlated with the common factor, it being feasible, on certain assumptions which can be scrutinized in each case, to evaluate this correlation although the strict connotation of the term "common general factor" escapes us. The evidence submitted both by Colonel Flack and Dr. Head proves that Flack's tests belong to the class I have mentioned, and the next stage of the inquiry is for combined physiological, psychological and statistical team work.
Section of Epidemiology and State Medicine.

President—Dr. E. W. Goodall.

The Causation and Prevention of Industrial Accidents.¹

By H. M. Vernon, M.D.

(Fellow of Magdalen College, Oxford, and Investigator to the Industrial Fatigue Board.)

(ABSTRACT.)

Industrial accidents occur much more frequently than would be inferred from the published statistics on the subject. The annual reports of the Chief Inspector of Factories and Workshops record all accidents which are sufficiently serious to necessitate absence from work for seven days, and the records show that every year over a thousand workers are killed, and between one and two hundred thousand are injured. However, these figures tell one nothing as to the actual number of accidents incurred. In munition factories I found that minor and unreported accidents occur about thirty times more frequently than the notified accidents, and though it is probable that in most industries the ratio of minor to major accidents is not so great as this, one would be safe in saying that at least 2,000,000 accidents are incurred per annum in this country.

Causation of Accidents.—Information as to the causation of accidents was obtained by examining the accident records in four large munition factories over periods of nine to twenty-five and a half months, and over 50,000 accidents were tabulated.² At a fuse factory employing 9,000 workers, the accidents were tabulated for a three-month period

¹ At a meeting of the Section, held March 11, 1919.
² See Memo. No. 21 of the "Health of Munition Workers Committee," Cd. 9016.
in 1915, when the hours of work were usually twelve a day and seventy-five a week, and for the subsequent two years, when they were ten a day and sixty-four and a half to fifty-four and a half a week. Three-fourths of the accidents were cuts, chiefly of the hands against the sharp lathe tools and drills. The number of cuts incurred was at a minimum in the first hour of the morning spell of work, and increased steadily throughout the morning, as can be seen from the diagram, which records the cuts in each hour, reckoned per 10,000 workers per week. Such an increase has been observed in other industries, and has generally been attributed chiefly to fatigue. This view appears to be erroneous, at any rate as regards the men, for when they were working the twelve-hour day their accidents were no more numerous than when they were working the ten-hour day, and they did not increase more rapidly during the course of the morning spell. In the women, on the other hand, the accidents showed a five-fold increase in the morning spell of the twelve-hour day period, as against a three-fold increase in the ten-hour day period. In the afternoon spell the accidents were 29 per cent. more numerous than in the morning spell, and they were more than twice as numerous in the twelve-hour day period as in the ten-hour day period. Even after allowing for the longer hours worked, the accidents were two and a half times more numerous in the twelve-hour day than in the ten-hour day, and this excess must have been due entirely to fatigue. Moreover, in the twelve-hour day period the women were treated for faintness nine times more frequently than the men, and were given sal-volatile (chiefly as a restorative) twenty-three times more frequently, whereas in the ten-hour day period they were treated for faintness and were given sal-volatile only three times more frequently than the men in each case.

Speed of Production.—The rise of accidents in the morning spell of work is due partly to increasing speed of production. It was found that the output of the workers at the fuse factory (as determined by observations on the electric current required to drive the machinery), increased gradually during the course of the morning spell as the accidents did, and after starting high at the beginning of the afternoon spell it fell away, again in fair correspondence to the accident incidence (cf. diagram). However, the variations of output were very much smaller than those of accidents, but this is only to be expected, as a small improvement of output, implying as it did, a relatively greater quickening in the manipulation of the lathe and other tools, meant a much greater liability to accidents.
Psychical Effects and Alcohol Consumption.—More important than speed of production is the effect of carelessness and inattention. This was shown by the night-shift accidents, which are at a maximum when the workers first come on, and steadily dwindle down till, at about 4 a.m., they are not much more than half their original number (cf. diagram). From this point onwards they keep steady or show a slight rise. That is to say, the incidence of the night-shift accidents is almost the reverse of that observed in the day shift. The reason appears to be that the night-shift workers get up about four hours before work, and they occupy this time in housework, shopping and amusements, and in having substantial meals. Hence they often come on to work in an excited state, but they calm down during the course of the night as they have nothing but breakfast and bed to look forward to. The day shift, on the other hand, are lethargic and depressed when they come on to work in the morning, but they brighten up gradually as they have some tea after they have done about two hours' work, and
they look forward to their mid-day dinner break. That is to say, they tend to get more and more inattentive, and their accidents go up correspondingly. This psychical effect tends to be enhanced by the effects of alcohol consumption. The day shift seldom took any alcohol until their day's work was done, as they could not get alcohol at the factory canteen. The night shift, on the other hand, if they took alcohol at all, did so shortly before coming on to work. Hence any effect of alcohol on accident causation would be greatest during the first part of the night shift and least during the last part. In correspondence with this conclusion, it was found that the men (who are well known to drink considerably more than women) showed a greater excess of accidents in the first spell of work, as compared with those in the subsequent spells, than the women did. Again, it was found that during 1916 and 1917, when the average sobriety of the nation greatly increased because of the increasing restrictions on the sale of alcoholic liquors, both men and women showed a steady diminution in this excess of accidents incurred during the first spell of night-shift work.

In three consecutive six- to eight-month periods the relative number of accidents in the first spell of work, when compared with those in the last spell taken as 1.0, varied as 1.9, 1.5 and 1.4 respectively in the men, and as 1.6, 1.4 and 1.3 in the women. In the day shift there was no evidence of any alcohol effect except when the twelve-hour day was in operation. The workers did not stop work till 8.30 p.m., except on Saturdays, so they had but little opportunity of drinking except at week ends. In correspondence with this fact, the accidents were found to be at a maximum on Monday, and they steadily fell during the course of the week till on Friday they were 32 per cent. less numerous in the men and 27 per cent. less numerous in the women. Then on Saturday they shot up to a fresh maximum. In the ten-hour day period, however, the workers stopped at 6 o'clock every evening, and they appeared to take their alcohol in more regular and moderate quantities, as their accidents showed no week-end excess. They were, in fact, rather more numerous in the middle of the week.

Temperature Effects.—Unsuitable temperature is a very important cause of accidents. The temperature of the fuse factory was registered continuously for six months by means of a thermograph, and it was found that accidents were at a minimum at temperatures of 60°F to 69°F. At 72°F, they were 21 per cent. more numerous than at 67°F., and at 77°F., 30 per cent. more numerous. At 57°F., again, they were 18 per cent. more numerous. In a not very efficiently warmed shell
factory the accidents to women were two and a half times more numerous on such days as the external temperature was at or below freezing point than when it was 48° F. or over, whilst at intermediate temperatures the accidents were intermediate in number. The accidents to men were similarly affected, and were twice as numerous on the very cold days as on the warm ones.

Artificial Illumination.—Defective artificial illumination is well known as a cause of accidents, but the factories investigated were all fairly well lit. Only one class of accidents was found to increase much by night—viz., foreign bodies (as metal or emery) in the eye. Apparently the workers tend to bend more over their work when the lighting is artificial, and it was found that, though accidents in general were 16 per cent. less numerous by night than by day, eye accidents were 30 to 60 per cent. more numerous in the worst-lit factory. At the best-lit factory they showed very little excess.

Accident Prevention.—Accidents can be largely prevented, not only by improving lighting and keeping the factories at a suitable temperature, but by the methods employed in the “safety campaign,” which has been adopted with such enthusiasm in the United States. This consists in the installation of safety devices and of as complete a system of mechanical safeguards as possible. Over twenty museums of safety appliances have been set up in various countries, and one is in course of erection in London. Committees of Safety are formed at the factories, on which both employers and employed are represented. These committees investigate safety devices and their installation, inquire into accidents and means for their prevention, and they post information concerning accidents—often of a striking pictorial character—on bulletin boards placed at the entrance of the workshops. By adopting these methods certain well-known companies have reduced their accidents by as much as 78 and 88 per cent., and in this country the accidents at Port Sunlight, where safety methods were introduced in 1917, were reduced to 43 per cent. of those experienced in 1916.

Other suggestions for accident prevention are obtained by comparing the accident frequency of men and women at different factories. For instance, it was found that, taking cuts as a basis, the women at the fuse factory experienced two to three times more sprains than the men, chiefly sprains of the wrist. These sprains were mainly acquired in pushing home the clamping lever on the lathes. The lever was designed to suit the stronger wrists of men, but it would be quite a
simple matter, by lengthening or otherwise altering it, to render it suitable for women. Again, the women at a big shell factory suffered about eight times more frequently from burns, and four times more frequently from eye accidents, than the women at the fuse factory, though their cuts and sprains corresponded in number. This excess of accidents was due chiefly to the steel turnings from the big shells being much larger and hotter, and more liable to jump out from the object turned, than the aluminium and brass turnings met with in fuse manufacture. But it is self-evident that the hands could easily be protected from burns by wearing gloves, and the eyes by wearing goggles.

The frequency with which septic wounds were treated varied greatly at the different factories; and as a septic wound is much more likely to interfere with work than a fresh wound treated directly it is incurred, it is important that the workers should be persuaded to attend the ambulance room directly they experience an accident, even if it be only a slight one.

DISCUSSION.

Mr. M. Greenwood: I have recently, in collaboration with Mr. Udny Yule, devoted a good deal of attention to another side of the problem of industrial accidents—viz., the personal factor in their aetiology. Mr. Yule and I were impressed by the fact that statistics displaying the number of operatives who, during a definite period of exposure to risk, had sustained 0, 1, 2, 3, &c., accidents, deviated widely from the distribution which would have resulted had the accidents occurred by chance, using the latter word in the conventional acceptation. We conceived that this anomalous distribution might have arisen in one of the following ways: (a) The initial liabilities of the workers might have been equal, but those who by chance sustained a single accident were eo ipso rendered more (or less) liable to sustain a second accident than their colleagues: (b) the initial liabilities of the workers to incur accidents might have been different owing to variations of natural capacity, skill in handling tools, &c. We devised mathematical formulæ representative of the distributions which would arise on each of these hypotheses and tested them upon a long series of carefully compiled data. The result was to show that the second hypothesis more satisfactorily (indeed very satisfactorily) accounted for the observed distributions, and the practical conclusion emerges that an important method of reducing the number of industrial accidents is the elimination of susceptible workers from processes attended by risk of accident. Carefully compiled ambulance room records would enable this elimination to be carried out without difficulty.
Dr. COLLIS: The prevention of industrial accidents has been too much neglected by the medical profession, who have here failed to see that this is a department of preventive surgery just as the prevention of diseases is preventive medicine. The matter has been left rather to engineers, under the impression that industrial accidents are only to be prevented by making machinery safe with guards and other appliances. Dr. Vernon has shown that other influences are at least equally important. A recent estimation made in America suggests that about 80 per cent. of all industrial accidents are entirely under the control of the worker, and that mechanical means for preventing accidents can only affect the remaining 20 per cent. Here then is a large and interesting field for preventive surgery, a field which concerns the individuality of the workers.

Dr. VERNON (in reply): I think that Mr. Greenwood's results are in agreement with one's natural expectations. One knows of certain people who are inherently clumsy and who frequently meet with accidents and misadventures. Conversely, we know of careful and cautious individuals who almost invariably manage to avoid them.
Section of Epidemiology and State Medicine.

President—Dr. E. W. Goodall.

Sydenham as an Epidemiologist.

By M. Greenwood.

Although the name of Sydenham is as well known as that of any medical man recorded in history, it would be an affectation to pretend that his writings are now studied by any considerable number of those who read without intending themselves to add to the bulk of printed matter; to the generality of medical men, the "English Hippocrates" is hardly more than the shadow of a name; at most his works perform the service rendered by Virgil and Horace to the eighteenth century parliamentarian, that of furnishing more or less appropriate quotations.

That Sydenham performs even this office is evidence that he was a man of mark; quotation, even second-hand quotation, after two hundred years is a tribute seldom paid to mediocrity. Still it is opportune to inquire whether we may derive other benefits from the labours of the illustrious defunct than a choice of mottoes; whether there is a body of doctrine, first formulated by Sydenham, or bearing the impress of his personality, still capable of either guiding our researches or warning us what we should avoid. It is peculiarly opportune to initiate such a discussion now because I hope that, at the next meeting, our President will submit results well adapted to test certain of Sydenham's theories.

The object of this communication is, therefore, to make readily available such of Sydenham's ideas as are concerned with our special branch of medicine. I am precluded from a discussion of his strictly clinical work and I do not propose to embark upon a voyage into the

1 At a meeting of the Section, held May 9, 1919.
seas of history and pure scholarship which are navigated by our colleagues
in another Section. Even thus limited, the object of the paper is more
difficult of attainment than might have been anticipated; it is, indeed,
hard to come to close quarters with the mind of Sydenham.

Sydenham is parted from us not merely by the gulf of two centuries,
but one personality must and—unless we choose to make use of an
instrument which is no longer an obligatory item in the educational kit
of the student—two personalities may intervene between us and him.
The whole of Sydenham's published works are in Latin, but the evidence
was strong enough to convince a distinguished former member of this
Section, Payne, and is probably sufficient to convince most of us that
the Latin is not Sydenham's. When Latham executed the Sydenham
Society's translation of the works, this opinion touching the authorship
of the Latin version was not universally accepted, but there was enough
in its favour to leave doubts in the mind of Latham which may have
led him to take more liberties with the text than might have been
thought respectful in the case of, say, Celsus.

It would ill become one whose Latin verse exercises at school
sounded depths of infamy which are painful to recall, to venture a
judgment upon nice points of classical scholarship. That Latham's
book frequently seems to avoid complexities of construction in which I
have entangled myself is no proof that Latham was not a faithful
translator; a great scholar once said that if he failed to comprehend a
passage he translated it literally. We all remember, too, how Scaliger
reconstructed a lost Greek original from a Latin paraphrase; so that a
free English version might be more faithful to the thought of Sydenham
than the Latin text itself or its literal translation. But it is always
possible that Latham was not a Scaliger; this possibility and a natural
obstinacy have induced me to struggle with the Latin text, but I
think very seriously over any interpretation of my own which differs
substantially from that of Latham.

Now when we grapple with the Latin text we are not, or, to speak
more modestly, I am not, so much delighted with its elegance as sundry
authorities assured us would be the case. There are two ways of writing
Latin if one does not happen to be a Roman. One is to treat it as a
living language and not to boggle at expressions which would have
given serious annoyance to Cicero or even to Apuleius; this was the
method of Erasmus, of Bacon and indeed of most scientific men writing
between the Renaisance and the nineteenth century; it is, or was,
the custom of the Pathology Section of this Society.
The second method is to play the sedulous ape to the classical stylists and forms a branch of literary art of which one of the most remarkable exponents, in prose, was Marc Antoine Muret. The practitioners of this art were not men of science. The translator of Sydenham's works was a literary artist whose model was Cicero and it is certainly easier to reproduce the prolixity of Tully than any other features of his style.

One can certainly choose passages from Sydenham, such as the eulogy of Hippocrates in the preface to the Medical Observations, which are really impressive, but the general effect of the long and involved sentences is wearisome and calls to mind Macaulay's gibe at the Man of Arpinum himself—viz., that a parallel to many of his speeches would be afforded by a barrister prosecuting a rioter who remarked that the occasion was a good one for instructing the younger auditors in the public gallery in the true meaning of the Bill of Rights.

But there is a further barrier against sympathetic understanding, that of temperament. A hasty reading of Sydenham would lead one to suppose that a large proportion of the inhabitants of London who died between 1660 and 1675 were the victims of our author's incompetent colleagues, and one is surprised that our forefathers left a window of the College of Physicians unbroken, but not at all surprised that Sydenham never presided over that illustrious corporation. After a few score pages, this portrayal of the one just man in a generation of charlatans is nearly as annoying as in the leading case of Aristides, and it is quite impossible not to notice with malicious satisfaction that the one just man who derided subtle speculations and traditional doctrines himself often adopted as axioms some of the most conspicuously feeble relics of the Galenical tradition, and, as a pathologist, differed very little from Dr. Caius or Chaucer's Doctor of Physik.

I mention these obstacles because they must be surmounted by anyone who desires to become acquainted with the thought of Sydenham, and it is no real service to the cause of epidemiology to pretend that its classics are more inviting than they really are. A medical Gilbert Murray might attract a few more readers to the works of Sydenham, but the Medical Observations will never secure admission, as did Harvey's treatise, to Everyman's Library. Sydenham's general doctrine of epidemics is contained in a small compass—viz., in the two concluding sections of the First Chapter of Book I of the Medical Observations and in the following chapter. It will be advantageous to give here a reasonably strict translation of the operative passages:—

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JU—1a
"As to the acute diseases (which I now purpose to discuss), some are generated by a secret and inexplicable alteration of the air infecting the bodies of mankind and are only conditioned by a peculiar crisis of the blood and humours in so far as the occult atmospheric influence shall have impressed the crisis upon the said bodies. Such diseases only ravage while the hidden constitution of the atmosphere endures and appear at no other time; they are termed epidemics.

"Other acute diseases being conditioned by some individual anomaly and not the resultants of general causes do not simultaneously assail many persons. Acute diseases of this kind, with certain exceptions to be considered when we discuss this genus, are independent of years and seasons. Such acute diseases I term Intercurrents or Sporadics; because they may accompany any Epidemics. I shall deal first with the Epidemics, giving pride of place to their general history.

"Nothing, I suppose, has more astonished the student of medicine than the protean character of epidemic diseases, not so much as referable to differences of weather as to differences of epidemic constitution in different years upon which they depend.

"This very evident diversity of the diseases in question is seen both in respect of symptomatology and of necessary treatment. From which it plainly appears that diseases which to the inattentive observer may seem congruous both in respect of external features and symptoms will be found by a judicious scrutiny to differ as chalk from cheese. I do not indeed know whether a sedulous examination (for properly carrying this out the brief space of man's life were hardly sufficient) might not teach us that certain Epidemics succeed one another in a series, forming as it were a circle, or alternatively that owing to an occult diathesis of the atmosphere and a mysterious succession they attack us indiscriminately. This only, fortified by a multitude of exact observations, I do confidently hold, that the aforesaid species of disease, in particular the continued fevers, may vary so enormously that you may kill your patient at the end of the year by the method which cured sufferers at the beginning of it; and so when by good luck I have hit upon the proper treatment of a fever of this kind, I can, under God's providence, nearly always reach my end by aiming at the same goal, respect being had to the age and temperament of the patient and such like matters; until that particular species becoming extinct and another emerging, I am again puzzled how to help my patients; and it is only by dint of the greatest caution and using all my wits that I can avoid, indeed, I cannot always avoid, risking the lives of one or two of my clients, until continuous observation leads at length to comprehension and I again steadily and intrepidly advance to conquest.

"Now although I have attended as diligently as possible to the more apparent diversities of atmospheric conditions in different years with the object of reaching an explanation of the vicissitudes of epidemics, I am fain to confess that I have made no progress at all; I very clearly perceive that years perfectly agreeing in their obvious meteorological characters may be utterly dis-
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Separate in the matter of diseases and conversely. This is the state of the case. There are different constitutions of years due to some hidden inexplicable change in the bowels of the earth when the air is contaminated by such effluvia as predispose and determine the bodies of men towards some disease or other: heat or cold, dryness or moisture, are not the causes: this state of affairs endures so long as the particular constitution is dominant and then yields its place to another. Each of these constitutions is characterized by a particular kind of fever, not seen under other circumstances, and fevers of this class I term Stationaries.

"In addition there are, if I may use the term, particular cases of the same year in which, owing to obvious meteorological factors, fevers following the general constitution of the year are more or less epidemical or arise earlier or later. Above all, however, such fevers as occur indifferently in all years (and I therefore term Intercurrents) trace their origin to some obvious character of the atmosphere; such are pleurisy, angina and similar diseases, which prevail when warmth suddenly replaces long and intense cold. Perhaps the sensible qualities of the atmosphere may operate in the production of such fevers as appear in any constitution, although this they cannot do for fevers special to any one constitution; but I must admit that the aforesaid atmospheric characters do more or less predispose our bodies to the generation of this or that Epidemic, which may also be said of an error in the six Non-Naturals." ¹

The above is a full translation of the first six sections, which are fundamental; it will be enough to summarize the remainder.

Some epidemics are perfectly uniform in their evolution and these ought to form the basis of an epidemiological history; others are variable from constitution to constitution, while, worse still, the same disease may vary within the same constitution (sections 7 to 9).

All epidemics fall into two groups, the vernal and the autumnal, but this division is to be taken broadly for "atmospheric conditions may play into the hands of some epidemic helping it to ravage prematurely, while on the other hand a want of correspondence may lead to the epidemic getting to work on the predisposed subject only late" (section 10).

Vernal epidemics ending by midsummer are Measles and the spring Tertians; Small-pox and Plague begin and end later. Cholera morbus is autumnal and beginning in August is over within a month, dysentery, quartans and autumnal tertians last longer. "As to fevers specially, they have always been named from symptoms. But since nearly every constitution, over and above the fevers it breeds, is prone to set going some one or more of the famous epidemics, such as plague, small-pox, dysentery, &c., I do not see why these fevers should not rather take

¹ Air, Meat and Drink, Motion and Rest, Sleep and Wakefulness, Mental Emotions; the Secreta and Retenta.—Greenhill's note.
their names from the constitution, since this favours the production of some one of these diseases at the time of their emergence,¹ than from any particular alteration of the blood or symptom which may be found equally well in specifically distinct fevers" (section 13).

In the next section, it is pointed out that the form of intermittents may be assimilated to that of continued fevers, and that the true state of the case is only revealed as the influence of the prevailing constitution wanes.

The fifteenth and sixteenth sections I give in full:—

"This is to be carefully noticed, that when several of these diseases infest the same year, one of them gets the mastery, as it increases the rest decrease, when it wanes they once more wax, and so they plague humanity in turns as the genius of the year and the sensible qualities of the atmosphere give one or other assistance. But it will be found that whatever disease rages most furiously and causes most havoc round about the autumnal equinox, gives its name to the constitution of the whole year, whatever disease takes the lead then will be found to have the mastery of the others the whole year through, assimilating the qualities of contemporaneous epidemics to its own so far as their nature will allow. For instance, when small-pox is prevalent, the fever appearing sporadically during the whole year partakes of that inflammatory type which begets variola. Each disease originates in a similar way, there is a great affinity between the characteristic symptoms of each (excepting in the variolous eruption and its accessories) as plainly appears in the tendency of both towards spontaneous diaphoresis and towards salivation."

I omit the second illustration, that of dysentery.

The eighteenth section is as follows:—

"Lastly, I must observe that when any constitution generates various species of epidemics these are essentially different from those bearing the same name but produced by another constitution. However, many distinct species may occur in one and the same constitution, they have in common one general factor, that derivative from the peculiar diathesis of the atmosphere; hence however distinct they may be in type and specific form, the constitution common to all so moulds the substance of each that the principal symptoms, other than those referable to the particular type of evacuation, are alike in all; the several maladies in this too, agreeing that they all at nearly the same time increase and remit their severity. It is further to be remarked that in the years in which these various species prevail at the same time, they all agree in the manner of onset and initial symptoms."

¹ The words are "Quatenus horum morborum alterutri producendo favet codem illo tempore quo comparebant." Latham translates: "That being determined by the particular disease which they usher in." I suggest that the version in the text conveys better what I take to be the thought—viz., not that the fever determines the constitution but the constitution the fever.
I do not think I have omitted any important statement of Sydenham's general doctrine, and now attempt to ascertain its meaning. There is no difficulty about the theory of intercurrents; it is that these diseases (amongst which he included scarlet fever) are independent of whatever influences generate an epidemic constitution. But it is not—to me at least—clear what the relation is between an "epidemic" and a "stationary fever." Are we to understand that the characteristic product of an epidemic constitution is its stationary fever, that it is in the type of stationary fever that one constitution differs from another? If so, is the stationary fever an independent phenomenon or an epiphenomenon of the "epidemic"? Is the "epidemic" determined by laws other than those describing the genesis of the "constitution"? It seems to me that Sydenham was not consistent, that he sometimes regards the stationary fever as an epiphenomenon of all "epidemics" occurring during a constitution, sometimes as a separate entity.

Take, for instance, the epidemic constitution of 1665-66. Here we have plague proper, and in addition a pestilential fever, which fever is held to be at bottom a pleurisy upon which the pestilential character has been grafted by, presumably, the constitution. Now we may suppose that this suggested the question whether plague proper were not in the same case—viz., a special graft (i.e., the stationary fever of the constitution) upon some intercurrent stock—and that if so the pestilential fever had just as good a right to be called plague as the plague. Anyhow, Sydenham does ask this question, and contents himself with the shy answer—"Febri autem illi, de qua modo loquebar, an pestis appellatio attribui mereatur, non ausim definito pronuntiare." So far as appears, we can suppose that the doctrine of constitutions is expressible in the symbolic form, thus: during any epoch the manifestations of acute diseases are the resultants of two components \((a + C), (b + C), (c + C), \ldots\), \(a, b, c, \ldots\), denoting the individual (intercurrent) bases, and \(C\) the common addition (stationary) contributed by the prevailing constitution. But this formula will not cover such a description as that of the two variolous constitutions, the regular of 1667 \textit{et seq.}, the irregular of 1674. As regards the former period, we are told that a continued fever prevailed which, being engendered by the variolous constitution was identical with small-pox save in the matter of the exanthem, and any signs and symptoms essentially dependent upon the exanthem. There is no suggestion that this variolous fever was an intercurrent (like pleurisy) upon which a constitutional stationary was grafted, but rather that it and small-pox were sisters, both daughters of
the constitution. Hence the representation would be that only one component is involved in the manifestation of acute diseases, which are $a, b, c, \&c.$, under one constitution, $a', b', c', \&c.$, under another. But then for the years 1670-72, we are told of an anomalous small-pox due to a constitution tending to the hotter and more inflammatory type, which constitution also engendered bilious colic; this time the account is more consistent with the two component interpretations.

I do not profess to have a settled opinion, but it seems to me that on the whole the more probable view is that Sydenham inclined to the first-mentioned interpretation of the doctrine of constitutions—viz., that of superposition upon distinct types of a common external form or group of allied forms. In this way, we can understand his separate characterization of the secular evolution of diseases, so that the complete epidemiological theory of Sydenham is, as I have elsewhere suggested,\(^1\) to be expressed in the following terms: "(1) There is a process of secular or long period modification in virtue of which a specific type becomes dominant in a particular epoch and then gradually or suddenly gives place to a rival. (2) There is another set of factors producing short period oscillations in the epidemicity of a given disease leading to the phenomena of seasonal prevalence. (3) We can connect these two trains of ideas by the conception of an epidemic constitution in virtue of which certain types of epidemic or certain features of morbidity tend to prevail at a given time to the exclusion of other types or symptoms. We may say that the amplitude of the first kind of wave is measured in centuries, that of the second in months, and that of the last in years."

We are now concerned merely with the doctrine of an epidemic constitution, so that I may expand my interpretation of it a little in order to give scope for criticism.

The complete morbid process of an epidemic disease is made up of two parts; the first specific (the $a, b, c, \&c.$, of the formula), subject to secular modification and also to short period oscillations—i.e., it is a doubly periodic function of the time. The second part is generic, common to all species of epidemic diseases and a function of some terrestrial conditions included under the term "Epidemic Constitution." Consequently, the total effect upon the community at any instant of small-pox is the resultant of two terms $f(t) + A.F(t)$, the contemporaneous effect of scarlet fever is $f'(t) + B.F(t)$, and similarly for all other diseases. The actual effect of the second member of each

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\(^1\) Seventeenth International Congress of Medicine, Lond., 1913, Sect. XVIII, Discussion No. 3.
expression is different (A and B are supposed to be constants), but for each disease one and the same functional term $F(t)$, is included; this is the product of the epidemic constitution.

I am alive to the dangers of anachronism in rationalizing (perhaps in the unfavourable Freudian sense of that word) an historical doctrine. Perhaps, what I have just written would have been repudiated by Sydenham with as much energy as the barons of Runnymede might have shown in rejecting the interpretation of the Great Charter that we learned at school. Indeed, one can quote numerous passages which are consistent with the belief that Sydenham did not realize the existence of any morbid specificity, in fact, regarded all acute diseases as variants of a fundamental inflammatory process (his pathology of inflammation was traditional in the worst sense, the mere journalese of Galenism). Still, the interpretation I have put upon the doctrine of an epidemic constitution at least divests it of the mysticism which became a cloak in the eighteenth century, and was not wholly in tatters at Montpellier in more recent times. The theory as stated may be false, but is neither trivial nor incomprehensible. Let us then discuss it.

So far as certain diseases are concerned, nobody doubts that there have been changes of clinical type. scarlet fever is the classical instance. This fact, however, makes neither for nor against the theory of an epidemic constitution. If, in my nomenclature, $a$ is the specific element of scarlet fever and C the constitutional epiphenomenon, the type of scarlet fever at epochs 1 and 2 might differ, because at 1 we have $a + C_1$ and at 2, $a + C_2$ agreeably to the doctrine of constitutions; but we should also have a disparity if $a$ itself varied, without invoking a C at all. The only crucial test would be to find C attached to $a, b, c, \&c.$, during one epoch, and $C'$ attached to the same $a, b, c, \&c.$, during another epoch. In other words, we should have specifically distinct maladies converging towards common clinical forms, the point of convergence varying with time. Not much light is shed on this by modern mortality or morbidity statistics, for the trend of official classification is towards primary divisions; we shall not discover from them if pulmonary complications of typhoid are more frequent in one year than another. A judgment on this point can only be pronounced by those whose clinical experience of zymotics extends over many years and whose memories or notes are trustworthy.

It is to be regretted that the eighteenth century annalists, like Huxham, have had few successors. Painful though the confession must be to a statistician, I do confess that tabular matter is not a perfect substitute for faithful annals.
The most plausible evidence in support of the theory of epidemic constitutions is that provided by the bacteriologists, who are not usually supposed to entertain much reverence for Sydenham. In the late epidemics of influenza a clinical convergence towards a grave type of pneumonia has been uniform, but the primary bacteriological findings have been multiform. If we are to define primary disease in bacteriological terms, there have been whole alphabets of $a$'s, $b$'s, $c$'s, &c., but a common C has been involved, the product of what Sydenham might have termed the peripneumonic constitution of the years 1918-19. Perhaps he would even have sketched a phrenetic constitution for some areas in 1915-16, and have similarly interpreted the simultaneous and successive occurrence of typhoid, paratyphoid A and paratyphoid B, admitting a specific factor of each (the vera causa of the bacteriologist) but asserting that this was complemented by a constitutional element.

I am not equally sure that our distinguished colleague Dr. Hamer is a whole-hearted supporter of Sydenham. Dr. Hamer, I think, would at any rate reject my formula, and not agree that the gross epidemiological phenomenon which we call "influenza" is $a + C$, $b + C$, $d + C$, &c., the complex termed "epidemic cerebro-spinal meningitis," $a + C'$, $b + C'$, $c + C'$, &c., the small letters being specific factors, the large letters generic or epidemic constitutional factors. He seems inclined to postulate a single mutating vera causa, and this does not appear to me to be the doctrine of Sydenham, although, as stated above. I recognize the essential ambiguity of much Sydenham advanced.

Returning to my attempted rationalization of Sydenham's doctrine, I suggest that it is really a very important one. If it is true that some common factor C is the divinity which shapes the ends rough hewn by different specific factors, the vera causa of the bacteriologists, we perceive that a very large part of the campaign against epidemic diseases must be directed against the general factor; because, by hypothesis, the tracking down and elimination (when practicable, as by specific immunization) of $a$, $b$, $c$, will still leave it open to the general factor C to complement $c$'s, $d$'s, and $e$'s, whole alphabets of small letters, yet undeciphered.

Of course if we adopt Sydenham's own theory of the epidemic constitutional factor C, this is a mere counsel of despair—much as was that form of the doctrine which presented itself to the mind of Watt in 1814 as the principle of substitution. But it no longer seems that we should
regard the basis of an epidemic constitution as beyond the compass of human intellect.

We have naturally very little patience in this Section with the appeal to some strange god hight Sanitation popularly identified with well-flushed water-closets, and invariably invoked by the opponents of serum therapy and experimental medicine. But we are, perhaps, a little too complaisant towards the advocates of a millennium attained by specific conquests of the alphabets of small letters.

Professor Gay, a distinguished American bacteriologist, in his recent monograph on "Typhoid Fever," begins his discussion by remarking that "it will, we believe, be evident that all significant information concerning the nature of the disease itself and its method of dissemination, as well as all effective means that have been devised to prevent and cure it, have depended on laboratory data and are based on the recognition of the single bacterial causative factor."¹

But within a few pages, our bacteriological colleague is to be found remarking that "all subsequent information, extended and elaborate as it is, has not supplantled the explanation of Budd (respecting the transmission of typhoid), and has for practical purposes added little to it."² Yet Budd lived and worked some time ago. The moral is that a general consideration of the facts of human life, the slow changes of normal social evolution, the drastic changes enforced by recent events may be of as much importance as an intense scrutiny of the specific *vera causa*, the *a, b, c* of the bacteriologist.

In making these remarks, I am, I fear, digressing beyond the limits marked out for my contribution to the discussion. I do not wish to assume the rôle, rather a foolish one, of an advocate for seeking salvation by a return to the standpoint of the seventeenth century. But I am concerned to show that the teaching of Sydenham, greatly over-praised by a few, ignored by most, contains some ideas which, true or false, may still usefully be discussed.

DISCUSSION.

The President: Sydenham is rightly looked upon as the founder of the modern science of epidemiology. It is doubtful, however, whether he had more than an inkling of what his work was destined to be the forerunner. In our time the epidemiologist stretches his view far beyond the "history of epidemic diseases." If we take those words in the sense in which Sydenham uses them—viz., a description of these diseases, it will have nothing to do with their cure, so far as the treatment of individual cases is concerned. The only cure he thinks of is the preventive, and I doubt whether the epidemiologist, pure and simple, cares even about that. His history of epidemics is not the history of the cases of which epidemics are made up, but of the epidemics themselves, from one season to another, from one year to another, from one age to another. And as he finds that in different seasons, years and ages, there are epidemics of different diseases and that the same epidemic diseases behave differently, so he tries to find out the causes of these differences, that is to say he is led on to study their aetiology, a thing with which Sydenham would have nothing to do, because of its difficulty. But Sydenham is the founder of our modern science only by accident. What was the object of his work? He has answered this question himself very plainly. The first edition of the treatise which deals with the subject with which we are now concerned was published in 1666 under the title of "Methodus curandi febris." In the preface to that edition he tells us that that method is founded upon his own observations, and that in the belief that his method would be beneficial to his fellow sufferers, he makes it common property. Be it noted, however, that his observations were made solely with the object of establishing a mode of cure. By the time the third edition appeared in 1676 the work and its title had both expanded, the latter to "Observationes medicæ circa morborum acutorum historiam et curationem." A history of these diseases is now added to an account of the method of curing them. In the dedicatory epistle to this edition, addressed to Dr. John Mapletoft, Sydenham writes as follows—(I quote from Latham's translation of Greenhill's edition, and shall use this translation throughout)—"The more I observed the facts of this science [medicine] with an attentive eye, and the more I studied them with due and proper diligence, the more I became confirmed in the opinion which I have held up to the present time, viz., that the art of medicine was to be properly learned only from its practice and its exercise; and that, in all probability, he would be the best skilled in the detection of the true and genuine indications of treatment who had the most diligently and the most accurately attended to the natural phenomena of disease. . . . I directed myself to the close observation of fevers and . . . at length hit upon a mode of curing them." He then goes on to say that since publishing the first edition he had observed several new forms of fever and that his experience had been
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much extended. Lastly, in section I, chap. ii, par. 3, of the "Medical Observations," he distinctly states that his close investigation of epidemics was undertaken solely for the purpose of finding out the proper method of curing individual cases.

Sydenham's observation was made chiefly upon the epidemics of the fifteen years 1661 to 1676. As a result of his studies he noticed several facts of a strictly epidemiological nature. First, he noticed the seasonal variations of diseases (I, ii, 2); secondly, he was aware that the characters of epidemics of the same disease varied in different years (II, ii, 2); thirdly, he recognized that infection might be spread not only by infected persons but by infected things, *fomites* (II, ii, 4); fourthly, he observed that after an epidemic of any disease, cases of that disease might still continue to crop up sporadically; fifthly, he mentions the prevalence of flies in connexion with certain abdominal diseases; sixthly, he pointed out that epidemics came to an end not because the food upon which they fed was exhausted, but for some other reason, an atmospheric condition; and lastly, he noticed that if cases of a given epidemic disease began to appear at an earlier time than was to be expected from an experience of former epidemics of that disease, then the epidemic was likely to be an extensive and severe one. But besides these epidemiological facts, Sydenham gives us descriptions of the epidemic diseases prevalent in his time. From a careful perusal of his writings I have no doubt that he saw small-pox, chicken-pox, measles, scarlet-fever, plague, typhus, relapsing fever, enteric fever, dysentery, autumnal diarrhoea, ague, influenza, cerebro-spinal fever, and epidemic encephalitis. His descriptions of some of these diseases is absolutely clear, especially of small-pox and plague. Indeed I doubt whether any writer of modern times has added a single important observation to his admirable and detailed description of natural small-pox. But of many of the others his accounts are vague. It is greatly to be pitied that he deliberately refrained from swelling out his pages with histories of particular cases and confined himself to general expression because he feared that repetition would be vain and wearisome. And here I may add that Sydenham's habit of thought led him to differentiate between diseases—to separate out new diseases. Of late years there have been some among us whose idea seems to have been to merge what most of us believe to be distinct affections into one and the same disease. To such I would recommend Sydenham's observation that although these epidemic disorders "may to a certain degree, both in their external characters and in several symptoms common to many of them, appear to the careless observer to coincide, they are, in reality, if we attend closely, of wholly different characters, as little like one another as coins and counters."

But it is by his doctrine of epidemic constitutions that Sydenham is most remembered, at any rate by the epidemiologist. I suppose that there can be little doubt that he derived the idea from Hippocrates. Yet there is a very great difference between the ideas on this subject as put forward by these two great physicians. So far as I understand them, there is nothing mysterious about the constitutions of Hippocrates. They were frankly determined by
weather conditions. With Sydenham, however, so far as an epidemic constitution is concerned, the weather plays quite a secondary part. It is by no means clear, however, exactly what Sydenham meant by the phrase, “An epidemic constitution,” for there is more than one epidemic constitution: Sydenham found five in fifteen consecutive years, and saw no reason why there should not be more—an epidemic constitution is a constitution of the atmosphere. It is not the same, however, as the manifest qualities of the atmosphere, wind, rain, &c. There is something mysterious, ineradicable, occult, obscure, peculiar, particular, and unknown about it. It is due to or connected with mysterious changes in and vapour from the bowels of the earth on the one hand or, on the other, skiey influences, such as the conjunctions of the heavenly bodies. But these mysterious unknowns, looming in the background, were the cause, each at a different time, of various epidemic diseases; yet by no means of all epidemic diseases. The five constitutions to which I have alluded above were those of the intermittent fevers, plague, small-pox, dysentery, and the comatose fever. In subsequent writings Sydenham added the constitution of the deputatory fever and the new continued fever. Now these were the most striking and prevalent epidemic diseases of his time, and according to him they were caused each by a special epidemic constitution of the atmosphere. The diseases which were thus caused were termed by Sydenham stationary fevers; other epidemic diseases, due to other and usually (at any rate in Sydenham's opinion) more or less obvious causes, such as the manifest, sensible and appreciable qualities of the air, wind, rain, heat and cold, &c., were called intercurrent or sporadic. Scarlet fever (and what Sydenham describes as scarlet fever was, in my opinion, that disease and not rubella)—scarlet fever was one of them. But then it was not an affection which bulked largely in the epidemics of that period. If it had, doubtless we should have heard of a scarlatinal constitution. While an epidemic constitution was the direct cause only of the stationary fever, it could and did influence the intercurrents. That was shown by the fact that there was a certain set of symptoms, such as natural sweating and salivation, which were common to all the concurrent diseases. In fact, Sydenham's hypothesis on this subject led him to differentiate certain fevers which, though they were not stationary fevers, yet bore an impress (without, however, the specific marks) of certain stationary fevers prevalent at the time. I refer to the variolous, the dysenteric, the pestilential, and the morbillous fevers. Personally I am of the opinion that in his desire to discover something new and in his belief in the influence of the epidemic constitution, he was led astray, and described unexistent “fourth” diseases. While the stationary fevers are due each to its own particular epidemic constitution of the atmosphere, to such an extent that without the constitution you cannot have the disease as an epidemic, yet these fevers are influenced also by the manifest qualities of the air; hence seasonal prevalences and variations in character of epidemics. Further, the manifest qualities of the air may exert some influence on the epidemic constitution, to the extent of admitting or excluding a stationary fever. Such influence is, however, only temporary.
I have stated above that Sydenham writes of the epidemic constitution of
the atmosphere as being of a most mysterious nature. He does so frequently.
Yet I am led to believe that at the back of his mind there was an almost
materialistic idea of it. In one passage he writes of "the particles which are
mixed with the atmosphere, which war against health and which determine
the epidemic constitution." And there is one curious passage as follows:
"We must consider, not that any particular diathesis is to be assumed from
the atmosphere itself (nulla ejusmodi diathesis in ipso aere supponi debere),
by which, whilst we have one epidemic propagated in one place, we may have
another, wholly different, elsewhere and at no great distance—if such were the
case every movement of wind would (as it sometimes does) invariably diffuse
a constitution—but that each particular tract of the atmosphere is filled with
the effluvia of some mineral fermentation, that these contaminate the air
through which they pass by their particles, that these particles are differently
destructive to different animals, and that they propagate diseases appropriate
to the different affections of the soil, until the whole mine of such subter-
ranean vapours be exhausted." 1 The interesting admission here to my mind is
the one that an epidemic constitution can be shifted by the wind.

Sydenham appears to be very inconsistent at times. He frequently tells
us that the epidemic constitution is the cause of certain epidemic diseases, and
leads his reader to consider that it is the sole cause. What, then, are we to
think when we come across the following passage: "Much as these two forms
[i.e., the stationary and the intercurrent diseases] may differ from one another
in respect to their origin in atmospheric influences, they agree in respect to
several of their external and predisposing causes. Laying contagion out of the
account, which occasionally gives origin to the stationary form of fever, and
laying also out of the account intemperance, which is the mother of both forms,
the commonest external and evident cause of most fevers is either premature
change of dress or exposure to cold after exercise." In another passage
Sydenham writes of the "coughs helping the constitution in producing the
fever." It is clear from these and other passages which could be quoted that
the epidemic constitution was not the sole cause of an epidemic disease.
There were exciting and adjuvant causes. There can be no doubt, indeed, that
he had misgivings as to the efficiency of the epidemic constitution as a cause
of epidemics. Plague was a disease which greatly puzzled him from this point
of view. He expresses grave suspicion that the mere atmospheric constitution,
however much λοιμώδης was by no means sufficient, in and of itself, to originate
plague. "Either the disease itself must continue to survive in some secret
quarter, or else either from some fomes or from the introduction from pesti-
lential localities of an infected person, it must have become extruded." But
he goes on to invoke the favourable atmospheric diathesis to explain the
outbreak of an epidemic. Now it is clear from his explanation of the result of
the action of the Grand Duke Ferdinand II, when the Duke effectually stopped
the plague from invading Tuscany by cutting off its communication with the

1 Observ. Med., V, iv, 3.
surrounding districts, that in Sydenham's opinion even epidemic constitution could be very local. It seems to me that Sydenham is not very clear on the question of causes very largely because he is not really interested in investigating them. Not for him "Felix qui potuit rerum cognoscere causas." He defends himself from blame for not having attempted to pierce the penetratalia, and writes "Etiology is a difficult and perhaps an inexplicable affair, and I chose to keep my hands clear of it." Moreover, according to him, we need not be troubled by ignorance of causes when we are seeking for cures of diseases: for it is experience and not knowledge of the cause that will prove the right guide.

But in spite of these seeming contradictions, I think we can form a fairly correct idea of what Sydenham really had in his mind in respect of the causation of epidemic diseases. While there were certain obvious causes such as the various states of the weather, contagion, intemperance, ill-advised changes of raiment, and the like, which could give rise to isolated cases, they were not sufficient to account at any rate for any but the less frequent and less severe forms of epidemic (intercurrents). In the causation of the more important epidemics some other factor was necessary. He was quite ignorant what it was. But he was fairly sure that its presence was necessary, for he states that you may have sporadic cases of plague, for instance, lingering perhaps from a previous epidemic, kept alive by some smouldering fumes, but there will be no epidemic in the absence of the pestilential constitution of the atmosphere. At the same time it was not necessarily the sole cause; it was a very important causative factor, overshadowing, but after some time capable of being influenced to a slight degree by the lesser, though more obvious, factors. This unknown factor waxed and waned, like the epidemic it produced: the waxing and waning of the epidemic was indeed the visible sign of the mutability of the constitution. No two or more constitutions could exist together. Certain characteristic symptoms were the manifest evidence of any particular constitution; some of these were confined to the particular stationary fever which was engendered by the particular constitution; others would be found not only in the particular stationary fever but also in the intercurrent diseases prevalent at the time. In answer to Mr. Greenwood's questions I should reply that the characteristic product of an epidemic constitution is its stationary fever, and that it is the type of stationary fever that one constitution differs from another; that the stationary fever is a phenomenon; and that while the epidemic is determined mainly by the laws which govern the genesis of the "constitution," it is also subject to other laws, as also is the constitution.

How far have we advanced in our conceptions of the causation of epidemics since Sydenham's day? Not very far, I fear, as regards what he called the epidemic constitution. I am of the opinion that we must still admit that there is a very important factor, or there are very important factors, still unknown, in the causation of epidemics. My conception of the causation of epidemics is that there are several causes at work, varying in number and
importance for different epidemics and at different times; that an epidemic is the sum of several factors. Of recent years factors unknown to Sydenham have been brought to light. We know more about the influence of the ages of the persons exposed to attack, of their surroundings, of the seasons, of the part played by insects and animals, and so forth. We also have added to our stock of knowledge the whole of the bacteriological evidence. I am not sure, indeed, that there are not some bacteriologists who would not claim that Sydenham’s epidemic constitution has been explained by the germ theory of disease; that the unknown factor he recognized was the micro-organism. I am sure, however, that no epidemiologist will admit that claim. Admitting a micro-organism as a factor, and a very important factor, in the causation of disease, we still are driven in most instances to explaining the causation of the epidemicity of the micro-organism; and in most instances, if not in all, we are very far from having attained that object. Sydenham recognized a few obvious causes of epidemics and epidemic diseases, more especially of the latter. But he was also well aware that other causes, which he believed to be the most important, were still unknown to him, and especially those connected with the more important epidemics. To speak more correctly, all we have done has been to reduce the amount of the contents of this large magazine of unknown factors by withdrawing from it certain factors which we have been able to name, and transferring them to the store of known factors.

Dr. CHARLES SINGER: As a professed historian I have listened with much interest to Mr. Greenwood’s paper because, as it appears to me, he has been doing what we very seldom see done, and he has been using history for its proper purpose. For the last half century it has been generally recognized that ideas, like other organic products, cannot be fully understood until their history is known. Ideas, like species of animals, have their history, and betray their history in their structure. It should be the function of the medical historian to trace that history as a continuous whole, and so to play his part in the illumination of medical ideas. Now the doctrine of epidemics, like other ideas, has had its history; and a history, moreover, for the writing of which we are provided with unusually abundant material. The history of the doctrine of epidemics may be summed up in one sentence, as a struggle between the ideas of miasma and contagion. Among early peoples all evil is contagious: misfortune, ritual, uncleanness, wickedness, and, of course, disease. The point might be illustrated from a thousand passages, and is to be found in the Bible. It is a test of how far Hippocrates had left primæval superstition behind, that he rejects the whole doctrine of contagion and is a believer only in miasma. The struggle of the doctrines of miasma and contagion can be traced through the ages, the doctrine of contagion gradually coming more and more to the fore until about the middle of the sixteenth century. It was three years after modern science had made its stately entry with the work of Copernicus and of Vesalius, the natural historians respectively of the macro-cosm and the microcosm, that—in the year 1546—a work of the highest
epidemiological importance appeared; the "De Contagionibus" of Jerome Fracastor. In this work, for the first time, the doctrine that epidemics were due to the spread of infection by minute parasitic organisms of specific nature, was for the first time scientifically set forth and philosophically maintained. During the century and a half that followed this very striking work the sound views of Fracastor were very widely held. It was a misfortune that the authority of Sydenham, who went back to the old and unexplained view of "epidemic" constitution, prevented the further spread of the theory of Fracastor and caused his views to remain practically forgotten until quite modern times. I feel therefore that, on the whole, Sydenham's influence on epidemiology proper was reactionary. It is rather, I think, in his work as a pure clinician and a describer of the natural history of infectious disease in the individual that his true greatness is to be sought.

Dr. Hamer: This most interesting paper calls for much more thorough study than most of us have presumably so far been able to give to it. As regards the barriers against the "sympathetic understanding" of Sydenham, I have never been troubled as to the "sympathetic," for, like Dr. Payne, I greatly admire the old Puritan rebel, but the "understanding"—well, that is a difficulty. But to come to Mr. Greenwood's most interesting analysis of Sydenham's work, I confess that though I had long recognized, as did the man who went to see Hamlet, that here was an author who made use of a large number of more or less appropriate quotations, it was not until 1915 that I began to entertain a hope of having a first very imperfect inkling as to what Sydenham might perhaps be driving at with his epidemic constitutions. In the course of inquiry in that year into the outbreak of cerebro-spinal fever in London, it transpired that the symptom-complex in question occurred in close relation with those of influenza, bronchitis, and pneumonia, and it was then further found that Sydenham had described the same phenomenon in his account of the new fever of 1685. Then, later, Colonel Dorgan worked out the same problem in military camps, and Dr. Crookshank found a like correlation between influenza and the Heine-Medin symptom-complex. Looking backwards it became clear that, examined year by year, there was abundant evidence in the records of these changes of type in influenza diseases, and it was realized that London was responding in 1915-18 to a "constitution" closely resembling that of nearly two hundred and fifty years ago; this "constitution" was traced year by year, through the outbreak so fully described in New York in 1916 and our own epidemics of 1917 and 1918. According to this conception, epidemic constitution represents something that appertains not to one year but to a series of five, six, or more years; then it dies away almost to the point of disappearance to rise into prominence again at a later time.

These inquiries led to closer study of the time relations generally of the outbreaks of the influenzal group of diseases, and incidentally to further examination of Sydenham's constitutions, and so ultimately to the conclusion (expressed at the meeting held here last October) that Creighton had found
the solution of the puzzle in his formula that there was "something more than accident in the association between epidemics of influenza and epidemics of ague." Examination of Creighton's "History of Epidemics" showed quite clearly, it was submitted, that the remarkable related "agues" which occur in the years round about all the great "posting" epidemics of influenza, throughout the whole of the recorded history, "are nothing more than those very gastro-intestinal, pulmonary, and nervous manifestations which actually constitute, as every epidemiologist realizes, part and parcel of the influenzal prevalences themselves." I have recently gone over the ground again and hope to be able to supply, some day, the detailed evidence that Sydenham's constitutions of the years round about 1661, 1675, 1679, and 1688 were influenzal constitutions.

There was a discussion here on November 13, 1918, in which Sir Arthur Newsholme referred to my "seductive hypothesis," but he clearly inclined to the view that the nexus which binds together the catarrhal group of diseases (including cerebro-spinal fever, encephalitis lethargica, and polio-encephalitis) was merely predisposing (telluric, climatic, &c.) and not an actual causal influence exerted by one and the same infecting agency operating in all of them. I have already submitted to the Section the main argument against this view. Well, "the hearing of these observations lies in the application of them" to the present paper. First as to Sydenham and the bacteriologists. In some coalitions credos do not count so much as other considerations, but in this particular instance the credos are strongly held. There are thus really only two doubts, and Mr. Greenwood is alive to both of them. There is the question as to what the bacteriologists may say, and the further question as to what Sydenham would say. I can only submit that if any timorous epidemiologist could ask him "Alas, my master, how shall we do?" he would tell him to lift up his eyes and behold the horses and chariots of fire, saying, "Fear not, for they that be with us are more than they that be with them." I am much disturbed, however, to find that Mr. Greenwood thinks I am not a true disciple of Sydenham. I accept, of course, all Mr. Greenwood says about me, but is he right in hinting that Sydenham appears to rule out a mutating vera causa? I sometimes wonder whether in the fullness of time, when epidemiologists, bacteriologists, protozoologists, and statisticians have worked out in complete detail all the available knowledge concerning the various roles in epidemic disease assumed, on the one hand, by ultravisible viruses mutating within the cycles of their multiannual or seasonal periodicities, and assumed on the other hand by the varying reactions exhibited by communities more or less immunized or sensitized; and when the whole thing has been expressed in the proper differential equations, and these have been triumphantly solved by the Brownlees, Greenwoods, Rosses, and Yules of the period, whether something approaching prodigiously close to Sydenham's conception of epidemic constitutions will not be the result. The people of that advanced era will be able to exclaim with Ecclesiasticus, "Let us praise famous men and our fathers that begat us," and perhaps will also say with Ecclesiastes, "There is nothing new under the sun."
Dr. G. C. Peachey: Before offering a biographical and bibliographical note, I desire to record my dissent from the assertion just made by Dr. Singer that “the history of medicine is a history of ideas and that biography is only of value in so far as it bears on ideas”: indeed, what I have to say goes some way towards disproving his statement. In my recent sketch of the life of John Pechey, Licentiate of the College of Physicians (1654-1718), I drew attention to the fact, not previously recognized, that besides being the author of “The Whole Works of Sydenham,” of which eleven editions were published between 1696 and 1740, and which has hitherto been regarded as the earliest English translation of Sydenham, he had previously published, in 1686 the first part, and in 1688 the second and third parts of a work entitled “Collections of Acute Diseases,” which consist entirely of extracts from Sydenham’s writings. That this work was unknown to Latham is plain from a statement in his preface, in which he says that “the previous version of Pechey represents in point of style and language neither the English of Sydenham’s time nor that of our own” (he is here referring to Pechey’s “Whole Works of Sydenham,” the first edition of which appeared in 1696); and he adds later that he realizes the difficulty of reconstituting Sydenham’s words in what may be supposed to have been the form in which they would have originally appeared (had they appeared in English at all) during the lifetime of the author. But Sydenham did not die till December 29, 1689, three years after the appearance of the first part of Pechey’s “Collections,” and, as I have shown in my memoir, there is some reason to suppose that this latter publication was known to Sydenham himself. That this was unrecognized by Latham, and indeed by all Sydenham’s biographers, is due primarily to the extreme rarity of Pechey’s “Collections,” only two copies of which have come to my knowledge after exhaustive inquiries in Great Britain, France and America (one of these being in the British Museum and the other, which wants Part I, in the Bodleian): and secondarily to the fact that the title-page of Pechey’s “Collections,” in five parts, dated 1691, does not state that the work consists of a translation of extracts from Sydenham’s writings. No copy of the original title of the first part, dated 1686, is forthcoming; but this is to be found in the Michaelmas Term Catalogue, No 24, December 13, 1686, in which the ascription to Sydenham is duly recorded. The practical value of my note lies in the suggestion that students of Sydenham may look upon Pechey’s translations, which Sir Norman Moore has described as “vigorou and idiomatic,” as contemporary with, and probably known to, Sydenham himself; for the text of the “Collections” corresponds almost verbatim with that of Pechey’s “Whole Works of Sydenham” (1696), which latter remained unaltered in all the subsequent editions to 1740. The subject is discussed at length in my paper entitled “The Two John Peacheys, Seventeenth Century Physicians: Their Lives and Times” (Janus, 1918, xxi, 121), and the results of my further study throw some new light upon the still vexed question of Sydenham’s Latinity.
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Mr. M. Greenwood (in reply): In our endeavours to master the secret of Sydenham’s doctrine, we need the help of our historical colleagues and the scholarly contributions to the discussion made by Dr. Singer and Dr. Peachey were very welcome. I regret never having studied the early translation of John Peachey whose contentious life has been so charmingly told by his namesake.

Dr. Goodall’s weighty observations have added what was necessary to my string of quotations. No doubt it is Utopian to expect that any medical student or even any newly qualified medical man will peruse the Proceedings of our Section; but were any such eccentric youth to escape from the prison house of his curriculum, I believe that a study of this afternoon’s debate would be of service to him. There are not unimportant aspects of preventive medicine hidden from the writers of elementary text-books and not fully revealed even to the medical correspondent of The Times.

I am not sure that the chasm which separates me from Dr. Hamer is a very deep one, but I am afraid there is a chasm. Perhaps it is addiction to those algebraical methods at which Dr. Hamer (although himself under the gravest suspicion of being at least a carrier of the mathematical virus) pokes gentle fun, which urges me to attempt—no doubt with small success—to reduce any proposed epidemiological theory to terms capable of precise discussion. I cannot be satisfied with merely praising famous men and our fathers that begat us; these lyrical strains always make me doubt with Charles Fox whether anyone could be as wise as Thurlow looked. Dr. Hamer, as I think, still shrinks from a close scrutiny of his, or of Sydenham’s, doctrine of an epidemic constitution. Does he believe that cerebro-spinal fever, encephalitis lethargica, poliomyelitis and influenza are mutants of one underlying cause, or does he believe that some common factor modifies all diseases prevalent during a particular epoch, long or short—confers upon fundamentally disparate diseases a common clinical and epidemiological type? If my interpretation and Dr. Goodall’s interpretation of Sydenham’s doctrine is correct, does Dr. Hamer regard influenza as the stationary fever of 1918-19 and cerebro-spinal fever as the stationary of 1915? I do not think this is a mere verbal dispute.

What separates us epidemiologists from the bacteriologists is, as Dr. Goodall remarked, that we do not think the problem of disease as a mass phenomenon so simple a one even in theory as the bacteriologist would have us think it. The bacteriologist—or, at least, his popular exponent—holds that all diseases can be attacked and conquered seriatim by specific measures; isolate the organism, “stamp it out” or procure an artificial immunity and that disease is “conquered.” But if my interpretation of Sydenham’s constitutions is correct, all that can be achieved in this way is either to reduce the number of stocks upon which a stationary fever may be grafted or to reduce the number of stationary fevers, leaving the stocks upon which the remainder may be grafted untouched. Naturam expellas furca, tamen usque recurret, hence Watt’s theory of substitution, and Sydenham’s constitutions.

JU—16
So the problem of preventive medicine becomes wider and we are led to ask whether what is wrong with the unhealthy may not be not infection with this or that germ but—unhealthiness, even as the chief trouble of the poor is not addiction to public houses but just plain poverty. But if Dr. Hamer’s mutating _vera causa_ is the secret, why, the future of the bacteriologist is a roseate one, the meningococcus, Pfeiffer’s bacillus, the parabacilli and even the filter-passers may be impostors; but the bacteriologists, _Hamer duce et auspice Hamer_, can advance with confidence: one day a single blow will place in their power the _vera causa_ of at least five diseases and the stamping out should be vigorous indeed!
Section of Epidemiology and State Medicine.

President—Dr. E. W. Goodall.

Periodicities of Epidemics of Measles in the Large Towns of Great Britain and Ireland.

By J. Brownlee, M.D., D.Sc.

(Director of Statistics, Medical Research Committee.)


The subject of periodicity of measles may I think best be approached by a general study of the figures relating to the deaths from that disease in London since the introduction of registration. For this purpose the number of deaths in each quarter-year have been graphed in a diagram (Diagram I). Each line of graph refers to $24\frac{1}{2}$ years. The beginning of each decade is marked by a line and the accompanying date. The reason for this particular distribution into epochs of $24\frac{1}{2}$ years depends on the fact that for each of these epochs the epidemic wave has closely reproduced itself. The fourth graph in the diagram represents the average of the sum of the upper three graphs. It shows obviously the same characters as each of the component parts. In the next section the chief epidemic period of measles in London will be shown to be 97 weeks in length or very nearly $1\frac{1}{2}$ years. On this hypothesis the dates on which epidemics are due are indicated in the diagram by circular dots. It will be noted that the epidemics apparently occur for considerable periods at the expected times but at other times they fail to make their appearance in the manner that might be expected. Yet the disappearance is only temporary and the re-appearance of the epidemic wave of 97 weeks in the expected place is observed to

At a meeting of the Section, held February 14, 1919.
have recurred three times. It must therefore be taken that the phenomena have some permanence and that the recurrence is not due to chance alone. The irregularity observed in this case is similar to that which has brought the analyses of some disease statistics into

**Diagram I.**

The three upper sections in this diagram show the number of deaths from measles in London in each quarter-year from 1840 to 1912. The total length of each section is 24\frac{1}{4} years. For purposes of reference the beginning of each decade is indicated. The circular black dots below the diagram are placed at intervals of 97 weeks to illustrate the main periodicity of the measles epidemics. In the fourth section of the diagram the three sections above have been added together and averaged. It will be noticed that for three-fourths of this section the circular black dots coincide absolutely with the situation of the epidemics. In the fifth section of the diagram the phenomena are illustrated which would be observed if two epidemics causing an equal number of deaths, of 97 and 87 weeks' periods respectively, were present in a large city.
disrepute. In the absence of long series of statistics permanence is difficult to demonstrate. What has held for a considerable time has seemed, in a number of cases, apparently to hold no longer. Change in the nature of a phenomenon leads much more readily to doubt, than permanence of limited duration to belief. In the case of London, however, a series of statistics of sufficient length exists to demonstrate that the disappearance of a phenomenon may be apparent only and not permanent. What then is the explanation of this permanence? I think it comparatively simple. It is that an epidemic is due chiefly to the properties of the organism causing it, and that the periodicity of epidemics which occur at regular intervals depends for the most part on the life-history of the organism. Many biologists and statisticians doubt this at present, but a step in the proof of the accuracy of this opinion is, I think, given by the phenomena under consideration. In a large city like London, the organisms which produce epidemics of measles belong not to one strain but to several, each of these strains possessing different properties. The property alone at present considered is that which determines the periodicity of the epidemic. Grant that one strain of an organism is capable of producing an attack of the disease which confers a certain degree of immunity against another strain, and grant that its life history on the average is the same, then the phenomena which follow will be of the nature of those observed. To illustrate this the fifth line of the graph in the diagram has been constructed. It shows what would happen if two epidemics occasioning an equal number of deaths occurred periodically, the one epidemic having a periodicity of 97 weeks, as is the case with the chief London epidemic, and the second a periodicity of 87 weeks. These epidemics are supposed to be at their maxima simultaneously at the point marked $\times$ in the diagram. With the first recurrence, the maxima of these epidemics will be separated by an interval of 10 weeks, while by the time the fifth or sixth recurrences are reached the maximum of the one period will coincide with the minimum of the other. A dead level of endemicity at this period is the phenomenon requiring explanation. As, however, time goes on the epidemics will again have their maxima simultaneously and epidemic outbursts will be associated with intervals free from the disease. Comparing the last graph with the combined graph of the $24\frac{1}{2}$ years' periods for the city of London the similarity is obvious. The etiology of the epidemic curve for London, when analysed, is not however quite so simple, but I think that the theory outlined roughly corresponds with the facts.
(II) Epidemics of Measles in London, 1840-1912.
Special Investigation.

In this part of the investigation the weekly numbers of deaths occurring from measles in London between 1840 and 1912 have been used. As the population of London doubled itself during the years in question some correction is necessary and as the great bulk of the deaths from measles occur under five years of age the deaths have been corrected on the theory that the population at these ages is stationary. The method of analysis applied to the statistics is fully explained in Appendix I. It consists essentially in writing out the figures in successive rows of gradually increasing length and summing the columns. If in the result the sums of the columns are all nearly equal there is no period in the neighbourhood. If, however, the sums gradually increase to a maximum and then decrease a period exists in that region. The height of the swing is measured by a quantity defined as the amplitude. When rough work is being done this is measured for most practical purposes by taking the heights of the maximum and of the minimum above the base line and dividing the difference of these by the sum. To avoid the introduction of the personal equation, however, the amplitudes in all the cases discussed in this paper have been calculated mathematically by a method described in Appendix IV.

The results of the investigation are graphed in Diagram II. Examination of this diagram shows that fairly large amplitudes exist in a number of situations. Taking for convenience 1,000 as the measure of the maximum amplitude, amplitudes of 200 occur at periods of six months and of one year, 161 at 87 weeks, 180 at 89 1/2 weeks, 394 at 97 weeks, 160 at 105 1/2 weeks, 212 at 109 1/2 weeks, and 205 at 114 weeks. Much the greatest amplitude is that at 97 weeks and this must be regarded as the main epidemic period of measles in the city of London. It is to be noted that this value corresponds exactly with what has been seen to exist in Diagram I. The main period thus could be easily found by direct observation, but the other periods discovered by the method of research employed, with the exception of the yearly period could, even if suspected, hardly be measured.

The question of the permanence of these periods immediately arises. With regard to that of the 97 weeks this admits of no doubt, but the others are not so definitely in evidence. This is seen from the
figures given in Table I. In this table the amplitudes of the periods have been calculated not for the whole period of 72 years but for four equal portions of this period, each consisting of eighteen years. The 87 weeks' period is seen to be most prominent in the last 36 years. The 89½ weeks' period in the first and third; the 109½ weeks' period is most marked in the first and fourth epochs, while the 114 weeks' period is specially important in the fourth. The conclusion, therefore, seems to follow that in London there are a number of epidemics of measles with different periods, these epidemics assuming greater or lesser importance. Of these the chief is the epidemic of 97 weeks' period which remains the dominant epidemic from 1840 to the present day.

Diagram II

In this diagram the amplitudes found when the figures relating to the number of deaths from measles in London from 1840 to 1912 are added together in rows varying in length from 25 to 160 weeks are graphed. It will be seen that the greatest amplitude corresponds to a period of 97 weeks, and that lesser amplitudes occur at 87, 89½, 105½, 109½, and 114 weeks. Annual and semi-annual periods are also observed.
TABLE I.—SHOWING THE AMPLITUDES IN GROUPS OF YEARS AND FOR THE WHOLE EPOCH FOR CERTAIN DIFFERENT PERIODS IN THE EPIDEMICS OF MEASLES IN LONDON, 1840-1912.

<table>
<thead>
<tr>
<th>Group of years</th>
<th>Number of weeks in period</th>
</tr>
</thead>
<tbody>
<tr>
<td>(I) 1840-1856</td>
<td>110 97 331 270</td>
</tr>
<tr>
<td>(II) 1856-1874</td>
<td>152 35 552 70</td>
</tr>
<tr>
<td>(III) 1874-1893</td>
<td>303 300 416 229</td>
</tr>
<tr>
<td>(IV) 1893-1912</td>
<td>310 180 464 325</td>
</tr>
<tr>
<td>Whole Epoch</td>
<td>161 167 417 212</td>
</tr>
</tbody>
</table>

Such are the main facts revealed by the data covered by registration. Prior to 1840 the only figures are those contained in the Bills of Mortality of London. These have been thoroughly investigated from 1703 to 1828. Owing to the inadequacy of the data the results of that analysis are not of great value. They furnish, however, sufficient evidence to deduce one very important negative conclusion—namely, that between the years 1755 and 1828 no epidemic with a period near to that of 97 weeks existed in the city of London. Between 1828 and 1838 the statistics in the Bills of Mortality are imperfect, but complete the evidence that the first epidemic of the present cycle was that which occurred in London in the year 1846. Since its introduction this organism has been the one which has caused the chief fatality from measles in the city.

(III) DISTRIBUTION OF MEASLES IN LONDON, 1890-1915.

The results given in the previous section refer to the periodicity of measles in London as a whole. It is clearly equally necessary to ascertain if the epidemics having different periodicities exist uniformly throughout the whole city, or if they are to any extent local phenomena. This part of the investigation has been limited to the twenty-six years, 1890-1915, and has been carried out, first, for the five main districts, north, south, east, west, and central; and, secondly, for the individual sub-registration districts of the West and South. What happens is well illustrated in the accompanying diagrams (Diagrams III and IV) in which the distribution of the epidemics of 87 weeks' and 97 weeks' periods is shown for the main districts of the city. It is evident that the epidemic with the 87 weeks’ period exists only south of the Thames. The amplitude found for this epidemic in this locality is about 500, contrasting with the 160 observed when the whole city was investigated.
On the other hand, the epidemic with the period of 97 weeks is well marked in all the districts of London, but specially so in the west, where the amplitude is extraordinarily large—namely, 913. In the north and south districts the amplitude of the epidemic is slightly over 500, while in the central and east districts it is about 330. The $109 \frac{1}{2}$ weeks epidemic is best seen in the west, where the amplitude is 570; in the north it is 390; in the central 500, while in the

![Diagram III](image)

**Diagram III.**

In this diagram the results of adding together the weekly deaths from measles in each district of London, 1890-1914, in rows of 87 weeks are graphed. It will be observed that very small amplitudes exist in the North, West, Central, East, but a very marked amplitude in the South.
south and east it is respectively 280 and 170. The epidemic with a period of 114 weeks is most marked in the central where the amplitude is 440, in the north it is 309, in the west 240, in the south 220, and in the east 160.

The method of the periodogram, however, throws light on one very essential point—namely, the method in which epidemics arise. Referring again to Diagram IV, it can be observed that there is little or no evidence that the epidemic having a period of 97 weeks spreads

In this diagram the results of adding together the same figures as in Diagram III in periods of 97 weeks are shown. It will be observed that the amplitude is the greatest in the West but well marked in all the other districts.
from one district to another. If anything may be said it is that the epidemic in the western district has its maximum two to three weeks later than in the north or the south, but as the incubation period of measles ranges from ten to fourteen days this is much too narrow a margin to allow of extensive spread: in addition the amplitude of the wave in the west is greater than that observed anywhere else. When this matter is examined in greater detail in the different sub-registration districts of the west and of the south no evidence is found of spread from any individual centre, the maximum time occurring at a constant or nearly constant date nearly in all districts in spite of the different concentration of the population and of the different social conditions. This simultaneous rise of an epidemic throughout the city must be held to be a very remarkable phenomenon. When, however, the manner in which the epidemic of 87 weeks' period spreads is examined, it is found that the seat of this epidemic is in St. Saviour's parish; that it spreads thence to St. Olave's and Camberwell, and from these to Lambeth, Wandsworth and Greenwich. Further, with the epidemic of 97 weeks' period both the maximum of the epidemic in point of time and the death-rate due to the epidemic is very nearly the same in all the sub-registration districts. Yet, with regard to the epidemic of 87 weeks' period, not only is the maximum of the epidemic later in each district as the distance increases from St. Saviour's, but the proportion of children who die becomes progressively less. This might be ascribed to loss of virulence on the part of the organism, but I prefer to interpret it as due to loss of infectivity, the smaller number of deaths being the result of a smaller number of persons being infected. It is to be remembered that the method of examination eliminates the probability that this is a chance effect. If it were only observed in one single epidemic it might well be due to the circumstance that the children in the periphery were protected by a recent attack of measles. But the figures give the average of nearly twenty epidemics, so that such an explanation does not seem to be feasible.

Before passing from this part of the subject, it is, perhaps, well to consider for a moment the appearance which may be expected when two epidemics intermix. This is shown for the south of London in Diagrams V and VI. The epidemics are arranged in the first diagram in 87 weeks' period, and in the second in 97 weeks' period, each unit compartment of the graph corresponding to the number of deaths in four weeks. A straight vertical line is drawn in both diagrams, through the point where the maximum of the average
epidemic is found by calculation, and in addition on each diagram a small black circle indicates the maximum of the epidemic of the other period. It will be noticed that most of the epidemics, though not all, fall on one or other system, and that in a considerable number of cases even where the epidemics occur within six or seven months of one another both epidemic systems are well marked. It

Diagram V.

In this diagram the result of the admixture of the two nearly equal epidemics in the south of London is shown: the length of each section is 87 weeks and the vertical lines indicate the points where the average maximum is found. The black circular dots indicate the places where epidemics of 97 weeks are to be placed.
is, however, to be noted that for two or three periods in succession an epidemic wave may not be manifest. Further, a very large epidemic in one system may apparently determine the absence of an epidemic in the other system at the expected time. These diagrams will repay some study. They represent typically the phenomena frequently found.

*Diagram VI.*

In this diagram the same phenomena are again exhibited with the difference that the sections are of 97 weeks' length and the position of the 87 weeks' period indicated by a solid black circle.
(IV) MEASLES IN THE LARGE TOWNS OF THE BRITISH ISLES.

Having thus determined that epidemics of measles may recur at various periods—87, 97, or 114 weeks—it is necessary to examine how far this is the rule in other places. In the year 1870 the Registrar-General of England began to publish weekly the statistics of the number of deaths from the different zymotic diseases for the chief towns of the country. From this list the following selection has been made: Newcastle, Sheffield, Liverpool, Manchester, Salford, Bristol, and Birmingham, in England; Glasgow and Edinburgh in Scotland, and Dublin in Ireland. These towns illustrate a great variety of climatic and industrial conditions.

Before discussing, however, the results of this investigation it is necessary to ascertain whether the two years period of measles so commonly believed in exists or does not exist. It seems commonest in towns of less than the greatest size, and is found at present or recently in Paisley, Dundee and Aberdeen. Even in such towns its presence, however, is not constant, and as in such towns it is unlikely that more than one strain of organism can maintain itself for long, it is in these that some of the phenomena may perhaps be best observed. Take the case of Paisley, for instance, a town of about 80,000 inhabitants. A complete résumé of the facts is given in the following table (Table II). Each row contains the number of deaths in each quarter-year for two successive years, beginning with 1856 and ending with 1917. It will be observed that from 1856 to 1873 inclusive the epidemics occur at intervals of two years with very little variation in date, the maximum number of deaths sometimes occurring in the last quarter of the even years, sometimes in the first quarter of the odd. With 1874, however, a change takes place and epidemics of measles no longer occur at intervals of two years but approximately at intervals of about 88 weeks, more accurate determination of the period not being possible on account of the short length of the statistics. This phenomenon lasted twelve years, when it was replaced for the following twelve years by an epidemic which had a period of greater length than two solar years, but which, like the last, did not persist long enough to permit of its period being accurately determined. With 1898 a biennial epidemic again appears. In this case, however, the maximum occurs in the first quarter of the even years in place of the first quarter of the odd. This epidemic has persisted from that date to
the present. During the latter part of this term in addition an epidemic of two years period in which the maximum varies from the second to the third quarter of the odd years has been running concurrently. The evidence of the figures seems to show then that there are at present two epidemics, one having its maximum in winter and the other its maximum in the second or third quarter of the year. The figures for Paisley thus furnish some evidence which suggests that different strains of the measles organism have been the cause of the epidemics of measles in Paisley, and that some of these strains have maintained themselves for twenty years. This example is of very considerable importance.

Table II.—Showing the Number of Deaths from Measles in each Quarter-year in Paisley, 1856—1917.

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Having thus illustrated the variations that may be observed in a simple example, the more complex cases of the larger towns may be submitted to the question. The periodogram has been constructed
for each of these in the same manner as for London (Diagram II). These periodograms are shown in Diagram VII. They fall into two groups: the first Edinburgh, Glasgow, Birmingham, Bristol and Sheffield; the second Liverpool, Manchester, and Salford. Beginning first with Edinburgh it will be seen that the values of the amplitudes increase gradually to a maximum with an amplitude of nearly 700, corresponding to a period of 98 weeks. There is also a further maximum of nearly the same amplitude corresponding to a period of

![Diagram VII](image)

**Diagram VII.**

In this diagram the amplitudes found on adding the figures together of rows of different lengths of 80 to 120 weeks is shown for ten large towns. This diagram corresponds to Diagram II for London and is fully discussed in the text.
110 weeks. Between these maxima the amplitude falls to zero, and at
the period of 104 weeks is not more than 12. There is thus no evidence
of a two-yearly period in Edinburgh. The manner in which the
amplitudes increase and decrease as 98 weeks appear and pass,
correspond closely to what would be expected mathematically from
the range of the statistics (Appendix V). The same holds for the
period of 110 weeks. There is therefore no evidence of any epidemic
period in Edinburgh of any other value than 98 weeks and 110 weeks.
These periods may thus be considered to provide an explanation of the
main phenomena observed in that city. Passing to Glasgow it will be
observed that the same class of phenomena are observed. There is a
maximum at 98 weeks and another maximum at 109 weeks. Between
these, however, the curve does not fall so closely to the base line, and
when the proper calculations are made there is found to be a residue at
about 104 weeks, having an amplitude of 200. This indicates that
there should be a small amount of measles in Glasgow due to the
organism which determines a periodicity of two years. Another
phenomenon observed in Glasgow but not in Edinburgh is the exist-
ence of a moderate amplitude with a period of 1.89 weeks, one of the
periods already found in London. In close correspondence with these
the graph of Birmingham takes its place. The main periods are 99
and 109 weeks respectively, while there is a subsidiary period of 86
weeks. The general form of the graph suggests also that there is also
a small period in the neighbourhood of two solar years. The very close
resemblance of Birmingham and Glasgow suggests that causes funda-
mentally similar must be in action in both cities. Bristol must also be
placed in this group. In this case the main periods are 98½ and 116
weeks. There is no period of 104 weeks, but a small amplitude exists
in the neighbourhood of 106 weeks. The period of 116 weeks is again
in proximity to that of 114 weeks already found to exist in London. It
would seem that in towns of the size indicated two or three epidemics
may become established and account for the great majority of the cases
of measles observed.

Falling in a certain measure in line with this are the phenomena
observed in Sheffield. Here the main period is about 96 weeks, the
amplitude in this case being very large—namely, 800. A less important
but quite considerable amplitude is found at 104 weeks, or two solar
years, while a moderate amplitude is seen at about 115½ weeks. In all
the above cases the periods again resemble those found in London, with
the exception that in Sheffield a two years’ period is well marked.
Liverpool, Manchester and Salford furnish a second group of towns showing different features. Liverpool exemplifies a new strain of organism. The main amplitude is at 92 weeks, and amounts to over 600. There is also a subsidiary period of 100 weeks, with an amplitude of 400. When Salford is examined we find the main period is 90 weeks, which has an amplitude of 600. This period is a little shorter than that observed in Liverpool. There is a second period with an amplitude of 400 at 104 weeks, or two years, and at 114 weeks or 115 weeks an amplitude in the neighbourhood of 300. The periodogram for Manchester suggests that this city has taken epidemics largely at random from its neighbours. Thus between 89 and 93 weeks there is a fairly high tableland, which may be explained as due to a number of epidemics of mixed periods, such as that of Salford—namely, 90 weeks, and the longer period of Liverpool—namely, 92 weeks. These epidemics may have been introduced temporarily, or have established themselves more or less permanently. At 104 weeks an amplitude is also seen to exist, but again the graph is too flat to be explained by the existence of this period alone. At 114 weeks, however, where the amplitude is barely 300, there is evidence of a small periodic wave.

The two towns left for discussion are Newcastle and Dublin. In Newcastle the graph indicates with some degree of certainty a period in the region of 83 weeks. The part of the graph referring to periods of greater length shows a continual high level of amplitude. This is due largely to the presence of one epidemic of a very much greater size than the rest. Dublin, in like manner, gives no evidence of the permanence of any regular phenomenon.

It seems important after the mathematical method has been carried out to return to the actual data and examine these in the light of the discoveries. Two examples have been graphed, Liverpool and Glasgow (Diagrams VIII and IX). In the former the chief epidemic is of 92 weeks period, the second is 100 weeks. The graph for Liverpool is shown in the diagram in sections of 92 weeks. Each vertical compartment of the graph corresponds to the number of deaths in four weeks. The epoch at which the maximum of the epidemic is expected is shown by a vertical line. It will be noticed that the epidemic falls closely to the expected date in most cases. Some variation from the date occurs (criticized in the comment on the diagram), and might of course be expected, but when the vagaries of animal organisms are taken into account this deviation does not seem excessive, though the uniformity
is not insisted on. It will be noticed also that there have been a number of epidemics in Liverpool which do not come in the scheme of 92 weeks. This of course is what is to be looked for in a large town like Liverpool, which is a great centre of immigration and of emigration.

Diagram VIII.

In this diagram the deaths from measles in Liverpool from 1870 to 1912 are shown. Each section corresponds to 92 weeks. A vertical line indicates where the maximum of the epidemic is theoretically expected.

Glasgow has been chosen as an example of a mixed period. The statistics are shown in biennial periods from 1872 to 1917. In this case each compartment corresponds to the calendar month. During
the first part of this period, from 1870 to 1883-85, a two-yearly epidemic is well marked. It is to be noted that this epidemic is a summer epidemic. From 1882 upwards the 109 weeks' period is well seen. It seems probable that at this date the epidemic, with a period of 109 weeks, entered the city: in any case it was from this period it

Diagram IX

This diagram shows the course of measles in Glasgow from 1872 to 1917. During the first years the epidemics are apparently biennial and occur in the spring of the years. This phenomenon persists possibly up to 1892. In more recent years the two main periods are 98 and 109 weeks. The epochs at which these epidemics are expected are shown respectively by black circles and by crosses. The 109 weeks' epidemic is not present before 1882, and the 98 weeks' epidemic was probably introduced about 1886.
### Table III.—Showing the Amplitudes in the Periodogram Analysis of the Epidemics of Measles in the Great Towns of England, Scotland, and Ireland.

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became specially important. About 1888 the 98 weeks' epidemic seems to have appeared. In the last few years the two epidemics have separated, as these periods require, and since 1914 yearly epidemics have occurred in the city of Glasgow, each apparently corresponding to one special strain of infection.

(V) Discussion of the Results.

The meaning of the phenomena found now falls to be considered. The common explanation of the periodicity of epidemics of children's diseases is that the susceptible children take the disease in sufficient numbers to limit the further spread. The epidemic thus dies out to recur when a further sufficient number of susceptible children have accumulated. This is quite a feasible theory and certainly explains periodicity of epidemics. The forms of epidemic curve which arise on this hypothesis are not unlike those actually found, the differences being no more than might be expected between a mathematical formula based on an hypothesis and the natural conditions to which the hypothesis is only an approximation. This explanation, however, must fail if epidemics of different periods can be shown to exist in the same town at the same time, and I think this has been shown. In London, which on account of its size might be assumed deserving of special treatment, the existence of periods of different length have been demonstrated. In Edinburgh, Glasgow and Birmingham also it has been shown that epidemics with periods in the neighbourhood of 98 weeks and 110 weeks intermix. The same epidemicity even applies to districts of London. In the West End of London we have almost a replica of what occurs in Glasgow, Birmingham and Edinburgh. The main period there is 97 weeks, the secondary period 109\(\frac{1}{2}\) weeks. In the South of London one period is that of 97 weeks, but almost equally prominent is that of 87 weeks. The whole evidence, therefore, seems to point to some condition in the organism which produces the disease as the potent cause of the difference rather than to the number of susceptible children. Compare the paramaecium which in natural conditions divides asexually for several hundred times and then dies out unless conjugation takes place. The resting stage following conjugation persists for some time.

There is, however, one point of great importance which must be considered. If an epidemic begin in a definite locality and spread from that locality, and if there is no loss of infectivity on the part of the
organism, it is demonstrable that a similar proportion of the population should be attacked in each zone as the epidemic spreads outward. On the other hand if the organism lose the power of infecting with the lapse of time, in each additional zone invaded the proportion of susceptible persons infected should become smaller and smaller. Of course this might not be true for any one epidemic, as in many parts of the area invaded the population might be more or less insusceptible because of recent attack of the disease, but when an average of twenty outbreaks has been taken this effect should be eliminated, the number of times the invading organism comes into contact with an insusceptible population being balanced by the number of times which it meets one more susceptible than the average. The method of spread of epidemic on the average should thus give some indication regarding the laws which determine the course of the phenomenon. Now with regard to London, the clearest facts refer to the 87 weeks', the 97 weeks', and the 109½ weeks' periods. The 97 weeks' period starts at the same time all over the city and there is no evidence of any special centre. The infection seems generalized. With regard to the 87 weeks' epidemic, however, the case is different. This seems to start in St. Saviour's parish and to spread thence to Camberwell, Lambeth, &c. In this epidemic the rate of spread can be definitely measured. The maximum occurs later and later as the distance from the centre is increased and the percentage of children infected is also easily observed to fall as the time increases. With regard to the 109½ weeks' period epidemic the facts are similar though not quite so definite. This seems to show that for at least two strains of organism the epidemic ceases because the organism has lost its power of infecting. It may be inferred then that an epidemic ceases because the organism varies in its potency to cause infection. A cycle of epidemics now coinciding and now differing in their maxima can thus be explained. Some kind of life cycle exists in the infecting organism. In this life cycle high powers of infecting are attained probably after a resting stage: a period of activity follows and gives place to a period of rest; the average length of the cycle is determined by the strain of the organism.

(VI) SEASONAL DISTRIBUTION OF MEASLES.

The question of the seasonal periodicity of measles demands some notice. The general results are shown in Diagram X. It will be noticed that for every district practically there are two seasons of the
year in which deaths occur, one having its maximum in the winter months, December and January, and the other having its maximum about the months of April or May. The significance of these phenomena is very difficult to explain. It can be proved at once, however, that there is nothing essential in the double periodicity. For instance,

in Paris during the thirty years 1881-1910, there is no evidence of special winter fatality due to measles. The maximum number of deaths occurs about the beginning of the month of May, and the
minimum in October. During the thirty years in question the maximum in individual epidemics has varied from March to July, occurring some years earlier and some years later. The number of deaths in May is more than twice the number of deaths in September, and nearly twice that in January. It is thus apparent that a seasonal prevalence having only one maximum may persist over very considerable periods of time. Looking back to the diagram which illustrates the course of epidemics of measles in Glasgow since 1870 (Diagram IX), it can be observed that in the earlier years an epidemic having its maximum in May occurred biennially with little indication of any seasonal effect of the winter months. Taking these two facts into consideration it would seem that there is in these cases a predilection for the late spring or the early summer. If the periodicity of the epidemics is rigidly one or two solar years, no other appearance would be possible once it was established. In this connexion it may be recalled that in Paisley between 1856 and 1873 the maximum death-rate was in the alternate months of January. In Dundee from 1874 to 1911 it was in the month of February. In both these towns it may be said that practically nothing but the biennial epidemic was present. In Paisley, further, during the last twenty years there have apparently been two biennial epidemics, one with its maximum in January and one with its maximum in the second or third quarters of the year.

In spite however of the fact that two-yearly epidemics can hardly be said to exist in any of the remaining towns examined, winter and spring maxima are characteristically independent of the length of the epidemic periods. In their similarity of behaviour it is only necessary to mention London, Birmingham, and Liverpool. In the latter town where the main periodicity is 92 weeks, both phenomena are specially marked. It would seem to be a fact that where a maximum of an epidemic is due between August and October little or no result follows. In these months it would therefore appear that something specially hostile to the production of a measles epidemic exists. For the present no further dogmatic statement can be made.

Returning to London it may be said in the first place that the seasonal distribution again seems independent of the period of the epidemic. As we have seen, in the northern district the chief epidemics are those with periods of 97 and 109 weeks; in the western district that with a period of 97 weeks is much the most important, and to a less extent that of 109 weeks; in the southern district the epidemics with periods of 87 and 97 weeks are equally important; yet there is no real
difference in the seasonal distribution of measles in these separate districts between 1890 and 1914, the years which the investigation covers. In London as a whole, however, between 1840 and the present day, great variations have taken place. These are illustrated in the diagram. In the first 18 years the winter period was much the most important, in the last 18 years the spring period. A point of special interest is evident in that the spring maximum has been appearing earlier and earlier in the year, at the same time as it has been increasing in importance. The maximum was over 800 in the first 18 years, nearly 900 in the second, over 1,200 in the third, and 1,300 in the last period. This suggests a phenomenon of greater complexity than that of which the combined figures for the seventy years, the only figures usually published, give indication. The interpretation of these phenomena must be left to some future inquiry.

APPENDIX I.

ON THE METHOD OF INVESTIGATING PERIODICITY IN DISEASE WITH EXAMPLES.

Of the different methods of determining periodicities there is only one which has any real importance in epidemiology. This is the method of periodogram analysis designed by Professor Schuster. Other methods have been suggested and in certain cases in astronomy and physical science these give true results, but they all suffer from the defect that they are exact mathematical methods and unless the figures given by the statistics differ by negligible amounts from the theoretical values they fail to give even approximate values of the true periodicities. Such regularity of occurrence is naturally not complied with in statistics relating to epidemiology. For instance, a single case of measles occurring in a large school will easily give rise to a much larger number of secondary cases than if it had arisen elsewhere. When the analysis is made by the method of the periodogram these divergences are of little importance. The method of the periodogram was originally designed for physical purposes and has been freely used by the author and others in the investigation of magnetic periodicity, the periodicity of sun spots, of variable stars, &c. Its use is, however, not confined to physical problems but can be extended equally to biological investigation, and the fullest use must be made of this method in the study of epidemiology before really important conclusions can be drawn. The only defect is the labour of application.

Briefly described the method is as follows: A long series of figures are taken representing the frequency with which some event has occurred during
equal intervals of time, say, for instance, the number of deaths from measles every week. These figures are written down in rows of a certain length. Thus, starting with a number such as fifty, the first fifty frequencies are written down in a row: the fifty-first frequency is placed below the first and a second row ending with the one hundredth frequency written down: the third row begins with the one hundred and first frequency. This process is repeated until as large a number of rows as the statistics allow have been written. The columns are then summed. A new beginning is then made, writing the figures in rows containing fifty-one consecutive frequencies and the whole process repeated. Proceeding by similar units each time the statistics are examined in the same way till the longest period which it is desired to investigate has been analysed. Either by inspection, or a more rigid application of a Fourier analysis, the instances among the series of rows representing the sum of the columns in which the greatest variation from maximum to minimum occurs, are selected. These give the most notable periodicities. The principle on which the method is based is that if a period of any length exists no additional row added will tend to make the maximum differ more markedly from the minimum, while if the same frequencies are added in rows of lengths which do not correspond to a period the maxima in successive rows will appear in columns which succeed each other and thus in the sum each will tend to neutralize the effect of the other.

The principle of the method of analysis is thus quite simple. It does not merely give one period but every period. The systematic investigation by gradually extending the length of each row allows every period to appear, since the presence of two or more periods—though making an investigation by guess-work almost impossible—does not raise any difficulties in the way of this method revealing the composite nature of the statistics examined unless the two periods are so similar in length that the series of statistics available is unequal to differentiate their effects.

As a first instance of the method a purely artificial example has been chosen to permit the mechanism of the process to be easily studied. Let us take a series:

\[
1 \quad 7 \quad 21 \quad 35 \quad 35 \quad 21 \quad 7 \quad 1
\]

which repeats itself regularly. Let there be added to this, term by term, a second repeating series:

\[
4 \quad 16 \quad 24 \quad 16 \quad 4
\]

It is obvious that before the compound series thus constructed repeats itself forty terms will be necessary since the one period comprises eight terms and the other five. The combination is shown on the following page (Table IV).

The series of terms thus obtained shows very considerable irregularity and certainly, though the longer of the periods might be guessed, the second seems sufficiently obscure.
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Applying the method the series of terms will be written out first in fours, then in fives, and so on, increasing by units till the rows contain nine terms. This is shown in Table V. From this table the method can be easily followed. When the terms are added in *fours* we find that the greatest differences observed in the sums of the columns vary from 268 to 308. This is a very slight variation. It is due to the form of the eight term period, as can easily be seen by adding the eight term period in the group of four terms each—

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In the next trial, in which a five term period is sought, the result is quite different. Here the sum shows a regular progress from 160 to 320 and back. Definite evidence of the presence of a period is found subject to the sums in periods of six terms showing less variation.

As six does not evenly divide 40 and as 120 is the least common multiple, the series requires to be repeated three times. For purposes of illustration the addition has been made in *three* sections: the first contains six rows, the second eight rows, and the third again six. When the three sub-totals are added, the total of each column is the same, namely, 576. This division into three sections has been made to illustrate the necessity of a sufficient number of periods being taken if certainty as to the existence or non-existence of a period is to be obtained or not. When the first sum of six terms is examined it is found that there is a regular progression from a term of 140 up to a term of 188. This might quite well indicate a period but not one of great moment.

It is, in addition, a form not often found, inasmuch as the progress from the maximum to the minimum alters suddenly between two successive terms and not gradually.
### Table V.

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<th>Terms summed in rows of Four</th>
<th>Terms summed in rows of Five</th>
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<td>Totals 308</td>
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Terms summed in rows of Six.

(a)

| 5   | 23 | 45 | 51 | 39 | 25 |
| 23  | 25 | 17 | 11 | 25 | 51 |
| 59  | 37 | 11 | 5  | 17 | 31 |
| 39  | 37 | 39 | 59 | 37 | 17 |
| 5   | 11 | 37 | 59 | 51 | 25 |
| 11  | 17 | 25 | 23 | 25 | 39 |
| Totals ... 140 | 152 | 174 | 156 | 188 | 188 |

(b)

| 51  | 15 | 23 | 5  | 5  | 23 |
| 45  | 51 | 39 | 25 | 23 | 25 |
| 17  | 11 | 25 | 51 | 59 | 37 |
| 11  | 5  | 17 | 31 | 37 | 39 |
| 39  | 37 | 31 | 17 | 5  | 11 |
| 37  | 99 | 51 | 25 | 11 | 17 |
| 25  | 23 | 25 | 30 | 51 | 45 |
| 23  | 5  | 5  | 23 | 15 | 51 |
| Totals ... 248 | 236 | 216 | 216 | 236 | 218 |

(c)

| 39  | 25 | 23 | 25 | 17 | 11 |
| 25  | 51 | 39 | 37 | 11 | 5  |
| 17  | 31 | 37 | 39 | 39 | 37 |
| 31  | 17 | 5  | 11 | 37 | 59 |
| 51  | 25 | 11 | 17 | 25 | 23 |
| 25  | 39 | 51 | 15 | 23 | 5  |
| Totals ... 188 | 188 | 186 | 174 | 152 | 140 |

Totals of (a), (b), and (c) summed.

| (a)  | 140 | 152 | 174 | 186 | 188 | 188 |
| (b)  | 248 | 188 | 186 | 174 | 152 | 140 |
| (c)  | 188 | 236 | 216 | 216 | 236 | 248 |
|      | 576 | 576 | 576 | 576 | 576 | 576 |
Brownlee: *Periodicities of Epidemics of Measles*

**Table V—(continued).**

Terms summed in rows of Seven.

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* Maximum indicated by the asterisk.
### Table V—(continued).

**Terms summed in rows of Eight.**

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(d)

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**Tables showing Sum of (a) (b) (c) and (d).**

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<th>1,152</th>
</tr>
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</table>

**Final totals.**
For seven terms in a row, it is necessary to write the forty figures in the series seven times, but as the original series examined reads from the end in identically the same terms as from the beginning it is only necessary to write out half of the analysis. To obtain the complete totals the sums of the columns as found and as written in inverse order were added together. The writing out has again been done in four sets of additions. In this case if only a few rows were added together a misinterpretation of the result might easily occur. As added together in sets of five rows quite large differences between the maxima and minima are found. It will be noticed, however, that the maximum in each set of five is in a different place. When the first four series are added together little evidence of a period is seen. When to the last sums the figures written in inverse order are added, the result becomes constant. Thus there is no period with seven terms.

With eight terms a completely different result is seen. Here there is a regular progress from a minimum to a maximum and again to a minimum, a result which would evidently remain constant no matter how many more complete sets of figures were added.

With nine terms again, the same phenomena as were found with regard to seven terms in a row are seen.

It is thus found that in a given case where a complex set of figures was deliberately constructed it is possible to obtain by the method information regarding the periodicities corresponding to the facts which we know beforehand to be correct.

If there are sufficient figures available periodicities can always be found by this method. If the series is small there may easily be doubt as to which of two near periods is the true one. However, every succeeding row added on tends to eliminate error. Take the case of seven terms already considered where the terms are successively summed in five rows. In the first sum \( (a) \) marked variation exists, but with the addition of each extra set of rows this difference disappears. This is shown in Table VI.

<table>
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<th>( (a) + (b) )</th>
<th>( (a) + (b) + (c) )</th>
<th>( (a) + (b) + (c) + (d) )</th>
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</tr>
<tr>
<td>( b )</td>
<td>258</td>
<td>264</td>
<td>( (b) )</td>
<td></td>
</tr>
<tr>
<td>( c )</td>
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<td>( (c) )</td>
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</tr>
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<td>( d )</td>
<td>542</td>
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<td>( (d) )</td>
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</table>

Thus in the first sum the maximum is nearly twice the minimum, while in the last it is barely 20 per cent. greater.

Frequently it will be found that the real period lies between two sets of sums. The method of approximating to this can easily be shown from the example considered. It is not an exact but an approximate method. If the rows of any definite length are not summed in one sum, but in a series of sums, as has been done in this case for seven and nine terms, it will be seen that the maximum and minimum do not occupy a constant position in the subtotals but move in a position from sub-total to sub-total. It is obvious that
if the period examined is too short the maximum will tend to be found further forward with each successive sub-total, since in the true period the maxima come truly under each other in place, and if the number of terms in the row is too small this maximum will move forward correspondingly with each new row. Likewise, if the number of terms is too great, the maximum will tend to move back. It is not possible to say with one set of figures whether the maximum is moving backwards or forwards, but with two sets, one on either side of a true period, or both on one side, only one solution is possible. It will be found, looking at the series of seven and nine, where the position of the maximum is shown by an asterisk, that a movement forward of the maximum in the seven term analysis agrees with the moving back of the maximum in the nine term analysis. Counting how many terms in the sum the maximum moves with each separate addition, and dividing this sum by the number of rows in each separate addition, we obtain an approximate value of the error in the period. Thus with the seven terms addition the maximum moves four spaces between (a) and (b), four and a half between (b) and (c), and four and a half between (c) and (d). Dividing these figures by five, the number of rows in each sub-total, we find that the true period should be 0'8 to 0'9 of a division longer than that tried.

In the same way with the nine term period, the maximum moves back six spaces between (a) and (b), five and a half between (b) and (c), and five and a half between (c) and (d).

The period of nine terms is thus 1'2 to 1'1 times too long. The mean of these extremes is exactly unity, and this we know to be the true difference from the actual period. This method can often be usefully applied.

Passing now to an example furnished by actual statistics, a simple instance relating to the periodicity of deaths from measles in Aberdeen during the years 1856-1885 has been selected (Table VII). This example possesses a double periodicity, the one due in some way to the seasonal factor and the other independent of this. The first period is annual, the second during the years mentioned almost exactly 2'75 years. Of course, absolute regularity cannot be expected in such a biological phenomenon, but it will be seen that the periodicity is singularly constant. In only one case is it found that the maximum of an epidemic occurs at any appreciable distance from the position required by the period considered. Each term in the series of figures refers to the number of deaths in one quarter of the solar year. These numbers have been summed in five different ways. A period of two years or eight quarter-years has been taken as the starting point. To this period in each succeeding test one quarter of a year has been added to the lengths of the rows till each row contains the figures for three years. The first addition seems to show that measles is more prevalent in alternate years, but it is chiefly of importance as illustrating the marked climatic factor. In the first and fourth quarter of the year twice as many deaths occur from measles as in the second and third quarters, as can be seen by adding the totals in four terms thus:
This method of addition obviously gives the same results as if all the original terms had been specially written in rows of fours. Examining the sum from two years upwards, it is easily seen that with the approach to 2.75 years the maxima tend to group themselves more and more nearly in a vertical line, and with that period the difference between the minimum and maximum become so great as to show that measles was practically absent except at very definite intervals. In addition the approach to the maximum and the decline from it becomes somewhat symmetrical. There is thus no doubt at all as to what period shows the greatest regularity in recurring. Variation from exactitude occurs, especially in the fourth and sixth rows, but when the eleven successive periods have been added this makes little difference in the result. As before seen in the examination of the theoretical example, this number of additions has been found quite sufficient to eliminate serious error. Thus though, as has already been seen, there is an undoubted association between the number of deaths from measles and the seasons of the year it is evident that the disease recurs at intervals which have no relation at all to the solar year, since it is obvious that in a series of 30.5 years with a period of 2.75 years the maximum of the epidemic has been in each season three times. Something independent of season is exercising a controlling influence. The proof of this does not depend on this example alone. Similar periodicities of different lengths and of length independent of the solar year are the rule, as has already been seen.

**Table VII.**—Analysis of the Statistics of the Measles Deaths in Aberdeen, 1856-1885, for Each Quarter Year, to Show Periodogram Methods.

Terms arranged in 2-year Periods.

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Terms arranged in 24-year Periods.

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Terms arranged in 24-year Periods.

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<td>102</td>
<td>182</td>
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<td>183</td>
<td>107</td>
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Terms arranged in 24-year Periods.

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<td>-</td>
<td>1</td>
<td>-</td>
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<td>1</td>
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AU—13
Table VII—(continued).

Terms arranged in 3-year Periods.

<p>| | | | | | | | | | | | |</p>
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<td>12</td>
<td>83</td>
<td>9</td>
<td>1</td>
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</tbody>
</table>

Totals 69 105 57 270 237 106 30 110 34 77 40 68

Having thus described the method and made application of it to theoretical and practical cases, a description of a complete investigation of the method, such as that referring to measles in London between the years 1840-1912, may well follow. From the year 1840 to the year 1912 the weekly numbers of deaths were extracted from the returns of the Registrar-General. As London increased so much in size that the population of children under 5 years of age more than doubled itself in this interval, the number of deaths was first corrected to bring the death-rates in each year to a comparable value. After this, the figures were written down in rows, beginning with a period of 80 weeks. With this starting point the periods were increased successively by one week till a period of 120 weeks was reached; from this period the increase was made by fortnightly intervals up to 160 weeks; after this increase by quarter-year intervals was found sufficient. In the actual working the additions of the columns were made not only for the complete series of years, but also in four divisions, each of which covered approximately eighteen years. In this way it was hoped to determine if any phenomenon observed in the full totals had been constantly or inconstantly present. It was found that during 72 years, the chief period between epidemics of measles in London, has been, on the average, 97 weeks exactly. The sums of the totals for each epoch of 18 years for the periods 95, 97 and 99 weeks, and the corresponding complete totals are shown in the accompanying diagram (Diagram XI). It will be observed how narrowly the main period is defined. In the first set of graphs, that referring to 95 weeks, the march of the maximum is steadily forward; in the last set of graphs, that referring to 99 weeks, the march of the maximum is as steadily backwards. With 97 weeks the maxima and minima fall in the same vertical line. Irregularities in the statistics of individual years thus tend to be smoothed out and the fundamental facts to declare themselves. For periods of 95 or 99 weeks the total sum shows very moderate variations from the mean line. For 97 weeks the variation is extremely marked. The sites of the other periods are easily observed by reference either to Diagram I or Table III.
This diagram illustrates graphically the results of adding the figures for London in periods of 95, 97 and 99 weeks. The additions have been made in four sections and for the whole epoch. The former are shown in the upper part of the diagram and the complete additions below. The scale on which the latter is exhibited is one-tenth that of the former. It will be noticed how small a swing in the complete totals is found at 95 or 99 weeks and what a marked swing at 97 weeks. A full description of this diagram is given in the text.
APPENDIX II.

Fallacy which may arise in Periodogram Analysis.

There is one fallacy which must be guarded against when carrying out the process of periodogram analysis with reference to disease statistics. This fallacy arises if a few epidemics much larger than the ordinary epidemics occur between which there is a considerable interval of time. What happens in the analysis will be best appreciated by considering a simple example. In this example it is assumed that two large epidemics occur with no cases of the disease in the interval between. In the first writing of the figures chosen (Table VIII, \( a \)) these two epidemics are shown in the same column associated with rows of ten elements. In the second writing (Table VIII, \( b \)) they come again into juxtaposition in the same column, associated with rows of fifteen elements.

Table VIII.

\[
\begin{array}{ccccccccc}
0 & 0 & 0 & 0 & 0 & 90 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 90 & 0 & 0 & 0 & 0 \\
\hline
\text{Total} & 0 & 0 & 0 & 0 & 180 & 0 & 0 & 0 \\
\end{array}
\]

\[
\begin{array}{ccccccccc}
0 & 0 & 0 & 0 & 0 & 90 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 90 & 0 & 0 & 0 & 0 \\
\hline
\text{Total} & 0 & 0 & 0 & 0 & 180 & 0 & 0 & 0 \\
\end{array}
\]

It is quite obvious that a maximum will be found in each of these positions, though there is no evidence of any real periodicity. This kind of feature has made the investigation of the Newcastle statistics impossible and it must always be watched for in practice.

APPENDIX III.

Method of Testing the Probability of Periods.

The method of testing the probability of a period is given by Professor Schuster. It is as follows: A region of the statistics in which there is no probable period is chosen as exhibiting the values which the amplitudes must take if chance alone determine their magnitude. It is advisable to take a
considerable range of successive values if such are available. They should be chosen at equal increments of the period. These amplitudes are squared and their mean square taken. The ratio of the square of any amplitude to this quantity determines its probability of independent existence. The probability that a period exists with the amplitude observed is ascertained after consulting the following table, which has been taken from Professor Schuster.

Table IX.

<table>
<thead>
<tr>
<th>k</th>
<th>$e^{-k}$</th>
<th>k</th>
<th>$e^{-k}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.05</td>
<td>0.9512</td>
<td>6</td>
<td>2.48 x 10^{-3}</td>
</tr>
<tr>
<td>0.10</td>
<td>0.9048</td>
<td>8</td>
<td>3.35 x 10^{-4}</td>
</tr>
<tr>
<td>0.20</td>
<td>0.8187</td>
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<td>4.54 x 10^{-5}</td>
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<td>0.40</td>
<td>0.6703</td>
<td>12</td>
<td>6.14 x 10^{-6}</td>
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<tr>
<td>0.60</td>
<td>0.5488</td>
<td>14</td>
<td>8.32 x 10^{-7}</td>
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<tr>
<td>0.80</td>
<td>0.4493</td>
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<td>1.00</td>
<td>0.3659</td>
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<td>1.52 x 10^{-8}</td>
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<td>1.50</td>
<td>0.2231</td>
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<td>2.06 x 10^{-9}</td>
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<td>0.1358</td>
<td>25</td>
<td>1.29 x 10^{-11}</td>
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<td>0.0498</td>
<td>30</td>
<td>9.36 x 10^{-14}</td>
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<td>0.0183</td>
<td>40</td>
<td>4.25 x 10^{-18}</td>
</tr>
<tr>
<td>5.00</td>
<td>0.0067</td>
<td>50</td>
<td>0.2 x 10^{-22}</td>
</tr>
</tbody>
</table>

$k = \text{ratio of square of amplitude examined to the mean square of the check amplitudes.}$

$e^{-k} = \text{probability that result is not due to a real periodic cause.}$

For London the values of the amplitudes for periods of 118 weeks to 160 weeks at intervals of two weeks have been chosen as the criterion values. These give a mean square amplitude of 4,315. This number divided into the square of 417, the amplitude at 97 weeks, gives almost exactly 40. Reference to the table thus shows immeasurably great odds in favour of the existence of a true periodicity. An amplitude of 200 gives a value of the ratio of 9, which shows a probability in favour of a true periodicity of between 3,000 and 20,000 to one. For Glasgow the value of the mean square is much larger—namely, 22,426—as is to be expected from the smaller length of the statistics. The amplitude of the main periods of 98 weeks and of 109 weeks are, however, high, 626'5 and 563'7 respectively, and the ratios are 17'5 and 14'2. The probability of these amplitudes representing true periodicities are thus in the region of millions to one. The amplitude at 89 weeks gives a ratio of 5'2, equivalent only to odds in favour of a period of about 180 to one, much smaller than those referring to the two main periods, and possibly not significant.
APPENDIX IV.

THE METHOD OF DETERMINING THE AMPLITUDE.

The method of determining the amplitude of a series of figures is now given in most text-books, but there are one or two points which make for simplicity in this kind of work which are usually neglected. To calculate the amplitude commonly 12 or 24 divisions are used. Twenty divisions are used in my office for the reason that division by 20 is much more simple than division either by 12 or 24.

In the summations referred to in the previous pages the total number of elements in the row have ranged from 80 to 160. To reduce this variable number of elements to 20 is a work of much greater ease than to reduce them to 24. Taking the case of 97 elements for instance, each of the 20 elements will then contain 4\(\frac{15}{52}\), or 4.85 of the 97 units. With a calculating machine it is a matter of considerable rapidity to obtain these 20 elements. The first figure in the 20 series contains the first four elements of the series in the statistics, and 0.85 of the next; the second figure contains the remaining 0.15 of the fifth element, the next four elements and 0.7 of the tenth element; the third set of figures contains 0.3 of the tenth element, the next four elements, and 0.35 of the fifteenth. The calculation thus can be quickly run through. Denote these twenty elements then by \(u_1, u_2, \ldots\), write them as follows, add and subtract:

\[
\begin{align*}
\text{Sums} & : v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9, v_{10}, v_{11}, v_{12}, v_{13}, v_{14}, v_{15}, v_{16}, v_{17}, v_{18}, v_{19}, v_{20} \\
\text{Differences} & : w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9, w_{10}, w_{11}, w_{12}, w_{13}, w_{14}, w_{15}, w_{16}, w_{17}, w_{18}, w_{19}, w_{20} 
\end{align*}
\]

The sum and differences have been respectively denoted by \(v\) and \(w\), with appropriate suffixes. Re-write these as below and make corresponding additions and subtractions.

\[
\begin{align*}
\text{Sums} & : a_1, a_2, a_3, a_4, a_5, a_6, a_7, a_8, a_9, a_{10}, a_{11}, a_{12}, a_{13}, a_{14}, a_{15}, a_{16}, a_{17}, a_{18}, a_{19}, a_{20} \\
\text{Differences} & : \delta_1, \delta_2, \delta_3, \delta_4, \delta_5, \delta_6, \delta_7, \delta_8, \delta_9, \delta_{10}, \delta_{11}, \delta_{12}, \delta_{13}, \delta_{14}, \delta_{15}, \delta_{16}, \delta_{17}, \delta_{18}, \delta_{19}, \delta_{20} 
\end{align*}
\]

If now the period is expressed by two harmonic terms, all ever required for practical purposes, the curve as usually written is given by the next equation.

\[
y = a_0 + a_1 \cos \theta + a_2 \cos 2 \theta + b_1 \sin \theta + b_2 \sin 2 \theta
\]
Then we have
\[ a_0 = \frac{1}{\sqrt{n}} (a_1 + a_2 + a_3 + a_4 + a_5), \]
\[ a_1 = \frac{1}{\sqrt{n}} (b_1 + 0.951 b_2 + 0.809 \beta_1 + 0.588 \beta_2 + 0.309 \beta_3). \]
\[ a_2 = \frac{1}{\sqrt{n}} (a_1 - 0.809 a_2 - 0.309 a_3 - a_4). \]
\[ b_1 = \frac{1}{\sqrt{n}} (0.309 \gamma_1 + 0.588 \gamma_2 + 0.809 \gamma_3 + 0.951 \gamma_4 + \gamma_5). \]
\[ b_2 = \frac{1}{\sqrt{n}} (0.588 \delta_1 + \delta_2 + 0.809 \delta_3 + \delta_4). \]

The amplitudes are of course, \( \sqrt{a_1^2 + b_1^2} \) and \( \sqrt{a_2^2 + b_2^2} \), and the proportional amplitudes \( \frac{\sqrt{a_1^2 + b_1^2}}{a_2} \times 1,000, \&c. \)

APPENDIX V.

ON THE RANGE OF AMPITUDES DUE TO A PERIOD.

When there is a limited series of statistics it is not always easy to determine from the periodograms (Diagram VII) whether only one period or a mixture of several periods is present. Had we always the range of statistics available for London there would not be much difficulty in determining this, but this range is in most cases lacking. To illustrate the point the data referring to amplitudes found in the neighbourhood of 98 weeks in the city of Edinburgh have been regraphed (Diagram XII), the observations being

![Diagram XII](image)

This diagram shows the method of graphically distinguishing whether the range of amplitudes found is what might be expected. The actual observations are indicated by crosses; the theoretical by circular dots. The unit of abscissa between the theoretical values is 1.15 times that of the unit of abscissa used in charting the actual values. The observations graphed in this diagram are the same observations as have been already shown on a smaller scale in Diagram VII in the first section of the periodogram for Edinburgh.
indicated by crosses. It will be noticed that the amplitudes relating to periods of 97 and 99 weeks are very nearly as high as that relating to a period of 98 weeks, while for 96 and 100 weeks the amplitudes are still very considerable. In order to furnish the mechanism of a ready test as to whether any such variation is in the range of what should be expected or not the following table (Table X) has been calculated:

<table>
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<th>Length of periods</th>
<th>Amplitudes</th>
<th>Amplitudes in case of Edinburgh</th>
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<td>96</td>
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<td>98</td>
<td>505</td>
<td>345</td>
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<td>99</td>
<td>757</td>
<td>517</td>
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<td>100</td>
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<td>639</td>
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<td>683</td>
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<td>102</td>
<td>236</td>
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<td>103</td>
<td>757</td>
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</tr>
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<td>104</td>
<td>505</td>
<td>345</td>
</tr>
<tr>
<td>105</td>
<td>236</td>
<td>161</td>
</tr>
</tbody>
</table>

The true period is taken as 100 weeks and it has been assumed that twenty rows of observations are added together. The amplitudes expected have been calculated on the assumption that the epidemics have been of uniform size and form. To apply this table arithmetically demands skill in calculating but to apply it graphically is exceedingly simple. The rule is as follows: Chart the amplitudes with a uniform abscissa of period (in our statistics one week) on squared paper. Let \( p \) be the period to be examined and \( n \) the number of rows in the actual statistics then the unit of abscissa used in the calculated table is to be multiplied by \( \frac{p}{100} \times \frac{20}{n} \) or \( \frac{p}{5n} \) before the figures in the table are charted. Before charting the ordinates in the table they are to be reduced in proportion to the maximum amplitude of the period examined.

In the present case, that of Edinburgh, \( n = 17 \) and \( p = 98 \), that is, the number of rows in the statistics is 17, and the period approximately 98 weeks. The new unit of abscissæ is thus \( \frac{98}{5 \times 17} \) or 1'15 weeks. Take, then, 683 as the maximum and proportionately reduce the values in Table X, as shown in the third column. When the diagram of the periodogram is examined, it is easily seen that it is nearly symmetrical about a mid-line corresponding to 97'8 weeks. Take this as the origin, then measure from it on either side successive units of 1'15 weeks and erect ordinates corresponding in length to the ordinates given in Table X. A smooth curve is drawn through the results. The correspondence in this case is nearly perfect. For a first approximation which is all this purports to be it may be regarded as proved that in this region only one period exists. It is not so, however, when the 96 weeks' period found in Sheffield is examined in a like manner. This can be
easily shown to be compounded of different close periodicities: the exercise is left to the reader. The period is there 96 weeks approximately and the number of rows 21 (vide Table III).

REFERENCES.

The following references bear on various aspects dealt with in the text:—

For Treatment of the Harmonic Analysis.


Ditto, and for a General Survey of the Methods of the Periodogram:—


Schuster, Arthur, F.R.S., Professor of Physics at the Owens College, Manchester. "The Periodogram of Magnetic Declination as obtained from the Records of the Greenwich Observatory during the years 1871-1895." Cambridge Philosophical Transactions, xviii, VI.


DISCUSSION.

Mr. Yule: With regard to Dr. Brownlee's exposition of the results that he has reached, I can speak from the statistical standpoint only. Some of the results seem to me very remarkable. In the first place there is the predominance of two periods—roughly 87 and 97 days—which bear no relation whatever to the solar year. If these periods represent, as assumed by the author, periods of some kind in the life-history of the causative organism, the fact that they bear no relation to the seasons is striking. Next, there is the appearance in Liverpool of a period that is quite inconspicuous in London, and still more remarkable a period in South London which, during the whole of the time covered by the observations analysed, never became significantly conspicuous in North London. If these different periods represent different sub-types of the organism, it is difficult to understand why the sub-group dominant south of the river never succeeded in crossing the bridges and becoming even moderately conspicuous in districts on the north bank. Finally there is the remarkable alteration in the form of the annual curve with its retrogression of the maximum to an earlier date of the year. With reference to this point, has Dr. Brownlee found any evidence of a period slightly shorter than the year, the presence of which might account for such a shift? Also, on a technical point, has he found any difficulty in dealing with the longer periods? I have not had occasion to use the periodogram method myself, but in giving some assistance to a friend we found a source of difficulty, and of possible fallacy, which does not seem to be noticed in the accounts of the method which I have seen. The difficulty is that, in taking the range of values observed in your row of totals for a period $2p$, you may be merely repeating the range for the period $p$, two complete waves of which will be included. I assume, of course, that Dr. Brownlee avoided this source of fallacy, but how did he deal with it?

Dr. W. H. Hamer: The author tells us he has long held that variations in the measles organism, or organisms, are of primary importance in determining the form of the measles waves. Many people have urged on the other hand, that primary importance must be attributed, as regards these measles waves, par excellence, to the numbers and availability of susceptible persons. I tried to illustrate this point of view in the Milroy Lectures of 1906. Studied from this aspect significance must of course be attached to rise and fall of birth-rates, movement and aggregation of population, crowding, facilities as regards transport, and note must also be taken of administrative action. Dr. Brownlee seems disposed to wave all such considerations aside and to pin his faith exclusively upon variations in the germs. I should like to allude, in illustration of the possibility that preventive measures may have some effect, to the question of the modification of the seasonal curve of measles mentioned by Dr. Brownlee. In my Annual Report for 1912 reference was made to the
changed form of the seasonal curve—the mean curve for 1874-1912 was contrasted with that for 1840-73—and the following suggestions were thereupon made. "It may be taken that the Elementary Education Act began to exert its influence about the year 1874. The accentuation of the curve of measles mortality during the latter series of years may in all probability be attributed to the increasing aggregation of population, combined with the effects produced by compulsory school attendance. Under modern conditions the natural tendency of measles to exhaust the susceptible cases finds freer expression, with the results that outbreaks climb more readily to the maxima; and, correspondingly, intervals are left in which the disease is less active than formerly." On the other hand the more complete preventive measures (excluding scholars, closing classes, &c.) of later years are doubtless in part responsible for cutting off the tops of the measles peaks. Dr. Brownlee points out that the summer maximum in London has in recent years moved forward from June to May or even April. I venture to suggest that this phenomenon (which is clearly exhibited in Diagram D of my Report for 1912) may be in part attributable to preventive measures.

Mr. M. Greenwood: I do not agree with Dr. Hamer's concluding remarks. It was no part of Dr. Brownlee's duty to consider the various agencies enumerated by Dr. Hamer, since the object of the research was to determine whether periodicity existed; the explanation of the phenomenon must come later. With Mr. Yule, I think that it is impossible upon the spur of the moment to discuss the very important and perplexing results disclosed and will merely refer to two points. May it not be possible that, in the case of some of the shorter series, the differences in length of the periods are within the limits of sampling errors, so that the total number of distinct periodicities, distinct that is from one geographical region to another, may be smaller than appears on the face of the statistics? The second point is whether the oscillation of the seasonal maxima is a sufficient proof that the production of these maxima cannot be attributed to meteorological factors. Only those who have attempted to use the periodogram method can realize the immense labour involved in the present inquiry. The gratitude of the Section is due to Dr. Brownlee and his staff, who have enabled us to include in our Proceedings so valuable a contribution to the quantitative study of epidemiology.

Dr. Brownlee (in reply): I shall refer to two criticisms that have been made. One of these was that death statistics were used and not case statistics. With regard to this point, there are only two sets of case statistics to which I have had access, those of Aberdeen and Glasgow. In the first case the periodicity of the epidemics is almost absolutely biennial, and the number of cases give no information which is not given by the number of deaths. In the other case—namely, Glasgow, the correspondence between the cases and deaths is also close. Compulsory notification has, however, never existed in Glasgow. The number of known cases also varies with the amount of distress in the
city. For instance, at times, when trade is bad, many more cases become known to the sanitary authorities than when trade is good, on account of the desire of the parents to have their children looked after. Making allowance for this, no facts have been discovered from the case statistics which have not been found from those of deaths. With regard to the effect of the season of the year, I have been unable to discover in what way the effect was produced. If I may make a prophecy, referring to Diagram I, I should feel inclined to say that something similar to the first three sections of the diagram will be found to describe the future course of measles in London. I hope to be alive in 1936 and to demonstrate this at that date.
Section of Laryngology.

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April 10, 1919.

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Mr. W. M. Mollison (p. li); Mr. J. F. O'Malley (p. iii); Mr. G. H. Pooley (Sheffield) (p. lv); Mr. E. H. E. Stack (Bristol) (p. lvii); Mr. W. Stuart-Low (p. viii); Mr. Herbert Tilley (p. ix); Mr. H. Lawson Whale (p. x); Dr. James Donelan (p. xi); Mr. L. V. Cargill (reply) (p. xiii); Mr. Arthur W. Ormond (reply) (p. xiv); Mr. E. D. D. Davis (reply) (p. xiv); Mr. G. Seccombe Hett (reply) (p. xv); Mr. E. Treacher Collins (p. xvi); Mr. Herbert Tilley (p. xvi).

The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.
Section of Laryngology.

President—Dr. James Donelan.

PRESIDENT'S ADDRESS.

British Laryngology and Rhinology.

By James Donelan, Ch.M., M.B.

(Chevalier and Officer of the Crown of Italy.)

It seems that the time is fully ripe for considering how far British laryngology and rhinology are in a position to take, how far do they deserve, a leading position in the great scientific movement that will shortly be taking place. What have we accomplished in the past, what promise does it afford for the future?

It is not my intention to attempt a detailed survey of the history of British laryngology. When one looks back on even the thirty odd years I have, myself, been connected with it, one cannot help seeing that so much has been accomplished that anything like an adequate survey, even a catalogue raisonné would take up more than the whole time at our disposal this evening. I hope, however, to refer to our scientific progress in such a form as to show at this critical moment, when the world is about to take stock of everything, that British laryngology and rhinology have ever been in the van of progress, and that, if we owe something to Germany, it has chiefly been the systematization, not to say exploitation, of ideas that very often had their origin not only in these islands but in this Section, or in the societies that were its forerunners.

I also propose to put before you some of the many suggestions that have been kindly offered to me, so that we may consider at some

1 At a meeting of the Section, held November 1, 1918.
more convenient time in what way, if in any, we can improve our organization and keep it fit to maintain the reputation I am told we enjoy as the most energetic, hard working and enthusiastic Section of this great Society.

It seems only natural that the country which is the parent home of laryngoscopy should be a leader in all that has resulted from the invention. Every one has his own view as to whom the credit of the invention belongs. I do not intend to discuss it now, I will only briefly mention the facts that go to prove that this is the parent home of laryngoscopy.

Though mirrors had often been used for seeing round awkward corners, no one seems to have thought of using a mirror for examining the larynx until the beginning of the last century. A mirror "on a long shank" for examining the larynx was first demonstrated on March 18, 1829, before the Hunterian Society by Benjamin Guy Babington, afterwards, like his father before him, physician to Guy's Hospital and founder of the Epidemiological Society. In the words of the account in the Hunterian Transactions, "the doctor proposed to call it the glottiscope." It is much to be regretted that the cholera epidemic of a year or two later induced him to turn his great talents to epidemiology, which was then by no means ripe for scientific study, instead of to diseases of the larynx. Babington's invention was not however, lost sight of, as is commonly supposed, for a laryngeal mirror, or "a mirror such as dentists use" is recommended, quite as a matter of course, in works on general surgery—Liston, for instance, in 1840—as a simple means of examining the larynx. It was this simple means or, as Manuel Garcia says in the paper read on his behalf at the Royal Society in 1854, "the method that I adopted," that he employed in the study of his own larynx. In that paper entitled "Observations on the Human Voice," Garcia makes no claim to having invented anything, and though I knew him fairly well and had many talks with him on this subject, especially between the years 1887 and 1891, as I am not discussing it now, I will merely say that he was undoubtedly the inventor of auto-laryngoscopy. As Manuel Garcia was domiciled here for some sixty-five years of his long life, and had worked as a professor of singing at the Royal Academy of Music for several years before he made his communication on the human voice to the Royal Society, I think, whatever views we may hold as to the inventor of the laryngoscope, whether Babington or Garcia, we may fairly claim that this is the parent home of laryngoscopy, although the credit of
first reporting any clinical work done with it appears to belong to the Hungarian, Johann Czermak, the teacher of Morell Mackenzie.

Czermak published the first edition of his book, “Der Kehlkopfspiegel und seine Verwerthung für Physiologie und Medizin,” in 1860. A copy of this very rare first edition is in the Society’s library. I do not find that Czermak, amongst his many subsequent writings, published anything in connexion with laryngology except reprints of this one work. In 1863, three years after Czermak’s book appeared, his pupil, Morell Mackenzie, had already founded the Hospital for Diseases of the Throat in Golden Square, being I believe the first institution ever devoted to the objects of our special study. It was there he did all his best work, and there he collected the bulk of that immense material for those works on every department of our study which at once placed British laryngology in the very front of the scientific progress of the world.

The time has happily gone by when English text-books dealing with every department of our subject could be written with scarcely a mention of Morell Mackenzie’s name, when a veil of silence was allowed to fall upon every reference to the work of the father of British laryngology even in the recognized centre of that science, or when it could be lumped together with that of humbler workers and contemptuously dismissed as the effort of an anonymous pioneer.

Morell Mackenzie’s writings will always be amongst the classics of our special literature in English. They are a remarkable achievement for one man—a man harassed from his boyhood by almost continuous attacks of spasmodic asthma—when one remembers that it is all based on over twenty years’ personal pioneer observation of his own cases and a wide reading, probably unique in its extent, of the cases of others. If his “Diseases of the Throat and Nose” has not attained to succeeding editions, like other text-books of proved value, it was because he was cut off while preparing the second edition. It would have contained much new matter and some corrections. The references in this work compass the history of our speciality from the earliest records up to the year 1884. It is not a dead bibliography, however, for the views of almost every writer of importance are discussed or commented upon. Scarcely any author is omitted, certainly none intentionally.

“Diseases of the Throat and Nose” was commenced in 1872. The first volume appeared in 1880 and the second in 1884. The writer explains the slow rate of progress and the changes owing to the rapid development of a new speciality that took place while the book was
being prepared. Even the second volume was not completed according to the design of the author, as the "Section on Diseases of the Neck" had to be omitted. This section was to have included his chapters on goitre, of which I myself brought the references up to 1891. These chapters then existed only in a single set of galley-proofs, which have since been lost. Besides the usual exhaustive history of the diseases of the thyroid gland from the earliest times, Mackenzie had previously brought the literature up to about 1884. He did not limit his view of the causation to geological peculiarities of the water supply, but, from evidence furnished to him mostly by Indian medical friends, held that it would probably be found that the water was infected by the excreta of goitrous persons. This view, as many of you are doubtless aware, has been taken and its truth brilliantly demonstrated by Colonel McCarrison, of the Indian Medical Service, in the epoch-making work he published last year. I should strongly recommend those who have not yet had an opportunity of studying this work that they should not omit to do so, and especially the pages on the relation of subthyroidism to enlargements of the pharyngeal tonsils. They are bound to have a considerable influence on present treatment.

It is of interest to recall the excellent pioneer work recorded by Mackenzie in his second volume in relation to œsophageal diseases. The results of his examinations, with very imperfect means, of a large number of cases, have a permanent value even in these œsophagosscopic days. Mackenzie's other works in relation to our speciality are: The series of "Essays on Throat Diseases," including: "Hoarseness, Loss of Voice and Stridulous Breathing in Relation to the Nervomuscular Affections of the Larynx"; "Growths in the Larynx," which will, I think, always be the universal classic of that subject although somewhat marred by the unfortunate tone of the Durham controversy. Other essays were: "Diphtheria, its Nature and Treatment"; "The Use of the Laryngoscope"; "Hay Fever," and "The Hygiene of the Vocal Organs." All these works were most favourably received by the medical profession and press throughout the world. Nowhere was the chorus of approval louder or more enthusiastic than in Germany, where the book was anxiously awaited, and where for many years after it was done into German it was the principal and indeed the only good textbook of our subject.

In considering the scientific value of Morell Mackenzie's work it should never be forgotten that the bulk of it was pioneer work. The most remarkable thing about it is that his views and conclusions have
stood the test of time and have been so little affected by modern progress or even improved statistical research. Some doubts have been expressed of the value of Mackenzie's statistics but the best proof of their reliability is that the most modern figures tend only to confirm them. It has also been said that Mackenzie, especially in his later years, did not keep case notes. From my own personal knowledge I can say that this is simply not true. As in most other practices the bulk of his cases had but little special interest, but even of these he kept a brief note of the names, ages, dates when seen, treatment ordered, and the result. He kept fuller notes of all cases of any special interest. In his case as regards material he suffered rather from an *embarras de richesse* which was really beyond the physical powers of a not very strong man to deal with adequately.

I have devoted so much time to the scientific work, hitherto neglected, of the father of British laryngology that I can refer only very briefly to the contributions of other British pioneers and workers who are dead. The work of Dr. Edward Woakes has been of permanent value in furnishing a useful working theory of the causes of intranasal suppuration and polypus formation, and I believe there are good grounds for regarding him as the first Englishman who wrote a scientific account of diseases of the pharyngeal tonsil and that at a time before Meyer's paper had become known to him or to other English readers.

Lennox Browne too, besides being the founder of the Central London Throat Hospital, accomplished work of the highest value in laryngology and rhinology, and those labours find a worthy monument in the second edition of his text-book, in which he was assisted by the regretted Dr. Cagney. The work of Spencer Watson, Adams and Walsham will be remembered especially by our senior members. Durham, whose tracheotomy tube is still unrivalled, will never be forgotten as long as humanity wants to breathe in spite of opposition. It is hardly necessary for me to recall the work of Henry Butlin, or of Cresswell Baber, whose contributions stand in the front rank of the literature of our subject of this or any other country.

Passing to the work of living laryngologists, almost all of whom are members of this Section, I hope none of them will think me discourteous if I follow a recommendation given me and refer only to British work without the names of the workers. Look at what has been done in this country, most of it pioneer work of the highest value, in the study of diseases of the nasal accessory sinuses and especially in regard to the operative treatment of frontal, maxillary and sphenoidal abscess; the
significance of paresis of the larynx as a forerunner or early symptom of phthisis; the great improvements in the diagnosis and treatment of nasal lupus and tuberculosis of the larynx which enable us to approach such cases with far more confidence of success than twenty or even fifteen years ago. Take what has been done here to advance the study of malignant disease of the larynx and to perfect the technique of the operations for its treatment. Technical advances which have made thyrotomy, for instance, one of the safest instead of one of the most risky of surgical enterprises. Let us also recall the immensely valuable investigation of the innervation of the larynx and of the symptoms to which the various paralyses give rise; the treatment of deformities of the nasal septum; the improvements in submucous resection; the introduction of submucous turbinectomy. The magnificent work done by our fellow countrymen and especially our fellow members in relation to diseases of the œsophagus and pharyngeal pouches is unequaled in any country, while if Brünings invented the bronchoscope it was only by the application of principles discovered by Newton, Dolland and Harris, and these had been made use of long before him by Fisher, of Boston, Désormeaux, of Paris, and Sir Francis Cruise, of Dublin, in the invention of the endoscope. Above all, at the present time, consider the admirable successes achieved by members of our Section in war surgery and especially in the repair of facial injuries.

It is useless to attempt to conceal the names of those who have accomplished all that is included in this long record of progress. They are indissolubly associated each with its subject, some with more than one. You will find them in any bibliography of our work, especially in those bibliographies in German publications where their ideas are not only acknowledged but taken as the guide to further developments, to which German names are attached and then sent back for the adulation of those who do not trouble to find out where they first came from.

It is for you to consider how all these advantages can be put to the best possible use in the time of reconstruction that is now upon us. Golden Square, as the first of throat hospitals, the scene of the work of the father of British laryngology, will no doubt always remain the Mecca of all interested in our subject, with Gray's Inn Road as the Medina, or place of only slightly less sanctity. But what about all the other hospitals?

A suggestion that has been made to me and which seems to commend itself to workers in other sections is: That the Royal
Society of Medicine should become a centre where any medical woman or man arriving in London, or living here with some time to spare for professional self-improvement, could at once find out how and where he can best employ that time according to his own wishes. We could have here in the office for inspection a more complete diary of forthcoming lectures, demonstrations and operations. In the case of private operations, if such were included, he could communicate with the operator and obtain his permission to be present. This would save the latter from being sometimes inconveniently crowded through his and his patient's generosity and wish that an instructive occasion should not be missed, while it would leave the decision as to the number invited in their hands. An important item in this programme is the improvement of the Journal of Laryngology.

As regards the Royal Society of Medicine, Fellows and Members should do everything in their power to make it widely known that this is the central representative body of British medical science. Persons who give large donations to individual hospitals are not aware of the fact that a similar donation to this Society is equivalent to a donation to every hospital in the country since it advances the science that is the foundation of their usefulness to those for whom they were instituted. The Royal Society of Medicine is eminently worthy of public support and it is to be hoped that now that medical science is becoming more adequately represented in the councils of the nation it will soon receive an annual grant from the public purse to facilitate its beneficial work. In Germany such a body would have been one of the first cares of the State from its inception.

A Tooth-plate impacted in the Oesophagus divided by Irwin Moore's Cutting Shears.

By Somerville Hastings, F.R.C.S.

A. S., a soldier, aged 23, was admitted to the Middlesex Hospital on August 14, 1916, with the history that the same morning he had swallowed a vulcanite tooth-plate. This plate had originally held four upper incisor teeth, but these had been broken off. There were also two partly broken from the back part of the plate. An X-ray photograph showed the denture at the level of the aortic arch.
In the late afternoon, under cocaine anaesthesia, the oesophagoscope was passed and the tooth-plate easily seen. It was impacted across the oesophagus and held so firmly that neither side of it could be moved by forceps. It was therefore divided into two slightly unequal halves by Irwin Moore’s cutting shears. For this two cuts only were required, as the instrument held firmly and did not slip. The smaller half at once slipped down to the stomach; the larger was seized by forceps, but while being disimpacted also slipped down.

The next day both pieces of the denture were seen in the abdomen by X-ray examination, and a few days later they were passed per rectum without difficulty.

Tooth-plate impacted in the Oesophagus for Eight Weeks; Three Unsuccessful Attempts at Removal; Death from Perforation into the Pleural Cavity.

By C. E. Woakes.

Patient, a male, aged 57, presented himself at Charing Cross Hospital on June 10, complaining that, six weeks previously, he had swallowed a portion of an upper denture, composed of platinum with one tooth and two metal hooks attached. There was considerable pain and difficulty in swallowing food and saliva. A skiagram showed that it was impacted in the oesophagus at the level of the aortic arch. On June 11 an oesophagoscopy examination was made, but the denture could neither be seen nor felt. A second attempt on June 13 also failed, although Hill’s expanding tube was used.

Dr. William Hill was next consulted, and he advised that the assistance of Dr. Irwin Moore should be obtained, also that the next attempt should be made with the help of the fluorescent screen.

Acting on these suggestions a third attempt, occupying one and a half hours, was made on June 20. The denture was located and grasped three or four times by its edge with Irwin Moore’s forceps, but so firmly was it fixed that each time extraction was attempted a small piece of the denture broke away in the forceps blades. On one occasion the denture was firmly grasped, but since it could not be loosened, it was considered unadvisable forcibly to extract it and risk tearing the oesophageal wall. The difficulties of extraction were also complicated by considerable bleeding.
Further attempts—e.g., by cutting up the plate—were postponed on account of the patient's condition. Death unfortunately occurred two days later. At the autopsy performed by Dr. Jewesbury, it was found that the denture, which measured $1\frac{1}{2}$ in. in its broadest diameter was embedded by two hooks in the anterior wall, and that there was an ulcerated track through the oesophageal wall into the right pleural cavity, causing general sepsis, pyo-pneumothorax and collapse of the lung. The perforation was not of recent date.

Skiagram exhibited: Specimen of the oesophagus with the denture in situ, also the artificial denture from which the swallowed portion had broken away.
Scarf Pin in the Stomach; Gastroscopy; Expelled by Vomiting.

By C. E. Woakes.

Patient, a girl, aged 8, whilst sucking a scarf pin on September 11, accidentally swallowed it, and was admitted three hours later to hospital. A skiagram showed the foreign body in the stomach, lying obliquely below the level of the seventh right rib, with the head towards the umbilicus and the point directed upwards, towards the right axilla.

An oesophagoscopic tube was passed down to the cardiac orifice, but no foreign body could be seen. Later the tube became blocked with semi-digested cabbage, which rolled up in masses and for the time prevented further examination: during arrangement for further treatment the pin was vomited up. It may be useful in such cases to feed the patient on some heavy, clinging food like boiled cabbage and suet pudding, then pass an oesophagoscopic tube to induce vomiting, with less danger than by administering an emetic. The pin measured 53 mm. (2\frac{1}{4} in.), in length, the diameter of its head being 9 mm., and it might have caused very serious symptoms had it passed further down the alimentary canal.

Pin in Bronchiole of Posterior Lobe of Right Lung; Failure to Remove it by the Bronchoscope; Pin coughed up Eighteen Months later.

By Hunter Tod, F.R.C.S.

This is the further history of a girl, aged 12, shown at the Section on March 2, 1917. Several attempts had been made to remove the pin, but it was out of reach of the bronchoscope. A surgical colleague was anxious to remove the pin by pneumonotomy, but I felt that there was less danger in leaving the girl alone than in letting her incur this grave procedure. Consequently the girl left the hospital.

On January 14, 1918, the girl was re-admitted to the hospital complaining of pain in the right chest, with sudden onset of cough and dyspnea. The patient looked ill; the temperature was 103° F.
respiration 40, pulse 120. On examination there were signs of consolidation of the right base. Bearing in mind the previous history, it was suspected that the pneumonic condition might be the result of the pin still present in the lung. An X-ray photograph showed that the pin had hardly moved. The course of the lung infection was typical of pneumonia, the crisis taking place on the seventh day: after which the patient got rapidly well, and was discharged from the hospital on February 1, 1918, fourteen days after admission.

Four months later (June, 1918) the girl came up to my out-patient department bearing in her hand a pin 35 mm. (1½ in.) in length (which I have with me now) saying that she had coughed it up the night before. In order to confirm this statement another X-ray photograph was taken which showed that the pin had vanished from the lung.

At no time, not even during the period of pneumonia, was there any offensive expectoration suggestive of an abscess of the lung, and all the X-ray photographs confirmed this by their negative results. To what extent was the presence of the pin the cause of the pneumonia? If the pin was indeed the predisposing cause of the pneumonia why did the latter run a typical course with complete resolution instead of leading to a pulmonary abscess?

DISCUSSION.

The President: The pneumonia appears to be incidental and to have no direct relation to the presence of the pin so long before and after.

Dr. William Hill: I think the general advice was to leave it alone. There are always dangers in leaving foreign bodies in the bronchi; more danger, I think, than when they are in the gullet. Here the procedure has been justified by the result, probably because a pin is not a very septic body.

Tooth-plate in Ösophagus; Ösophagoscopy; Removal.

By Hunter Tod, F.R.C.S.

The patient, a sturdy young police constable, came up to the hospital early one morning complaining that during the night he had swallowed his denture, which was a small one, consisting of two upper incisor teeth. Apparently he did it when half asleep and did not realize what had happened. He had a certain amount of pain in the neck and vomited whilst attempting to take his breakfast. I saw him
French: *Dental Plate removed from Oesophagus*

in the out-patient department the same morning and at once had him put under the X-ray screen. The plate was localized at the upper level of the clavicle. The same day he was given an anaesthetic.

On passing the oesophagoscope the plate could be seen lying horizontally across the oesophagus with the teeth pointing forwards. One of the clasps was seized with a pair of forceps, but broke off. This procedure tilted the denture so that it was possible to seize its posterior margin with the forceps. On withdrawing it over the posterior surface of the larynx it projected forwards so that the teeth got fixed in the interarytenoid region. The patient at once got dyspnœic. It was a very uncomfortable moment and I thought I should have to do tracheotomy. Before doing so I took a short, stout pair of forceps and, getting on to a stool so as to be well above the level of the patient, I passed the forefinger of the right hand down until it could feel the plate, and with the forceps in the left hand got a firm grip of one of the teeth and pulled the denture out. The patient made a complete recovery.

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**Two Cases of Dental Plates removed from the Oesophagus.**

By J. Gay French, F.R.C.S.

The patient, a man aged 50, was knocked down at 1 p.m. on March 28, 1915, and was taken to the Hornsey Cottage Hospital in a dazed condition. He was put to bed, and in a couple of hours recovered his mental powers. It was then found that his dental plate was missing, and he was observed to bring up some blood-stained sputum. He, however, complained of no discomfort. It was thought that the plate must have slipped down, and he was therefore X-rayed. This showed the dental plate to be fixed in the oesophagus, just below the cricoid.

At eight o’clock the same evening the man was given an anaesthetic (chloroform) and I passed down an oesophagoscope. A small portion of the dental plate was then found to be showing just above the cricoid cartilage. This was seized with a pair of Brünings’ forceps, but was found to be firmly fixed. The cricoid was gently pushed forwards and, after considerable manipulation, the plate was freed and removed whole. The man left the hospital two days later, quite recovered.

X-ray plate and denture shown.
Summary of Case of Foreign Body in Oesophagus, occurring at a Royal Naval Base Hospital.

By J. Gay French, F.R.C.S.

The patient, a young deck hand, R.N.R.(T.), aged 22, awoke suddenly during the night of November 30, 1917, with a feeling of suffocation and alarm. He at once missed his dental plate, which carried two teeth, the central and lateral incisor of the left side, upper jaw. Contrary to his usual practice, he had gone to his hammock with the plate in situ. He awoke at about 4 a.m. and started vomiting food in small quantities, and, later, could only breathe with difficulty—apparently from spasm. Attention was then drawn to the absence of his false teeth, and he was brought ashore at 10 a.m. for medical aid.

On entry into the hospital patient was suffering from shock and had a pulse of 120, was pallid, and had a rather anxious face. Voice rather weak and husky; temperature 99.4° F. On examination, digital pressure over the anterior triangles of the neck, in a lateral direction, produced a pricking sensation on the left side of the “throat.” This was found to have been caused by one of the lateral hooks with which the plate was fitted. X-ray examination with the screen revealed the plate lying low down in the oesophagus, opposite the intervertebral disk between the last cervical and first dorsal vertebrae. The teeth were lying on the left side, the hooks on the right side, viewed from in front, the patient lying on his back. The shadow moved “up and down” with deglutition, and could be seen to move “with” the oesophagus on lateral pressure applied from the neck, being obviously wedged in position. No food could be taken, not even a drop of fluid, as it at once produced reflex spasm and retching.

I went down to the Royal Naval hospital on Sunday, December 2, when the man was given an anaesthetic, consisting of intravenous saline and ether. On passing down the oesophagoscope I found the plate fixed very low down; the mucous membrane was considerably inflamed and oedematous. I found that it was quite impossible to move the plate, and therefore passed down one of Dr. Irwin Moore’s shears, clipping off the side of the plate. This enabled one of the catches to be turned round, and the plate was then removed, a small portion slipping down into the stomach.
At the time of operation the man had a temperature of 102° F.; this dropped the same evening, but on the following morning was again 102° F., when he was given an injection of atropine sulphate. The temperature subsequently dropped, and the man made an uninterrupted recovery. After the removal of the plate the patient was fed rectally for twenty-four hours, and then by the mouth, small amounts of fluid only being administered.

X-ray photograph and denture shown.

Foreign Bodies impacted in the Food and Respiratory Passages recorded at the Section of Laryngology of the Royal Society of Medicine since 1938.¹

By Irwin Moore, M.B.

The total number is 111, made up of three in the pharynx, sixty in the oesophagus, one in the stomach, and forty-seven in the respiratory passages.

(I) Food Passages.

Of the three in the pharynx, one was extracted by oesophagoscopy, and two by suspension laryngoscopy. There were no deaths.

Fifty-three of the sixty patients who had foreign bodies in the oesophagus recovered, and seven died.

In the fifty-three recoveries, the foreign body was successfully removed by oesophagoscopy in forty-seven cases, in five it was released by the passage of the oesophagoscope (two being vomited up—one through the oesophagoscopy tube and the other after removal of the tube, whilst three were later evacuated per anum). In only one case was the foreign body removed by oesophagotomy.

Two of the seven deaths were due to injury caused by the previous blind use of the bougie, two to previous ulceration and perforation into the trachea, and one to previous abscess and perforation into the mediastinum. In these five cases the foreign body was extracted before death. There was one case where the foreign body was not removed, the patient dying as a result of previous ulceration and perforation into the pleural cavity. There was only one failure to find by

So we may say that out of the total sixty cases of foreign bodies impacted in the oesophagus, fifty-eight were either removed by means of, or as the result of, the employment of the oesophagoscope, and that no death or failure could be attributed to its use—a very creditable record. The only foreign body reported in the stomach is that of the scarf pin shown at this meeting. It was spontaneously expelled after the first introduction and removal of the oesophagoscope.

(II) Respiratory Passages.

In eight cases of a foreign body in the larynx, six of them were extracted by direct laryngoscopy, and one by suspension laryngoscopy. In one case which had been diagnosed and treated for diphtheria a collar stud was found post mortem impacted in the larynx.

Of three foreign bodies in the trachea, two were extracted by peroral tracheoscopy, and one was removed through a tracheotomy incision. There were no deaths.

Of thirty-six foreign bodies in the bronchi, twenty-nine were extracted by peroral bronchoscopy and only two by tracheo-bronchoscopy. Three were spontaneously coughed up (one through the bronchoscopic tube while in situ, one following operation for pulmonary abscess and empyema, and one two years after failure to find by bronchoscopy although located by X-rays). In one case thoracotomy was successfully performed after failure to find by bronchoscopy though located by X-rays. There were no deaths. One autopsy was reported where a foreign body was discovered, after death from empyema and gangrene, but this case was not referred during life to a specialist (endoscopist).

These remarkable results show the splendid work which has been achieved in this country by British endoscopists.

In connexion with Mr. Hunter Tod’s remarkable case, I have collected the following similar cases:

Some Statistics and Results of Pins Accidentally Inhaled into the Lungs.

(These include only ordinary and glass-headed pins, and not safety pins.) Fifteen of these cases have been reported by Chevalier Jackson

(Philadelpbia); three by Fletcher Ingals (Chicago); one by Costa (Madrid); and eight by present members of the Section of Laryngology of the Royal Society of Medicine.

Of these twenty-seven pins recorded, ten were definitely stated to have entered the right lung, and sixteen the left—i.e., nearly two-thirds were found in the left lung.

Sixteen were situated in the bronchi of the upper or middle lobes, and were all extracted without difficulty by bronchoscopy, whilst eleven were in lower lobe branches. Of these, only four were successfully extracted, while seven could not be found by bronchoscopy, although located by X-rays.

The seven failures to find were all in posterior branches of the lower lobes (three on the right side, and four on the left). Of these, five recovered and two died.

Of the five recoveries, thoracotomy was performed in two cases, the foreign body was coughed up in one case, the pin after a time disappeared in one case, whilst in the fifth case, when last reported, the pin was gradually working its way towards the periphery.

One patient died after thoracotomy, and the other of pulmonary abscess one and a half years later, the patient having refused operation.

It will thus be seen that out of the total twenty-seven cases, the foreign body was successfully removed by bronchoscopy in twenty cases, and in only one case was a pin spontaneously coughed up (Hunter Tod’s case).

Postscript.—Since summarizing these cases I have come across a case reported in 1886 by Colquhoun (New Zealand) where a pin 1¾ in. in length was coughed up after sixteen years’ sojourn in the right lung. It had been inhaled at 5 years of age, and had caused no symptom for fifteen years, when inflammation of the lungs occurred, followed by acute phthisis. A few days before death the pin was coughed up in three pieces much eroded and very brittle. No autopsy was held, hence the actual position of the pin was not ascertained. Botella’s


Section of Laryngology

(Madrid) has also reported an interesting case of a pin in the base of the left lung. All attempts both by peroral- and tracheo-bronchoscopy failed to locate it. A strong electro-magnet was also used. The final result of this case is not recorded.

DISCUSSION.

Mr. HERBERT TILLEY: In my earlier cases I found difficulty in removing foreign bodies because I used too narrow a tube. We ought to employ the largest tube which it is possible to insert with safety into the oesophagus. When we remember the size of the boluses of food that are passed, we may conclude there is no inherent danger in the passing of a large tube. In the earlier days of endoscopy it was thought that the smaller the tube the easier it was to pass, and the less the danger attending its passage. Further, those of you who have not had an extensive experience in direct vision instruments should not try to pass the oesophageal tube immediately behind the arytaenoids, because that will mean disappointment for yourself and possible harm to the patient if it is persisted in. The correct method is to pass the end of the tube first into the pyriform fossa, and then sweep it into the middle line, where it will enter easily into the gullet.

Dr. D. R. PATerson: If the foreign body is in the upper third of the oesophagus, or immediately below the cricoid, it can be treated in a different manner to a foreign body lower down. If further down it is practically outside the domain of external operation. I have been present at an attempt to remove a tooth-plate which had long been impacted behind the cardiac area, where, after he had got to it, the surgeon found he could not remove it on account of the dense cicatricial tissue in which it was embedded. In the case of foreign bodies which are immediately below the cricoid, we ought not to forget that, under certain conditions, it may be impossible to remove them, or at all events that there is some danger in making the attempt. We should then consider the external method. That is impressed upon my mind particularly by two cases occurring during the past eighteen months. From one of the cases I showed the specimen here—a bone of considerable size with a very sharp corner which had become impacted below the cricoid. It had been there three or four days when I saw the patient, and the swelling was great. We used Dr. Irwin Moore’s shears, but it was difficult to get a hold. I take it the instrument needs some counter-resistance behind it in order to get the blades to bear on the foreign body. The man had a very short and thick neck, and it was unadvisable to do an external operation. At the risk of damaging the structures, it was extracted after disengaging the sharp corner, but septic trouble ensued after a few days. Eight months later I had another and similar case, the foreign body being a broken vulcanite tooth-plate which had become impacted four days before. Pressure on the gullet appeared to be considerable, as there was much swelling, and, what I always regard with suspicion, a
distinct odour, which I take to indicate some ulceration. I made a very cautious effort to cut the plate, but was afraid to apply much pressure. As this patient had a long thin neck, I had no hesitation in opening from the outside. When I had cleared the oesophagus and put my finger down to locate the foreign body, the wall perforated: the sharp point of the body had worn the gullet through to such an extent that even slight pressure produced the rupture. The man did well. I call your attention to the two different kinds of neck. In the case of the man with the short thick neck, it may be a very dangerous operation, whereas in the case of a thin neck it is a reasonably safe one. Of course, the character of the foreign body has much to do with one’s decision as to what to attempt, as has also the length of time it has been impacted; but in particular one has to take note of the amount of swelling, and whether or not there is ulceration. I remember the peculiar odour, specially in the case of a soldier from whom I removed a piece of bully beef tin the size of half-a-crown: the pressure of the body on the gullet wall alone had caused considerable ulceration, as no attempt at extraction had been made.

Dr. W. HILL: In many of the cases the ulceration is due to the Bacillus coli communis. In one case a denture had become lodged, and I could not get it out on account of the hooks. The body had been there some days, and there was an odour in the neck, and the tract was blackish-green. The patient died of pleuro-pneumonia following mediastinitis due to the perforations which had taken place beforehand. I tried to remove the denture by the direct method, but failed, and then I resorted to external oesophagotomy, regarding it as a point of honour to remove foreign bodies in a situation in which they may be dangerous. It was below the level of the clavicle, but I was surprised how easy an operation it was.

Sir STCLAIR THOMSON: Mr. Hunter Tod’s cases are important. There has been a little undue tendency to try to remove at once every foreign body. We must take into account the nature of the body, whether it is a metallic substance like a pin or a tin tack. We remember Chevalier Jackson’s case, for instance, in which a woman, in order to gain admission into the wards and elicit sympathy, used to pass tacks into her mouth and inspire them into her bronchi. And she never died of it! I show you a foreign body which had a long sojourn in the oesophagus for two and a half years, and yet the patient was fairly well. Sometimes in trying to take an article like a penny from a child’s gullet we are apt to dislodge it and it descends into the stomach. Therefore if we are nervous about bringing a foreign body up from the oesophagus, we may remember that it may be wise to pass it downwards. And, thirdly, as in Mr. Tod’s case, when we cannot remove a foreign body we should not give up hope.

Dr. IRWIN MOORE: This is a most unique collection of cases of impacted foreign bodies shown here to-day. After searching the literature I find that Mr. Somerville Hastings’ case is the first recorded where a tooth-plate has been
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cut in half in the oesophagus with any cutting instrument. Killian\(^1\) reported in 1900, a case in which he separated a vulcanite tooth-plate into three pieces by burning with a specially devised cautery knife, but this was a most dangerous procedure. Mr. Hunter Tod’s case is a unique one, for Chevalier Jackson says that although a few foreign bodies may be coughed up from a lower lobe bronchus, yet it is practically impossible for a pin to be coughed up, and the five cases of failure to locate by bronchoscopy to which that authority refers, were, as in this case, pins in a similar situation—i.e., in a posterior branch of a lower lobe bronchus. I have collected (p. 14) the statistics of foreign bodies which have been removed by members, and reported at meetings of this Section since 1908. Though they do not by any means represent the total number of successful cases in this country, they undoubtedly consist of the most serious cases referred to the specialist, and present many points of great interest.

Mr. Somerville Hastings: Dr. Paterson said he found that Dr. Irwin Moore’s forceps frequently slipped in his hands. That was not my experience in the case I have recorded to-day. The notches on the blades seemed to catch on the tooth-plate, and the shears cut very easily. I have tried the shears with other objects and I have not had any difficulty owing to the shears slipping.

[Dr. Paterson: I was referring to them slipping on a round smooth piece of bone.]

Dr. Irwin Moore (in reply): In reference to the cutting shears, they do not require resistance behind them, for they are designed to draw up the tooth-plate as they cut. But the great point in using them is to twist slightly to the left whilst cutting. Again, if possible a tooth-plate should not be cut through the centre, which is the thickest part, but at the periphery. The first attempt to cut through an impacted tooth-plate with these shears was made by Dr. Peters\(^2\) in 1912. The first cut disimpacted the tooth-plate and held it so firmly that he was able to withdraw the denture without further cutting. The denture showed that if the cut had been completed, the widest diameter of the plate would have been reduced by ½ in., and it would have been easy to turn it into its narrowest diameter, but owing to the secure grasp of the shears this was not found necessary for its safe removal.

\(^{1}\) Deutsch. med. Wochenschr., December 20, 1900.

Demonstration of some New Instruments recently designed for the Removal of Foreign Bodies from the Lungs by Peroral Endoscopy.

By Irwin Moore, M.B.

(1) Universal Non-slipping Forceps.—These are applicable to all types of foreign bodies. They are a facsimile of the exhibitor’s oesophageal forceps but adapted for lung work by reducing the diameter of the shaft and blades.

(2) Bronchial Dilating Forceps.—These were specially devised for difficult cases of a foreign body tightly impacted in a bronchus. With them a bronchus may be dilated whilst the blades of the forceps are passed downwards and around the foreign body so as to grasp it securely. They are invaluable for round objects, e.g., glass beads and fruit stones, also for those which are cube shaped and for this reason difficult to seize. On account of their shape the blades cannot be approximated much nearer than the diameter of the foreign body, hence breaking it up into fragments is avoided. They are therefore specially useful for friable bodies—e.g., swollen beans or oval seeds, or partially disintegrated bodies.

(3) Ring Forceps.—These are suitable for teeth inhaled with their fang directed downwards and then impacted, e.g., incisors or canines. These are adapted from Cuthbert Morton’s modification of one of Killian’s forceps.

(4) Single Curette Forceps.—These may be passed down between an impacted foreign body and the bronchial wall, so disimpacting and retrieving it from behind forwards. These are adapted from Quer’s aural curette.

(5) Curettes of Aural Type for the same purpose.

(6) Hooks.—These are of three patterns, and are designed to take the place of those dangerous examples supplied with the present endoscopic outfit, which may get caught in the bronchi.
Sarcoma of Maxillary Antrum; Lateral Rhinotomy; Recurrence in Glands; Radium Treatment.

By IRWIN MOORE, M.B.

Further notes of a female patient, aged 50, upon whom lateral rhinotomy was performed for sarcoma of the right antrum. Previously shown at a meeting of the Section on November 3, 1916, when the full history was reported.


I am indebted to Dr. Lynham, Radium Institute, for the following later notes of the case:

"Lateral rhinotomy was performed on September 18, 1916, by Dr. Irwin Moore, for removal of a round-celled sarcoma from the right antrum. The growth had arisen apparently from the ethmoid, and had almost filled the antrum, penetrating its bony wall, and extending into the soft parts of the cheek, but not invading the skin. The floor of the orbit was intact, and proptosis was attributed to upward pressure of the floor, and not to any invasion of the orbit. The growth was thoroughly removed, together with the ethmoid cells and a portion of the muscular tissue of the cheek. The section showed round cells with a fibrous stroma.

"In July, 1917, the patient noticed some swelling in the parotid region, and this has slowly increased, without any pain.

"At date (March 19, 1918) the patient looks healthy. The operation cicatrix is scarcely noticeable. There is no sign of active disease in the nose, cheek, or zygomatic or temporal fossae. There is some degree of proptosis of the right eye. In front of the right ear is a smooth oval tumour, 4·3 cm. by 5 cm., firm, fixed, not adherent to skin; not tender or causing pain. A shot-sized gland is felt at the right extremity of the hyoid. Treatment by radium carried out on four successive days, March 20 to 23 inclusive; applicators containing 160 mgr., and

Irwin Moore: *Sarcoma of Maxillary Antrum*

**Fig. 1.***

Sarcoma of the right maxillary antrum recurring in the pre-auricular gland and orbit. Before radium treatment.

**Fig. 2.***

The same—showing disappearance of the pre-auricular tumour after radium treatment.
screened with 1\(\frac{3}{5}\) mm. of lead, being applied for sixteen and a half hours, distributed over four consecutive days.

"April 10, 1918: The tumour has subsided, leaving the pre-auricular gland larger than a pea with a trace of diffuse infiltration round it. Treatment carried out on four successive days, April 10 to 13 inclusive, on the same lines as previously: applicators containing 100 mgr., and screened in similar fashion, being applied for twelve hours, distributed over four consecutive days.

"July 16, 1918: The gland is now shot-size, but still palpable. Patient recently has had further diplopia. There is some induration above the right inner canthus firmly adherent to the side of the nose, and extending up to the margin of the orbit above the canthus. There is a second hard, movable, almond-sized mass felt above the right eye, between globe and orbit, pressing slightly on the globe. The eye seems a trifle more prominent, but the photograph taken in March does not confirm this. Treatment carried out on five successive days, July 22 to 26 inclusive; applicators containing 75 mgr. being applied for twenty-five hours on the same lines as on the previous occasion, and distributed over five consecutive days."

Patient reports, August, 1918: "The lump over the top of eye has entirely gone, and the one at the side is only very slight now. The double sight has quite gone, and the sight is much clearer."

The patient is shown to demonstrate the great advantages of radium treatment in cases of recurring sarcoma.

Photographs showing the distension of the antrum and distortion of the face previous to the operation were published in the Proceedings, 1917, x, p. 29. Photographs showing the recurrence in the pre-auricular gland and disappearance after radium treatment are now exhibited (figs. 1 and 2).

DISCUSSION.

Mr. Herbert Tilley: I wish members would try opening the antrum underneath the cheek in these cases. The incision should reach from the malar process to the median line: turn up the soft parts, open the canine fossa, separate the soft tissues from the vestibular aspect of the ascending process of the superior maxilla, and remove that structure with strong bone forceps. That gives at once a large opening into the antrum: you can see the ethmoid region, and even the sphenoidal sinus. Only those who have tried this method can have any idea of the extraordinarily good field of operation which it affords. At the close of the operation the soft parts fall into position, two or three stitches are inserted, and rapid healing occurs. If my colleagues
Irwin Moore: *Carcinoma of Maxillary Antrum*

will try the method I do not think they will revert to the external operation of lateral rhinotomy.

Mr. W. Stuart-Low: Two years ago I showed some cases here in respect of which I contended that the antral route was much the best. With the patient well above you and using a good light you can see the whole area of operation. Take the whole of the inner wall of the antrum away, and, if necessary, remove the ascending process of the superior maxilla. You can remove everything to the base of the skull if required by that method. There has been no recurrence in my cases so treated. It is a complete operation, and leaves no outside mark or scar. I have operated by this method in cases of epithelioma and sarcoma of the maxillary antrum and in similar conditions of the nose where the antrum has become affected.

Mr. W. M. Mollison: Surely Mr. Tilley does not intend to recommend the antral route for carcinoma? It is in many cases not merely a matter of taking away the growth and bone, subcutaneous tissues must often be removed as well. In the cases of carcinoma upon which I have operated, it would have been impossible by this means (the antral route) to have removed growth far enough back. Of the three sarcomata upon which I have operated one might have been done in that way, but the others might have been torn. By the external operation the growth could be shelled out without damaging it at all.

Mr. Tilley (in reply): A growth which has penetrated the bony walls and infiltrated the soft tissues would not be suitable for the method I advocate. I recommend it for a localized growth in the antrum, where the bony walls are intact and where the symptoms point to malignancy. It is especially suitable for growths involving the inner antral wall or the neighbouring ethmoidal region.

Dr. Irwin Moore: Can anything more be done for this patient, because since I saw her in August she has been complaining of terrible headaches, and says her sight is deteriorating. Dr. George W. Thompson, who kindly examined her for me, reports that there is pressure on the right optic nerve, and I think there is no doubt that this is due to some recurrence in the ethmoid region on the right side.

Carcinoma of the Maxillary Antrum; Lateral Rhinotomy; Recurrence.

By Irwin Moore, M.B.

Specimen (right side of face) of a female, aged 62, upon whom lateral rhinotomy was performed for epithelioma of the right maxillary antrum in November, 1916. Patient was shown at the meeting of this
Section on February 2, 1917, three months after operation, to show the satisfactory results obtained; the face wound had healed in five days, and everything appeared to be going on well.

Recurrence was observed three months later, rapidly extending from the zygomatic fossa to the orbit, necessitating removal of the eye.

Patient died in November, 1917, one year after the first operation.

Post-mortem.—The growth was found to have spread, involving the remaining portions of the antral and orbital walls, and extending backwards into the sphenoidal sinus. The side of the cheek had sloughed away and left a large cavity.

These two cases operated upon at the same time, and kept under observation side by side, are interesting as illustrating the greater degree of malignancy of epithelioma in this situation as compared with small-celled sarcoma.

Photographs of the patient three months after operation are also shown.

Case of Incipient Singer's Nodules in a Vocalist.

By J. Dundas Grant, M.D.

The patient, a young lady student of singing, complained of huskiness, of about a year's duration. At the junction of the anterior and middle third of each vocal cord was a minute projection; during phonation the vocal slit was thus divided into an anterior and posterior segment and on separation of the cords a small string of mucus stretched from one nodule to the other. The voice could not be carried above the middle "C," on which the transition usually takes place between the thick and the thin register. She has been instructed how to practise the "pmawing" exercises devised by Curtis. It is anticipated that as the nodules are at the very earliest possible stage in development they will subside.

DISCUSSION.

Mr. W. M. Mollison: The inability to sing certain notes is possibly connected with a very slight chronic affection from above, such as post-nasal catarrh. There are septic tonsils and glands on both sides of the neck and

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it might be worth while enucleating the tonsils and exploring the sinuses. I saw a slight irregularity of a cord on one side, but that may have been due to mucus.

Dr. Grant (in reply): It was my intention to draw attention to the condition of the upper air passages and tonsils, because much septic material can be squeezed out of the tonsils, and these should be treated in addition to the rest of the voice and the exercises devised by Holbrook Curtis, which are very well known.

Later Note.—The projections are already less prominent and the string of mucus is much diminished. She can sing with much greater ease and can already produce a number of notes above the middle "C" without difficulty. —D. G.

Method of inducing Cough and Expectoration by the Inhalation of Oleum Sinapis.

By J. Dundas Grant, M.D.

This is a useful method of getting a little expectoration at the time of examining the patient. I have used it in the out-patient department of the Hospital for Consumption, Brompton, and in private practice. It is valuable in the case of malingerers suspected of bringing tuberculous sputum from other persons. A few drops of aromatic oil of mustard are placed in an empty six-ounce bottle where they volatilize. The patient sniffs this from the neck of the bottle: he soon begins to cough, and will in many instances bring up expectoration which, otherwise, it would be difficult to procure at the time.

Sarcoma (?) of the Left Tonsil.

By Andrew Wylie, M.D.

Patient, female, aged 56, complains of a swelling in the left tonsil, growing slowly for seven years. There is no pain, no real difficulty in swallowing or breathing. It is fairly movable, elastic to the finger. The soft palate is slightly adherent to it. This swelling was punctured several times, and found to be of a solid nature. No improvement with potassium iodide. Slight, but not definite enlargement of cervical glands. Exhibitor considers that the growth can be removed fairly easily, and the exact diagnosis made by a pathologist.
DISCUSSION.

Dr. H. J. BANKS-DAVIS: I think there is pus in the palate as well. These cases often get it, and when an incision is made to release it from the palate the growth fungates through the incision, making subsequent operation more difficult.

Dr. W. HILL: If it is sarcoma, it is a very good case for the application of radium. If it is a fibrous tumour it should be shelled out.

Dr. GRANT: This may be a "mixed" tumour between the layers of the palate, such as can sometimes be shelled out. If it were sarcoma, probably the patient would have enlarged glands. If, however, it turns out to be sarcoma, the opening made for the scooping out would do for the introduction of radium tubes.

Mr. O'MALLEY: As stated by Dr. Grant, this probably originated in the layers of the palate as an adenoma. I have shown two cases of that type, but the condition had not progressed to the extent here seen. Owing to the length of time it has been growing, it has depressed the tonsil. When you get the patient to open her mouth, the mass beneath the jaw disappears, but when the mouth is closed, the mass can be felt. There does not appear to be involvement of glands outside.

Mr. HOWARTH: I had a similar-looking case two years ago which turned out to be an endothelioma. It was more extensive than at first appeared and it extended along the internal pterygoid plate to the base of the tongue. As a block dissection of the glands was being done I was able to deal with it bi-manually.

Dr. JOBSON HORNE: The growth is probably more extensive than it appears to be when viewed from the mouth. I do not think it would be such a simple matter to remove it as stated in the notes of the case.

Dr. WYLIE: (in reply): I brought the case to show the Section before I tried to enucleate the growth, which is solid, not a cyst, as I punctured it to find any fluid or pus.

Three Dental Plates removed from Æsophagus.

By A. BROWN KELLY, M.D.

These plates were extracted through the mouth. In two other cases the plate slipped into the stomach and was passed \textit{per rectum}, and in another it was removed by Æsophagotomy. In each of the six cases the patient was a young man, the plate was swallowed during sleep and became impacted in the upper part of the gullet, and recovery was uneventful.
Absorption of the Pre-maxilla in Tertiary Syphilis of Nose.

By Dan McKenzie, M.D.

The patient is a male who came to hospital six months ago because of the "shrinking of his jaw." The history was that two or three months previously the gums about the upper incisors began to shrink, and these teeth loosened and dropped out. After they had been shed the shrinking still went on until none of the gum was left. He was unaware of any disease in the nose.

Examination bears out the patient's complaint. Not only is the alveolar process but also the whole of the anterior arch of the palatal processes of the superior maxilla considerably reduced in size, the mucous membrane and submucous tissues of the gums being thrown into ridges and folds through the shrinking of the underlying bone. There is no ulceration in this part of the mouth, but high up in the gingivio-labial recess in the left canine fossa there is definite firm infiltration with a fissure running through it. The incisors of the lower jaw project beyond the shrunken upper jaw by about an inch, and the upper lip has fallen in, giving to the patient's face an unsightly underhung appearance.

The nose is the seat of active tertiary syphilis. In the region of the bony septum and floor of the nose there is a sequestrum embedded in densely infiltrated tissue showing a nodular surface. When he first came to us this piece of dead bone was still quite firm, but it is now beginning to loosen, and I anticipate being able ere long to remove it. At first sight the bony absorption, coupled with the nodular or "tubercular" characters of the infiltration in the nose brought to mind the possibility of leprosy. But the patient has lived in England all his life; the Wassermann reaction is positive; and microscopic examination of the nodular infiltration in the nose shows dense fibrous and granulomatous characters with the vascular changes of syphilis. Presumably, therefore, the absorption of bone in the pre-maxillary region is due to destruction of the nasopalatine nerve, and also perhaps of the dental branches of the superior maxillary nerve, from involvement in the extensive infiltration.

The patient is receiving vigorous anti-syphilitic treatment (novarsenobillon, mercury and potassium iodide).
Dr. W. Hill: I have had two cases in which the pre-maxilla gave trouble. In one it came away as a sequestrum, in a case of congenital syphilis. In the other case I did a Rouge's operation in order to get away a sequestrum from the antrum. But Rouge's is a bad operation, especially in a syphilitic patient, and this was a boy, aged 16. The last stage of that case was worse than the first. I got the sequestrum up, but half the pre-maxilla came away; there was a hole from the lip into the nasal cavity, and there was more depression in the nose than when I started operating. That was many years ago.

Case of Mutism of Ten Months' Duration.

By J. Dundas Grant, M.D.

The patient, a soldier, aged 20, was blown over by a shell on December 30, 1917, when in France, while making an attack. He was unconscious at first, but does not remember for how long. When he recovered consciousness he was voiceless and speechless. He went first to a hospital at the base, then to one in London, and then to another military hospital, and from there went to the West End Hospital on October 29 of this year. I saw him two days later, and on laryngoscopic examination found the vocal cords separated to the utmost possible extent. By means of the application of faradism to the neck, and of persuasion, his voice was restored, though his speech remained stammering.

[Later Note.—On November 5 the stammering was greatly subdued by the prolonged application of faradism to the sub-mental muscles, and when seen on the 7th he could speak quite well.]

Case of Functional Aphonia of Three Months' Duration.

By J. Dundas Grant, M.D.

The patient, a soldier, aged 29, stated that he was "gassed" in July, 1918. He went straight to the Casualty Clearing Station and when there his voice got weak, and after two days it quite disappeared till I saw him yesterday (October 31), when the voice was completely restored by laryngoscopy and a very mild application of faradism to the neck.
Case of Functional Aphonia of Ten Months' Duration, with Laryngitis.

By J. DUNDAS GRANT, M.D.

The patient, a soldier, lost his voice suddenly on January 26, 1918, when in Italy. A bomb fell outside his billet in the night. He was restless and lost his power of speech. On February 1 his temperature rose to 104° F., and went up and down for a fortnight owing to an attack of broncho-pneumonia. He was in the Casualty Clearing Station for three weeks, and during that time was so weak that he had to be fed with a spoon. He was then sent to a general hospital, and had inhalations for two months; he was next sent to another hospital in France, where he remained for six weeks; he was sent back here in May, and has been in a London military hospital ever since, with the exception of a fortnight in a V.A.D. hospital at Willesden. I saw him for the first time at the West End Hospital for Nervous Diseases on October 31.

The vocal cords are pink, shiny and swollen, and do not approximate in their interarytenoid portions, though they do in the middle third, where they are somewhat "bellied." His muscles are weak and flabby, and in view of the possibility of his being a subject of tuberculosis I have postponed any energetic treatment for the functional aphonia till his sputum has been examined for bacilli.

[Later Note.—I have to report that I saw him again on November 5, when the vocal cords had lost their tumidity; his voice had returned shortly before my expected visit; this was probably due to the persuasive effect of the electrical and other suggestive treatment on his companions. There were no tubercle bacilli found in the sputum.]

DISCUSSION.

Dr. Andrew Wylie: If Dr. Grant were to remove the tonsils and adenoids in two at least of the patients, even if the operation did not cure the aphonia, the moral effect would do so.

Mr. O'Malley: If these tonsils are removed the patients will probably talk very well on recovering from the anaesthetic.
The President: In one of the cases the vocal cords are still very red from congestion.

Mr. W. M. Mollison: All functional cases recover by suggestion. Three days ago I saw one of these cases of functional aphony: he had been dysphonic since February, and after ten minutes of suggestion he talked very well. His cords were red, but when I saw him again two days later the redness had already decreased. The redness of the cords in these functional cases is due to the patients having used their voice wrongly.

Dr. Smurthwaite: To treat all these cases of aphony as if they were functional is to invite serious mistakes, as many of them turn out to be tubercular. Out of 400 cases in the last three years, I have selected fifteen which were tubercular. To treat even suspected functional cases without looking into the larynx is madness. I agree that functional cases can be cured by psychic treatment. I always look into my patients’ larynges and satisfy myself that I have to deal with a functional case before I start my intensive suggestive process: in purely functional cases there is no reason why the patient should not speak before he goes out of the room.

Mr. O’Malley: All my treatment in these cases consists of a method of suggestion I have already described. With regard to not examining an aphonie case before treating it by suggestion, I will give an instance of a striking pitfall in a case which was sent to me with the note that already the patient had had three injections of salvarsan and asking whether there was a serious lesion in the throat to prevent him speaking. After two or three friction movements of the laryngeal mirror he spoke well. It was a case of simple aphony.

Dr. Perry Goldsmith: A man may have aphony as a symptom of some other disease. If you believe a case to be functional, you must make the patient realize that you have faith in the process, and that he must have faith in it too. And to carry out the method you must have him by himself: you will not get a good result if you try it in a spectacular way, for all the ward to see.

Dr. Grant (in reply): I have not had a case of functional aphony under my care which has not been cured. The cases I have brought to-day present different points of interest. One, a healthy-looking man, had been aphonie for some months, and yesterday, after a few minutes, I had him talking perfectly well. A more unusual case is the one of complete mutism, in which the cords were widely abducted. He is now at the stage of stuttering, a stage often resulting immediately after restoration of the voice. The third case is the most interesting of all, because the vocal cords are thickened and red and bellied, and the condition has lasted some fourteen months. The man’s musculature is particularly soft, probably there is some wasting. He had
suffered from broncho-pneumonia, therefore I made him expectorate a little. Until I have excluded tuberculosis, I shall refrain from attempting to hustle him into restoration of voice (vide later notes appended to description of cases, pp. 29, 30).
DISCUSSION ON DILATATION OF THE ŒSOPHAGUS WITHOUT ANATOMICAL STENOSIS.

DR. WILLIAM HILL.

Diffuse dilatation of the œsophagus without anatomic organic narrowing in the region of the cardia is usually in its early stages, at all events, of the spindle-shaped type, though later lateral lobulations may develop, especially near the phrenic level where there is sometimes an abrupt obtuse termination of the dilatation. The ectasia is in most cases limited to the thoracic gullet, though in very advanced ones the cervical œsophagus, the pharyngo-œsophageal orifice, and even the lower pharynx are much enlarged. In a few observed cases some elongation of the gullet has been demonstrated in addition to great increase in calibre, the viscus then taking a sigmoid course in the chest. There is stasis of ingesta due to its passage being distinctly impeded at the level of the diaphragm, but when the dilated gullet in these cases is removed and examined post mortem the lumen of the phreno-cardiac segment is found to be not subnormal, and in certain and rare cases, to be alluded to later, it may even here be actually enlarged. We have to do generally, therefore, with a normal potential lumen of the gullet at the phrenic level, and immediately below it, in combination with a functional stenosis and arrest of food at the phrenic level, and ectasia above.

In some instances the cause of this stenosis is obvious—e.g., in cases of either hernia or of eventration of the diaphragm in which the stomach

1 At a meeting of the Section, held December 6, 1918.
assumes a thoracic position considerably higher up than normal under the left ribs, thus producing angulation or kinking of the subphrenic oesophagus.

Dilatation of the whole of the gullet, including the phreno-cardiac portion, has exceptionally been observed in cases of hour-glass constriction of the stomach and in marked pyloric stenosis of long standing. This latter group, like the previous group—viz., that due to upward displacement of the stomach—can be differentiated by the aid of the X-rays from the ordinary type of dilated thoracic oesophagus in which there is neither external pressure nor angulation nor intrinsic anatomic narrowing of the lumen below, and in which the inability of the food to pass freely through a potentially normal lumen appears to justify the term "functional stenosis." The later condition presents a constant X-ray picture as the bismuth meal in well-marked cases of oesophagectasia is seen arrested at the phrenic level, not passing immediately into the stomach. The same picture is, however, observed in fibrous stricture and other forms of true anatomic stenosis of the phreno-cardiac gullet and the differential diagnosis can only be certainly made in all cases by endo-oesophageal inspection of the phreno-cardiac region by means of large-sized endoscopic tubes. I employ an oesophagoscope 18 mm. in diameter for choice, and if such tubes can be passed into the stomach there is no anatomic stricture. Blind bougieing has been much relied on in the past in order to ascertain the non-anatomic character of the stenosis; and when a large bougie passes readily into the stomach it is strong presumptive evidence in favour. But a sense of stricture is sometimes obtained due to spasmodic gripping of the bougie behind the cricoid and so leads to error. When a soft rubber hollow bougie of large calibre filled with mercury is seen with the aid of the X-ray screen to pass without impediment into the stomach this test can be relied on as pointing to the absence of an organic stricture; but if the gullet presents a truncated and lobulated dilatation at the phrenic level the nose of the bougie may fail to hit off the entrance to the phreno-cardiac gullet; the test then fails to differentiate between a functional and an anatomic organic stricture in this region, so that the superiority of the oesophagosopic method as a certain method of diagnosis and applicable to all cases is evident.

Most physicians, surgeons, and radiologists, even at the present day, speak of the site of the stenosis being at the cardia; we, however, of course know that the X-ray findings show that the arrest of the bismuth is at the level of the diaphragm. Chevalier Jackson was, I think, the
first to call attention to the correct level, and he discarded Mikulicz's term "cardiospasm" as applied to these cases and substituted that of "phrenospasm" in the first edition of his book. Cardiospasm is a term that was, and still is, employed in reference to two types of obstruction near the lower end of the gullet—viz. : (1) to cases of hypertrophic anatomic—i.e., organic—stenosis with ectasia of the thoracic gullet above, which are supposed to have originated in pure hypertonic spasm of the cardiac circular fibres and with subsequent hypertrophic muscular narrowing of the lumen; and (2) to cases of spasm of these fibres without subsequent hypertrophic anatomic stenosis. Whether the first sequence actually occurs is now being questioned in some quarters, but many hold not only that it does occur, but that long-standing cardiospasm is always followed by hypertrophy locally and ectasia above, and that the absence of hypertrophy in functional non-anatomic stenosis necessarily excludes true cardiospasm. Some—e.g., Brown Kelly—however, who up to recently employed the term "cardiospasm," were careful to explain that they only meant a state of non-relaxation of the circular fibres of the cardia which should normally occur as a part of the neuro-muscular co-ordinate act of swallowing. Hurst, believing that a true spasm leads to hypertrophic stenosis, therefore substituted the term cardiac "achalasia" for that of "cardiospasm" where only functional stenosis was inferred from the free passage of a large mercury-filled tube. This terminology is more acceptable to many than the old one of cardiospasm, and it may perhaps be that this explanation of want of relaxation is a correct one of what occurs in some few cases, though personally I have not satisfied myself that the stenosis has been at the cardiac level in any of my patients. The term "cardiospasm" I have discarded for the same two reasons, and in addition it may be pointed out that the special mark of a hypertonic spasm in the alimentary canal is painful cramp or colic, which as a primary phenomenon is not evident in my experience, though frequent enough as a secondary symptom when food is impacted or arrested.

Holding that there is no convincing evidence that either cardiospasm or cardiac achalasia ever exist as primary phenomena and seeing that the level of stenosis is not at the cardia but at the level of the hiatus oesophageus of the diaphragm (where the gullet is embraced by the crura, which constitute a potential extra-oesophageal sphincter), I regard these terms as likely in course of time to become obsolete. In reference to the phrenospasm hypothesis of Jackson, it may be said that as a secondary symptom we are all familiar with it, but if
Jackson's former views had been correct then we should have had to assume in the majority of these functional cases a primary spasm of the crural fibres without the usual mark of diaphragmatic spasm—viz., hiccough. I do not know if Jackson has advanced matters very much in his new edition by throwing over the term "phrenospasm" and substituting that of "hiatal œsophagismus," meaning apparently thereby both an accentuation or spasm of the normal tonic contraction of the crura surrounding the gullet at the hiatus œsophageus of the diaphragm and also of the circular fibres of the gullet itself at that level. As a mercury-filled tube in these cases passes readily through the gullet at the hiatal level there can certainly be no powerful hypertonic spasm of the crural fibres or of the circular fibres of the gullet itself and no very obvious evidence of any distinct obstructive tonic contraction which is assumed by him to occur here.

All these hypotheses, be it noted, proceed on the assumption that general ectasia of the thoracic gullet is secondary to primary obstruction of one or other portions of the phreno-cardiac segment of the œsophagus brought about by muscular action or overaction.

I tentatively suggested some years ago that there might possibly be a block or impediment to the free passage of food due to an opposite cause—viz., neuro-muscular paresis and absence of co-ordinate active opening up of the phreno-cardiac gullet during the act of swallowing. At rest the gullet is collapsed, not open at the hiatal level, being embraced and possibly gripped by the crura; at the same time the hole for the passage of the gullet through the diaphragm (hiatus œsophageus) is a narrow lanceolate slit; during the act of swallowing the hiatus enlarges and its margin assumes a circular form and a wider cross section brought about by phrenic and possibly sympathetic nerve action on the muscular crura; and instead of a collapsed œsophageal lumen at the hiatal level the gullet here follows the expanded hiatus and thus we have an open tube. The exact sequence of events and the factors involved in this neuro-muscular effort, resulting in the active opening up of the phreno-cardiac gullet during the act of swallowing, has not been satisfactorily elucidated, but paretic abeyance of this normal act of active opening up of the lumen by muscular action would necessarily lead to a less free passage of food. The difficulty in the way of securing acceptance for an alleged strictly localized paresis in this circum-hiatal neuro-muscular mechanism is further increased by postulating that the derangement producing such chronic and far-reaching and gross results is primarily of a purely functional character, seeing that it occurs equally
in males and females and not especially in neurotic subjects. On the other hand there is no evidence of a primary organic paralysis of the phrenic nerves which would account for such interference with the co-ordinate act of swallowing at this level such as would tend to stenosis and secondary dilatation of the gullet above. If a localized neuro-muscular "block" really occurs at this level, however loth I am to fall back on the functional neurosis explanation, I can find no other likely pathogenesis at hand. I regard the neurosis as extra-oesophageal rather than intrinsic—i.e., it is not in the actual oesophageal musculature, as has been generally assumed by others, but probably referable to the crural fibres surrounding the hiatus.

Mr. Shattock, in a discussion on a paper by Batty Shaw and Woo, states that he is led to the hypothesis that oesophagectasia "is due to an inco-ordination of the nervous impulses transmitted by the vagus during deglutition, which impulses should normally cause contraction of the tube above and an active dilatation of the cardia." This explanation nearly corresponds with that of H. D. Rolleston and of Hurst. Whilst agreeing that the essential primary cause in these cases is of the nature of neuro-muscular inco-ordination I regard the enlargement by crural action of the hiatus in the diaphragm [for the oesophagus] as of much more importance than mere relaxation of the cardia. If the peccant mechanism is really in the diaphragm then Mr. Shattock is wrong in asserting that dilatation of the oesophagus is idiopathic and due to sympathetic and vagal vagaries; it would largely be secondary to an extra-oesophageal phrenic factor in the complex co-ordinate act of deglutition.

The older authorities, even up to and including Zenker and Morell Mackenzie, felt no difficulty in regarding dilatation of the thoracic oesophagus without anatomic stenosis below as a primary idiopathic condition and due to a sort of myasthenia or diminished contractile power of the musculature; as however hypertrophy rather than muscular atrophy is a fairly constant feature except at the site of greatest dilatation, where there may be partial atrophic thinning of the muscular coats, this hypothesis was generally abandoned even before the introduction of X-rays demonstrated that fairly strong, though ineffectual because not water-tight, peristaltic contractions occurred when bismuth paste was swallowed. This shows that the circular fibres remain active, so primary atony is apparently ruled out. It is

interesting to note that Zenker and von Ziemssen, in their well-known contribution on "Diseases of the Öesophagus," not only regard the ectasia as primary in cases where there is no anatomic stenosis immediately below, but they do not suggest any purely functional stenosis below as entering into the pathogenesis; spasm they describe as a separate morbid entity, but state that it does not lead to subsequent ectasia. They state that in addition to stagnation ectasie above an anatomic organic stricture there are also "rare cases with considerable and occasionally enormous ectasie in which there is no underlying stenosis whatever," thus apparently excluding functional stenosis not only of a spasmodic but also of a paretic nature. The circular fibres are supposed to be innervated by the sympathetic and it is curious to note that so far back as 1895 H. D. Rolleston suggested that the paresis primarily affected only the longitudinal fibres which are supplied by the vagus and that this vagal paralysis "would allow dilatation of the tube to occur and at the same time by interfering with the opening of the cardiac sphincter would induce hypertrophy of the circular muscular coat."^1 How paralysis of the longitudinal fibres innervated by the vagus exactly interferes with the opening of the lower end of the gullet and alternately what part, if any, the longitudinal fibres play in the active opening up of the phreno-cardiac öesophagus, Rolleston does not explain, but I have long thought that there is nothing inherently improbable in the suggestion that the longitudinal fibres of the gullet and the longitudinal and oblique fibres of the stomach possibly play some accessory part in the opening up during deglutition of the, at other times, closed phreno-cardiac gullet.

Jackson has pointed out that with a large dilated atonic stomach filled with gas there is sometimes dysphagia, which is at once relieved when the patient has got rid of the flatus by eructation; and I have observed that in cases with phreno-cardiac stenosis, whether organic or functional, some patients testify that they can swallow better after they have succeeded in relieving the wind in the stomach by belching. These facts have led to the suggestion that affections of the stomach other than those noted earlier in my remarks may, by producing angulation of the subphrenic gullet, be the primary cause in some cases of öesophagectasia without intrinsic anatomic stenosis. In several of my own cases there has been not only a history of chronic gastric disorders but in some marked gastroptosis has been seen by the X-rays. But

Shattock states that there is no post-mortem evidence of angulation of the subphrenic gullet in these cases, and I may add that, though I have looked for it, I have never seen such angulation by the X-rays; the bismuth passes either in a straight or else in a slightly curved stream, not in the least approaching a kink. Whilst hesitating, then, to accept the hypothesis that gastric disorders (especially where there is dilatation and fermentation) are a primary cause of functional stenosis of the lower gullet followed by ectasia above, I admit that these disorders aggravate the condition when established and that treatment of the stomach, especially by lavage, relieves to some extent the dysphagia and is an important part of the treatment.

Dr. Brown Kelly will deal more especially with the question of treatment; but successful methods of treatment sometimes give clues which help to clear up obscure points in etiology, and I must make some remarks from that point of view. As functional stenosis has been held to be due to paresis, it might be expected that the therapeutic application of electricity would supply some corroborative information. This is not so. The faradic current only acts on striped muscle which does not extend much beyond the upper third of the gullet. The use of the ordinary constant galvanic current is contra-indicated in a moist tube like the gullet on account of the danger of electrolytic action. The sinusoidal current is safe, but in the only case in which I tried it it failed. It is generally held that the alleged functional stenosis at or near to the lower end of the gullet with ectasia above is best and more or less successfully treated by the same methods of dilatation of the phreno-cardiac portion as are appropriate to unequivocal organic strictures. Personally I have not obtained the uniformly gratifying results recorded by others by bougieing and other dilatation methods, in cases where the diagnosis of absence of anatomic stenosis was unequivocal—i.e., where a mercury-filled tube easily passed into the stomach, clearly demonstrating that the lumen of the phreno-cardiac gullet was not merely potentially but actually normal. This test appears to me not only to demonstrate a normal lumen but to exclude intrinsic spasm and probably also achalasia of the circular fibres of the gullet both at the hiato-phrenic and cardiac levels, and also spasm and achalasia of the crural fibres (at the hiatal level of the diaphragm). The effect of dilatation by large bougies, hydrostatic and pneumatic bags, and by the passage of endoscopic tubes of large calibre, is of course very beneficial in cases wrongly assumed to be purely functional, but in which actual intrinsic anatomic narrowing is present; but the milder
form of the same treatment—viz., bougieing—might be expected to prove useful in paretic conditions by exercising a stimulating effect; but it is hardly to be expected that such mechanical stimulation, more especially by bags and divulsors, would relieve spasms and other contractile derangements. I agree that the "bougie effect" sometimes does temporarily improve to a substantial extent some cases of non-anatomic stenosis, but the improvement in my experience is more often only slight in unequivocal cases of functional stenosis, and such improvement as is obtained I regard as due to stimulation and relief of the inertia of the crural neuro-muscular mechanism in the region of the hiatus, by which the collapsed hiatus and gullet are induced to open up widely at this site. When Sir StClair Thomson was advised last year in this Section to dilate up with large bougies a case of œsophagectasia with a widely open phreno-cardiac gullet as seen with the œsophagoscope in position, he pathetically inquired, "What am I to bougie"?

Mr. Shatock suggestively asks if "there may not be a fault on the side of the mucosa which fails to supply a proper afferent stimulus" for the requisite active opening up of the lower portion of the gullet. He, however, makes no suggestion that the inertia or block is localized at the hiatal level and concerned with the impaired action of the crural fibres of the diaphragm as I hold, but he accepts the usual view that there is wrong action of the circular fibres of the cardia supposed to be innervated by the sympathetic nerves of the plexus gulae. The improvement in the relief of dysphagia in my cases I have attributed just as much to accessory methods of treatment as to such dilating methods as the passage of bougies.

Bags and divulsors I have discarded as there is no evidence forthcoming that either actual inflammatory rigidity or adhesions of the margin of the hiatus exists in these cases. I teach the patient in the early stages of treatment to pass all food directly into the stomach by means of a soft rubber stomach tube, when the orifice of the phreno-cardiac gullet is easily hit off, or failing that, by a gum-elastic tube. He or she is taught to wash out the dilated œsophagus, and also the stomach when there is dyspepsia, before taking a feed through the tube, and if the obstruction is so great as to lead to a considerable accumulation of saliva in the gullet this is removed between feeds as well, so as to relieve strain and help the relaxed walls to gain tone. This lavage further helps to cure the sodden state of the mucosa and to prevent the formation of erosions and ulcers which tend to result from
continued stasis of stale food. After three weeks short drinks are permitted by the mouth, but any fluid remaining after ten minutes is removed by the patient with the rubber oesophageal tube. The improvement is sometimes so marked that soft food and even ordinary well masticated meals can be swallowed either directly into the stomach or else they pass more gradually through without much delay. Care must be taken to eat slowly and little at a time, and the presence of stasis must be frequently tested by the patient by suction or by lavage through the oesophageal tube. I have never known a real cure, but the improvement may be so great that the disability is undetected by others present when meals are taken and the patient swallows fairly normally provided he does so carefully, deliberately and forcibly. In some cases the relief is only moderate, possibly due to the treatment not being religiously adhered to; relapses at all events are frequent.

Some cases improve scarcely at all under any method of treatment, and in these instances Mr. Shattock has suggested that it might be justifiable to excise the subphrenic gullet and make an end-to-end junction between the dilated thoracic oesophagus and the stomach. I think the fixing of a non-slipping thoracic oesophagus or rubber intubation apparatus in the phrenocardiac gullet would be a feasible procedure if made in two portions, the lower one inserted and joined up through a gastrotomy, and it would be less dangerous than excision. I have tried Guisez's apparatus but it slips up out of position just as does a Symonds' funnel here.

In conclusion, let me state that I fully realize that there is still a possible doubt as to whether oesophagectasia without anatomic stenosis below is a primary or a secondary condition, and it cannot be claimed that any of the explanations given, including my own, are anything more than unproven hypotheses and perhaps all very wide of the mark. I think, however, that the phrenic factor in the deglutitory act to which I called attention in 1911 cannot be ignored, as appears to be the case at present.

Ætiology.

With so much obscurity as to the essential nature of these ectasie, even as to whether there is an underlying functional stenosis or not, it necessarily follows that nothing much is really known as regards the predisposing and exciting causes. There is no sex predomination, and only a small proportion of the patients affected can be classed as neurotic. The condition is not limited to any period of life. Some few cases observed in the very young have been held to be congenital.
Zenker is probably wrong in his statement that "ectasie seem to be developed more frequently in youth than at any other time," even if those following cicatrical stenosis are included in the statement. There is no reliable information regarding the exciting causes of the condition. Claims made by reporters of individual cases of the supposed influence of lifting heavy weights, imbibing large draughts of hot water, and the impaction of hot dumplings and other ingesta are not convincing.

**Symptomatology.**

I have devoted so much time to the obscure question of pathogenesis that I feel deterred from dealing at length even with the commoner symptoms, much less the occasional and exceptional ones. Suffice it to say that the commoner ones are more or less identical with those met with in other varieties of ectasie, viz., those developed above a cicatrical stenosis; the X-ray pictures are also identical, and the essential difference is only brought out by the esophagogoscopic findings and by bougieing on which the differential diagnosis ultimately depends. Dysphagia varying in degree is nearly always a late symptom in oesophageal stenosis of any kind, as the lumen can be considerably reduced without interfering much with deglutition. In this form of functional dysphagia, however, some difficulty is noted comparatively early before marked secondary dilatation has occurred. It is often intermittent in the early stages with quite prolonged periods of normal deglutition followed by relapses.

Later regurgitation is added to mere dysphagia. The dilated gullet when overfilled from above and unable to empty itself sufficiently quickly below necessarily tends to cause an overflow into the pharynx, and when this is accompanied by peristaltic actions the accessory muscular actions usually associated with gastric vomiting may be called into play, and thus we get what is known as oesophageal vomiting. Regurgitation is often involuntary, but on account of the discomfort felt in the overloaded chest the patient frequently reinforces the involuntary act by a voluntary one. Coughing and even deep breathing may lead to regurgitation when the gullet is filled or nearly filled with fluid or other form of nutriment.

The questions of waterbrash and of rumination occasionally observed in oesophagectasia I am precluded from dealing with at length now.

A dilated gullet which is at the same time over-filled is made evident to the patient by symptoms varying from mere discomfort to evident distress and up to genuine pain. The pain is of the nature of a
secondary spasm, or cramp, or colic, or angina, and is brought about by hypertonic muscular action—i.e., violent peristalsis in an ineffectual effort to force impacted food through the stenotic lower end of the gullet. Violent pain may last from a second or two to fifteen minutes. With the throat mirror frothy fluid can often be seen in the pyriform fossæ, and when there is increased salivation, as often happens in well-established cases, much saliva which has been first swallowed and later regurgitated in a frothy form has frequently to be expectorated.

As regards loss of weight it is exceptional to find marked and dangerous emaciation, even in cases of long-standing, though a few patients have been gastrostomized for symptoms bordering on aphagia, and cases have even been allowed to succumb to asthenia from starvation. In these instances a proper investigation should have established the correct diagnosis and led to the employment of so simple a remedy as the stomach-feeding tube long before such a state of affairs had become established. There is usually a history of some loss of weight in long-standing cases even when the patients look healthy and well covered; a patient often states that he lost a stone or two to begin with in the course of six or twelve months, and then managed by careful deglutition to maintain a constant, though below par, weight for succeeding years. When the amount of nourishment which gradually trickles through from the dilated gullet into the stomach is large compared with the amount regurgitated the patient may manage to maintain normal weight and strength for three or four years or even more after the onset of the dysphagia. The main point is that there is not usually in functional stenosis, the steadily progressive loss of weight such as is met with in organic (i.e., anatomic) stricture.
SKIAGRAMS OF FUNCTIONAL PHRENO-CARDIAC STENOSIS.

Fig. A (one-third natural size) shows the sausage-shaped column of arrested bismuth paste in the thoracic gullet of normal calibre; between the level of the hiatus in the diaphragm and the cardiac orifice there is a thin indication of bismuth passing through the phreno-cardiac portion of the gullet; a more pronounced stream is seen passing down the nearly vertical lesser curvature of the stomach. The patient had suffered from moderate dysphagia for six weeks only, and this disappeared after massaging the gullet by bougieing twice daily with a hollow tube for feeding purposes for two weeks. It is believed that this case represented the early stage of the condition under discussion. There was no evidence of anatomic stricture found on oesophagosscopic examination.

Fig. B (half natural size).—This patient, a man, aged 43, had suffered from intermittent moderate dysphagia for two months only. On endoscopic examination there was no evidence of anatomic stricture in the phreno-cardiac region or elsewhere. The man died shortly afterwards under an anaesthetic prior to gastroscopy, and, post mortem, no anatomic stricture was found anywhere, but the thoracic gullet was slightly dilated, as seen in the picture.

Fig. C (half natural size).—In this case an anatomic fibrous stricture was found on oesophagosscopic examination about an inch above the diaphragm. The site of the stricture in this case, as seen by radiography, at once clinches the diagnosis of anatomic stricture and excludes functional stenosis, in which latter case the arrest of the bismuth is invariably at the hiatal level. When anatomic stenosis commences at the latter level, radiography does not help diagnosis, which can only then be decided by endoscopy or by the passage of a mercury-filled red rubber tube of 13 mm. diameter. This tube passes freely in functional stenosis, as shown in fig. F (half natural size).
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Fig. A.

Normal Gullet
? Porosis
Early step
Slight
Disphago
Looked at 2 mos.

Air in Stomach

Fig. B.

Ed. under Anaesthesia

Fig. C.

Hiatal level

Fig. D.

Functional Shëli
Nerary filled lube
in Stomach
SKIAGRAMS OF FUNCTIONAL PHRENO-CARDIAC STENOSIS.

Figs. D and E (half natural size).—These skiagrams were taken from the same patient. In fig. D the gullet was temporarily relaxed when filled with bismuth. In fig. E the picture was taken during peristalsis. Fig. D shows the picture usually observed in oesophagectasia following functional phreno-cardiac stenosis, but is identical with that seen in anatomic stenosis in the same region, the differential diagnosis being possible, not by radiography but by either endoscopic examination or by the passage of a mercury-filled rubber tube. This case was practically cured by the mode of treatment described in the text by feeding through a stomach tube. The gullet in functional cases is found full of fluid food post mortem, and, according to Irwin Moore, this observation hardly favours either the spasmodic or the achalasia theory.

Figs. G and H are both from a patient, aged 55, the first being taken immediately after swallowing a teacupful of bismuth paste; the greatly dilated gullet is contracted round the paste, which is arrested and remains, for the most part, arrested for several hours. After the usual treatment adopted by me had been carried out for a month, it was found that the bismuth paste passed straight into the stomach and none was arrested sufficiently long to enable a photograph to be taken showing it in the gullet. Although the dysphagia was so successfully relieved, the gullet remained as large as ever, though with increased contractile power presumably. The gullet looks even larger than before treatment, but that is due to the empty gullet being flattened by pressure of the thoracic viscera. Guisez's statement that a dilated gullet can undergo marked contracture after dilating treatment is contrary to my numerous observations checked by endoscopy and radiography.
Dr. A. Brown Kelly.

Treatment.

The preliminary part of the treatment of cardiospasm is carried out by aid of direct inspection. If the oesophagoscope is to be passed for the first time a general anaesthetic should be given, otherwise the nervous strain caused by the examination may aggravate the disease. In introducing the tube more resistance than usual is encountered at or near the mouth of the gullet. This experience, together with the fact that these patients often refer their dysphagia to the cricoid region, and not to that of the cardia, indicates that obstruction at the lower end of the gullet may be associated with spasm at the upper end, just as pyloric stenosis sometimes gives rise to reflex spasmodic stricture of the oesophagus.

If the patient is lying on his back with the head slightly lowered, which level is the best position for the examination of the cardiac end of the gullet, a quantity of cloudy grey fluid runs out of the tube as soon as it enters the gullet. More may be drained off by further depressing the head and shoulders. The masses of soft food in the lower parts are then removed and the walls of the dilatation inspected.

The hiatal and subhiatal regions\(^1\) are now examined. These are in the axis of the oesophagus and easily found if the dilatation is spindle-shaped. Most dilatations, however, are flask-shaped, and in advanced cases there may be considerable sagging of the wall to the right and downwards, so that the hiatus comes to be situated on the lower part of the left lateral wall while the fundus of the dilatation is at a lower level. Under these conditions, instruments introduced along the axis of the oesophagus impinge on the floor of the sac, and to reach the epicardia they must be directed well to the left and passed beneath the great bulging produced by the heart. It seems to me that oesophagoscopy in such cases affords a particularly favourable opportunity of studying movements of the heart.

The first point to note for future use in regard to the hiatal gullet is its distance from the upper teeth. In reports of cases of cardiospasm striking measurements of the gullet are sometimes given, but I

\(^1\) Dr. Wm. Hill's terminology has been largely adopted in this paper.
am not aware of any statement to the effect that its abnormal length is a constant feature of this disease. The chief conditions affecting the length of the normal gullet are sex, height, and age. In the male of medium height the distance from the upper incisors to the cardia is 40 cm. on an average, and in the female 38 to 39 cm. The following table shows the sex and height of the patient and the distance to the hiatus—i.e., to a level in the gullet 3 to 4 cm. above the cardia. It will be seen that while no individual is above medium height and several below it, the gullet in most is considerably elongated; this lengthening was greatest in the severer cases. The increase in length becomes more striking if to each of these measurements at least 3 cm. be added, which is the distance of the hiatus from the cardia (Table I).

Table I.—Hiatal Æsophagus.

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>Height</th>
<th>Distance from upper incisors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F.</td>
<td>19</td>
<td>5 ft. 2(\frac{1}{2}) in.</td>
<td>37 cm.</td>
</tr>
<tr>
<td>2</td>
<td>F.</td>
<td>60</td>
<td>5 (\frac{1}{2}), 4 (\frac{1}{2})</td>
<td>42 (\frac{1}{2})</td>
</tr>
<tr>
<td>3</td>
<td>F.</td>
<td>56</td>
<td>5 (\frac{1}{2}), 5 (\frac{1}{2})</td>
<td>47 (\frac{1}{2})</td>
</tr>
<tr>
<td>4</td>
<td>M.</td>
<td>58</td>
<td>5 (\frac{3}{4}), 7 (\frac{1}{2})</td>
<td>47 (\frac{1}{2})</td>
</tr>
<tr>
<td>5</td>
<td>M.</td>
<td>33</td>
<td>5 (\frac{3}{4}), 5 (\frac{1}{2})</td>
<td>47 (\frac{1}{2})</td>
</tr>
<tr>
<td>6</td>
<td>M.</td>
<td>24</td>
<td>5 (\frac{3}{4}), 6 (\frac{1}{2})</td>
<td>43 (\frac{1}{2})</td>
</tr>
<tr>
<td>7</td>
<td>M.</td>
<td>18</td>
<td>5 (\frac{3}{4}), 4 (\frac{1}{2})</td>
<td>37 (\frac{1}{2})</td>
</tr>
<tr>
<td>8</td>
<td>M.</td>
<td>59</td>
<td>5 (\frac{3}{4}), 7 (\frac{1}{2})</td>
<td>43 (\frac{1}{2})</td>
</tr>
<tr>
<td>9</td>
<td>M.</td>
<td>41</td>
<td>5 (\frac{3}{4}), 6 (\frac{1}{2})</td>
<td>43 (\frac{1}{2})</td>
</tr>
<tr>
<td>10</td>
<td>M.</td>
<td>22</td>
<td>5 (\frac{3}{4}), 5 (\frac{1}{2})</td>
<td>44 (\frac{1}{2})</td>
</tr>
</tbody>
</table>

Average distance in males ... ... 40 cm.
" " females ... ... 39 "

The aspect at the hiatal Æsophagus (or hiatus œsophageus as commonly but wrongly termed) next calls for attention. Some writers maintain that it is normal; others that it is normal but with the folds of mucous membrane more firmly pressed together so as to close the lumen; others that it is peculiar to cardiospasm and may differ in slight and severe cases; and lastly, there are several who in discussing this disease have omitted to describe the hiatal Æsophagus, probably having come to no conclusion as to its typical appearance, or regarding this as not different from the normal.

In most of my cases the lumen of the hiatal Æsophagus was surrounded by a stellate arrangement of folds of mucous membrane (figs. 1 and 2, p. 50); in others it was V-shaped, or merely a slit with a prominent cushion in front and behind. The size of the lumen was constantly changing, enlarging with inspiration and contracting or
closing with expiration. (All the patients had $\frac{1}{100}$ gr. of atropine injected hypodermically before being anaesthetized.)

The appearances and movements just mentioned were noted while the oesophagoscope was about 3 cm. above the hiatal level. From this view-point the aspect of the hiatal oesophagus in my cases of cardio-spasm was therefore normal. But as soon as the instrument was introduced a little farther and came into contact with the parts around the hiatus, this at once was closed, and remained closed until the tube was withdrawn a few centimetres, when the rhythmical opening and closing of the epicardia recommenced. This sequence of events could be produced over and over again in five of my cases; it was absent in one in which the patient had almost recovered; and was not observed in the others, which were examined before this heightened reflex activity had attracted my attention. In patients suffering from affections other than cardio-spasm in whom the sensation has been tested it has invariably

![Fig. 1.](image1)

![Fig. 2.](image2)

been found that the opening and closing of the epicardia continued in spite of its being touched by the tube, and ceased only when firm pressure was applied to the parts immediately surrounding the orifice; even then the movements occasionally persisted. The number of examinations I have made of patients with and without cardio-spasm in reference to this feature is still too small to warrant the statement that it is pathognomonic of cardio-spasm.

Various writers mention that in cardio-spasm firm closure of the gullet at the level of the hiatus follows any attempt to pass a tube through it, but they do not contrast the normal aspect and movements it presents when untouched with the spasmodic closure it undergoes from slight and distant irritation, nor do they appear to have met with the hyperæsthesia as constantly and over as large an area of the gullet as I have indicated; and certainly none of them, so far as I know, has ascribed the cardio-spasm to this hyperæsthesia.
These observations help to reconcile the apparent discrepancies in the descriptions of the hiatal œsophagus in cardiospasm, and show that the varying statements really correspond to the varying conditions of the opening. Further, they carry us a step onward in our knowledge of the nature of the disease by proving the presence of a hyperaesthetic region for some distance above the hiatal level. Irritation of this region is seen to produce contraction or spasm of the œsophageal wall, and not compression such as might be caused by the action of the crura of the diaphragm. Illustrations of the œsophagus at the hiatal level in cardiospasm that have been published also give the impression of contraction and not compression. If these observations and deductions prove correct we may discard the term "cardiospasm" and adopt that of "suprahiatal hyperæsthesia." Both, however, merely mark stages in the progress of our knowledge of the pathogenesis of œsophageal dilatations, as did the other names that have been successively applied—namely, idiopathic, paralytic or atonic, spasmogenic and cardiospastic. We are still ignorant of the primary cause of the disease; whether the next clue is to be sought in an undue irritability of the autonomous nervous system, and this in turn in a disturbance of some internal secretion, or of certain constituents of the blood, must meantime remain in the realm of speculative pathology.

The drugs chiefly recommended for the affection under consideration have been: bromides, valerian, belladonna, atropine and papaverin. I have not used the first two as none of my patients has been of a neurotic type. Belladonna or atropine I have occasionally tried on the assumption that the underlying disturbance was vagotonia, but without obtaining any appreciable improvement. Papaverin is said to be of assistance in slight cases.

The systematic washing out of the dilated gullet with an alkaline solution will be useful if the lining membrane is inflamed or otherwise unhealthy, but I have found it necessary only exceptionally owing to the stretching treatment employed having nearly always been followed by cessation or amelioration of regurgitation. On the other hand, some patients use a stomach tube frequently in order to evacuate the contents of the dilatation before going to bed, or to open the cardia, or even to introduce food into the stomach if the spasm is specially troublesome.

The food taken should be thoroughly masticated or soft and swallowed slowly. The disease is said to have been caused in some
instances by bolting food. The patient should neither eat nor drink anything likely to irritate the wall of the dilatation.

At one time the local treatment consisted chiefly in the passage of sounds and bougies. In slighter cases this may suffice. Thus, two of my patients have been cured for seven and two and a half years respectively by having had metal or ivory olives passed twice or thrice (Tables II, III, and IV).

**Table II.—E. B., aged 19.**

<table>
<thead>
<tr>
<th>Date</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>June 1</td>
<td>Dysphagia for seven years; subsisted for past year on biscuits,</td>
</tr>
<tr>
<td></td>
<td>Benger’s food, eggs, &amp;c.; regurgitates very little; weight, 6 st. 11 lb. 12 oz.</td>
</tr>
<tr>
<td>June 28</td>
<td>Passed small olives.</td>
</tr>
<tr>
<td>July 5</td>
<td>Passed larger olives.</td>
</tr>
<tr>
<td>July 12</td>
<td>Can swallow meat.</td>
</tr>
<tr>
<td>August 1</td>
<td>On ordinary diet.</td>
</tr>
<tr>
<td>September 27</td>
<td>Very stout; being overfed; weight, 9 st. 94 lb.</td>
</tr>
</tbody>
</table>

**Table III.—F. T., aged 60.**

<table>
<thead>
<tr>
<th>Date</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>June 21</td>
<td>Dysphagia for one and a half years; occasionally complete for four or five days; has lost 1½ st. since onset.</td>
</tr>
<tr>
<td>June 30</td>
<td>Food measured (see Table IV).</td>
</tr>
<tr>
<td>July 5</td>
<td>Olives passed.</td>
</tr>
<tr>
<td>July 8</td>
<td>Deglutition improved; no regurgitation; olives passed.</td>
</tr>
<tr>
<td>July 12</td>
<td>All food swallowed; no regurgitation.</td>
</tr>
<tr>
<td>November</td>
<td>Can eat anything; never regurgitates; has gained 3 st. since June 21, 1916.</td>
</tr>
</tbody>
</table>

In most cases, however, the amount of stretching produced by even the largest bougies or olives is insufficient to dispel the cardiospasm. An important contribution to the treatment of this affection was made in 1898 when Dr. J. C. Russel,⁠ of Southport, described the condition we have under discussion and recommended for its cure the stretching of the cardia by means of silk-covered rubber bags of increasing size. Unfortunately this paper was generally overlooked. Plummer, however, refers to it, and gives Russel the credit he deserves.

Five years later v. Mikulicz demonstrated the benefit obtained by overstretching the muscles around the cardia. He accomplished this

at first with two fingers, later with specially constructed forceps, introduced through the stomach.

In order to obtain the same effect without gastrostomy Gottstein and Geissler devised an instrument for introduction through the mouth consisting of a tube fitted at the lower end with an hour-glass-shaped distensible bag. When the constricted part of the bag was embraced by the cardia, water or air was injected until the desired amount of dilatation had taken place. The success that attended the use of this instrument led to the construction of various modifications. In some of these the distensible bag has been retained, as in Plummer’s apparatus. In others it has been replaced by two or more metal blades which can be separated to the desired extent by a graduated screw (Abrand, Mosher) or a dynamometric spring in the handle (Lerche, Brunings).

**Table IV.—F. T., aged 60.**

<table>
<thead>
<tr>
<th></th>
<th>Breakfast</th>
<th></th>
<th>Dinner</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>oz.</td>
<td></td>
<td>oz.</td>
</tr>
<tr>
<td>7 a.m. Porridge</td>
<td></td>
<td>6</td>
<td>12.30 p.m. Soup</td>
<td>3</td>
</tr>
<tr>
<td>Milk</td>
<td></td>
<td>4</td>
<td>Chicken and potatoes</td>
<td>2</td>
</tr>
<tr>
<td>7.30 Water</td>
<td></td>
<td>4</td>
<td>Jelly and milk</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cup of milk</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>14</td>
<td>Total</td>
<td>15</td>
</tr>
<tr>
<td>Regurgitated</td>
<td></td>
<td>8</td>
<td>Regurgitated</td>
<td>8</td>
</tr>
<tr>
<td>Retained</td>
<td></td>
<td>6</td>
<td>Retained</td>
<td>7</td>
</tr>
<tr>
<td>Tea</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.30 p.m. Tea</td>
<td></td>
<td>5</td>
<td>7 p.m. Milk</td>
<td>6</td>
</tr>
<tr>
<td>Bread and butter</td>
<td></td>
<td>1</td>
<td>Water</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>6</td>
<td>Total</td>
<td>9</td>
</tr>
<tr>
<td>Regurgitated</td>
<td></td>
<td>2</td>
<td>No regurgitation</td>
<td></td>
</tr>
<tr>
<td>Retained</td>
<td></td>
<td>4</td>
<td>Retained</td>
<td>9</td>
</tr>
<tr>
<td>Totals</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Swallowed</td>
<td></td>
<td>44</td>
<td>Regurgitated</td>
<td>18</td>
</tr>
<tr>
<td>Retained</td>
<td></td>
<td>26</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Guisez in 1911 had used Gottstein’s balloon in fifteen cases and had obtained improvement in all. He advised that the bag be distended until its diameter was 6 to 7 cm. He had found that the capacity of the dilatation diminished rapidly after treatment.

Plummer, who probably has had greater experience than anyone in the treatment of this affection, employs a dilator connected with a water tap and furnished with a gage indicating the amount of pressure. This is gradually raised at successive treatments until satisfactory results are obtained. In one case unusually high pressure caused
rupture of the oesophagus. In 1912 he published his results in ninety-one cases of diffuse dilatation of the oesophagus without anatomic stenosis. Of these, seventy-three were completely relieved of dysphagia, eleven were not completely cured, four had died and three could not be traced. Most of the patients with extreme inanition needed only one treatment to effect a cure. In three patients he found that the dilated oesophagus had returned to the normal size.

Chevalier Jackson considers that it is difficult to place dilating bags accurately by blind methods and therefore prefers a mechanical divulsor, such as Mosher’s, which can be introduced through the oesophagoscope and the action of which can be regulated by the sense of touch. His 53-cm. oesophagoscope is passed into the stomach and afterwards the divulsor through it. Under guidance of the eye the oesophagoscope is withdrawn until the expansile portion of the divulsor is exposed and its greatest diameter placed in the hiatus of the oesophagus. It is then expanded to about 20 to 25 mm. and kept in for from five to ten minutes. As divulsion is painful he advises ether anaesthesia. He finds from one to six divulsions at intervals of a week necessary.

I have used Gottstein’s instrument almost exclusively. The method adopted is as follows: Having obtained during the oesophagoscopic examination the distance from the upper teeth to the hiatus, this is measured from the middle of the bag along the stem of the instrument and marked with a thread or a sliding piece of rubber tubing which also serves to protect the bougie from being bitten. Before introducing the instrument, the contents of the gullet are removed by suction, or, if thick, washed out. Patients unaccustomed to the procedure will at first be cocainized, but they quickly learn to dispense with this. The dilator is then passed. It may meet with slight obstruction at the hiatus, but if gentle pressure be maintained for a few seconds it glides onwards. When the measurements indicate that the bag is in position it is gradually distended by water. In the instrument I use, four to six syringefuls are usually injected before the patient begins to complain of pain, whereupon the injection is stopped. Sometimes the distended bag is left in situ for a few minutes, after which the water is allowed to escape and the apparatus withdrawn.

A method, by means of X-rays, of determining whether the bag is in position is to place a small lead square or circle over the site of the stream of bismuth passing from the gullet to the stomach and afterwards to use a dilator the lower part of which contains a lead rod. The latter is watched crossing the lead window and the distance estimated. It will
usually be found to pass considerably to the left in entering the stomach. One or a few dilatations may produce a cure, or improvement lasting for months. In more obstinate cases I have repeated the dilatation at intervals of a few days over a period of several weeks, but the benefit obtained has not been proportionate to the amount of treatment. The dysphagia, as a rule, is immediately relieved and the day following the first stretching the patient eats a hearty meal such as he has not enjoyed for months or years. He quickly gains weight and vigour and may remain well indefinitely or for a variable period (Table V).

**Table V.—H. M., Aged 58.**

<table>
<thead>
<tr>
<th>Date</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1913</td>
<td></td>
</tr>
<tr>
<td>May 14</td>
<td>Dysphagia seven years; weight under 10 st.</td>
</tr>
<tr>
<td>May 27</td>
<td>Passed thick stomach tube.</td>
</tr>
<tr>
<td>June 18</td>
<td>Swallowing soft food; no regurgitation for ten days.</td>
</tr>
<tr>
<td>August</td>
<td>Weight, 10 st. $8\frac{1}{2}$ lb.</td>
</tr>
<tr>
<td>1914</td>
<td></td>
</tr>
<tr>
<td>May 3</td>
<td>Dysphagia returned three weeks ago; complete for one week. Dilator, two and a half syringefuls.</td>
</tr>
<tr>
<td>May 17</td>
<td>Dilator, three and a half syringefuls.</td>
</tr>
<tr>
<td>May 18</td>
<td>Greatly improved.</td>
</tr>
<tr>
<td>1916</td>
<td></td>
</tr>
<tr>
<td>May 3</td>
<td>Well until fortnight ago; all regurgitated since.</td>
</tr>
<tr>
<td>May 15</td>
<td>Dilator, six syringefuls.</td>
</tr>
<tr>
<td>May 31</td>
<td>Better than for years.</td>
</tr>
<tr>
<td>1917</td>
<td></td>
</tr>
<tr>
<td>February</td>
<td>Very well; no regurgitation; weight 11 st. 12 lb.</td>
</tr>
<tr>
<td>November</td>
<td>Died of pneumonia; took food well till last illness.</td>
</tr>
</tbody>
</table>

**Table VI.—Mrs. M.A., Aged 62.**

<table>
<thead>
<tr>
<th>Date</th>
<th>Total swallowed</th>
<th>Regurgitated</th>
<th>Retained</th>
</tr>
</thead>
<tbody>
<tr>
<td>October 25</td>
<td>97 oz.</td>
<td>86 oz.</td>
<td>11 oz.</td>
</tr>
<tr>
<td>... 26</td>
<td>36</td>
<td>26</td>
<td>10</td>
</tr>
<tr>
<td>Cardia dilated.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>October 30</td>
<td>89 oz.</td>
<td>16 oz.</td>
<td>73 oz.</td>
</tr>
<tr>
<td>31</td>
<td>108</td>
<td>17</td>
<td>91</td>
</tr>
<tr>
<td>November 1</td>
<td>113</td>
<td>11</td>
<td>102</td>
</tr>
<tr>
<td>2</td>
<td>105</td>
<td>22</td>
<td>83</td>
</tr>
</tbody>
</table>

The regurgitation also ceases, or is distinctly diminished (Table VI). Sometimes, however, there is retention without regurgitation and the presence of food and fluid in the dilatation may escape notice unless looked for. The contents of the oesophagus may be removed by the Senoran’s suction apparatus; and the quantity obtained from time to time gives an indication of the progress towards recovery. Another method of determining the presence and amount of retained fluid is to
cause the patient to swallow a capsule three-quarters full of bismuth which floats. In one of my patients from whose gullet about 300 c.c. of fluid could usually be withdrawn, the capsule could be seen about the level of the episternal notch moving up and down with respiration. Although the amount of retained fluid in this case was so considerable, the patient suffered but little inconvenience from it and often was unaware of its presence.

There appears to be no doubt that the dilated oesophagus occasionally returns to its normal size; several authorities have made observations bearing on this point.

I shall say nothing about gastrostomy excepting to recommend it for patients who have been reduced to a moribund state by prolonged fasting, and in whom an examination with the oesophagoscope and dilatation treatment would meantime be contra-indicated.

The only death I have had directly due to cardiospasm was that of a woman, aged 56, who had suffered from dysphagia for twenty years. Four weeks before death the obstruction at the cardia had suddenly become almost complete. She was first examined by me five days before her death, when on introducing the oesophagoscope a large quantity of bismuth in suspension was drained off: this had been swallowed six days previously for X-ray purposes. A tube was passed into the stomach under direct observation, and left in situ. After having been fed through this for two days she withdrew it. She was then able to swallow a little fluid naturally. The same evening she died of heart failure. At the post-mortem examination the gullet was found to be greatly dilated. There was no anatomic stenosis at the lower end and no sign of malignancy anywhere (fig. 13, p. 91).

Case of Cardiospasm.

By A. Brown Kelly, M.D.

Lieutenant —, aged 28. First experienced difficulty in swallowing early in 1913, after having had his tonsils enucleated. The dysphagia gradually increased, so that by the end of 1914 he had to be fed entirely by the stomach tube. He then consulted Dr. Birkett in Montreal, who dilated his cardia. In January, 1915, he returned to this country, and has been treated at intervals since by the exhibitor.
His condition varies from day to day, and he has periods of improvement and exacerbation. His chief symptoms are holding up of food at the cardia, accompanied by a feeling of weight in this region, and regurgitation. He must eat slowly, and if the dysphagia is troublesome the food has to be washed down with large quantities of liquid, otherwise it is regurgitated. Occasionally after a meal he "gets filled up" so that he dare not speak or move for fear of regurgitating. Before going to bed he excites retching or passes a tube in order to empty the gullet and prevent regurgitation while asleep. The exhibitor has always found fluid in the oesophagus, and has withdrawn from 65 to 150 c.c. The oesophagus is greatly dilated. Systematic stretching of the cardia was carried out early in 1915, and twice in 1917—the last time after the patient had been to the Front. The patient keeps in good health, his weight is maintained, and he carries on his military work, which is chiefly administrative.

Note.—The case was demonstrated at the meeting: 130 c.c. of cloudy fluid were removed by suction from the dilatation—the last food and drink had been taken fully two hours previously. Gottstein's dilator was then passed without local anaesthetic by the patient himself, and when the constricted part of the bag was in the cardia-epicardia this was stretched by distending the bag with water.

The President.

For the present I will only ask you to concentrate, as far as possible, on the two chief points raised by the openers. One of these is whether the cause of these dilatations is cardiospasm, and the other is the influence claimed by Dr. Hill for the diaphragmatic fibres about the oesophageal foramen.

Professor S. G. Shattock, F.R.S.

At times the oesophagus will pass entirely through the right crus, and it is conceivable that under such circumstances an anatomical variation might lead to obstruction. In one such case which I have seen, however, dissection showed that the oesophagus above this abnormal entry was not dilated.

With regard to spasm: There have been put as alternatives that spasm may be one cause of this obstruction, and that the other may
be inco-ordinated action, that is to say, a want of co-ordination between
the descent of the contents of the œsophagus and the dilatation of the
cardiac orifice. If we accept the observations of Dr. Brown Kelly—
and I think they are most important—we have a very simple and
satisfactory solution of the cause of the obstruction.

I have long held myself that a hyperæsthetic condition of the
mucosa furnishes the most satisfactory explanation of hypertrophic
stenosis of the pyloric canal; so that the mere passage of food over
the membrane excites a spasm in the muscle, which may completely
prevent the passage of food. That, therefore, is a theory congruous
with Dr. Brown Kelly's observations upon the œsophagus. There is
one difference, anatomically, in the case of the pylorus; there is an
overgrowth of muscular tissue at the site of obstruction. That we do
not see in œsophagectasia. In an excellent preparation at St. Thomas's
Hospital, e.g., in which a man, after thirteen years' obstruction, died
of inanition, there is no muscular thickening whatever at the cardia.
This may, therefore, in the minds of some, still make for the theory of
neuro-muscular inco-ordination; for the closure beyond the area of
excitation might have been due, not to an active spasm, but to a failure
in the co-ordinated dilatation.

When we cast about for somewhat analogous conditions, one of the
nearest perhaps is that of the urinary bladder. There are certain cases
of idiopathic dilatation of the urinary bladder, male and female, in which
one can find no trace of urethral obstruction. I have examined some
such, and they have been described also by others, and what is more, they
occur in the fœtus. They might be thought to be cases of infection and
of spasm incited in the urethral muscle by the passage of urine over an
inflamed mucosa, but the fact that the conditions may occur before birth
will exclude such a view. There is one example in Guy's Hospital Museum,
another in the College of Surgeons, of female children whose bladders, at
birth, were greatly distended without discoverable cause of obstruction.
That some such are due to neuro-muscular inco-ordination may be guessed
from the phenomenon of what Paget called "stammering" bladders;
where after the escape of a few drops of urine, a complete block will
ensue, which may last long enough to lead to painful distension. In
reflecting upon this subject I considered the possibility of there being
not a hyperæsthetic but a hypoæsthetic state of the vesical mucosa
(pharyngeal hypoæsthesia or complete anaesthesia is well known in

The view seemed to me, moreover, applicable as an explanation of megacolon, where the colon slowly dilates without any trace of organic obstruction; and this, commencing in the pelvic portion where there is no anatomical sphincter. In connexion with this subject I devised the following experiment: Into the bladder of a female cat a little cocaine solution was injected through a fine catheter; after a few minutes the bladder could be indefinitely distended with salt solution, none of which escaped by the side of the instrument, as it did before the local anaesthetic was used. Obviously, one of two things had happened; either the mucosa had been rendered anaesthetic and had failed to furnish the proper reflex for the excitation of the detrusor urinae, the sphincter remaining co-ordinately contracted, or the cocaine had reached the muscular tissue and produced motor paralysis. And this leads me to conclude by suggesting that one way of discovering whether we are dealing with an extraneous factor like the constriction, actual or virtual, of the diaphragm, or with the intrinsic factor of proper spasm or achalasia, might consist in the use of this local anaesthetic. I am sure the skill of laryngologists would enable them to cocainize the cardiac orifice or the cardiac end of the œsophagus. You will either anaesthetize the mucosa, or, better still, paralyse the musculature of the cardiac orifice. And then, if it were found that the obstruction was removed, Dr. Hill's view that all these cases are due to phrenic conditions would, I think, be negativèd.

I may add this further criticism: that if the obstruction is really where Dr. Hill suggests it is, a study of the preparations on the table will show that the dilatation later on proceeds down to the actual orifice of the stomach: on his view this should not be so. For let us assume that the dilatation started at the diaphragm: as it proceeded it would overcome the obstruction, and on reaching the cardia the obstruction should be removed. But that is not the case. The dilatation, in these preparations, ends only at the gastric orifice, and we must conclude, I think, that we are dealing either with spasm of the cardia or with a form of obstruction due to inco-ordination.

Mr. W. Stuart-Low.

I regret that more definite statistics have not been given with reference to the incidence of this affection of the œsophagus as regards the period of life at which it occurs and in which sex. Neither of the introducers of the discussion have given attention to this important
My cases have all been females between the ages of 45 and 50. I have two well-marked instances under my care at the present time both of which have yielded to treatment along the lines I have always pursued.

The condition of the mucous membrane in every instance that I have treated has been one of desiccation. There is a chronic state of deficiency of mucous secretion, so that the back of the pharynx and the oesophageal lining are seen to be but very slightly protected and lubricated by mucus which, normally, is abundantly present. Such an imperfectly protected mucous covering becomes very readily congested and irritated by sepsis from the mouth and throat; this frequently occurs when the teeth are carious and pyorrhoea alveolaris and septic tonsils are present. If this congestion and irritation go on long enough oesophagitis results and this is followed by the pain on swallowing so often complained of by these patients, and then by spasm.

The most efficient treatment is summed up in one word—lubrication. This may be carried out by means of paroleine or liquid paraffin, but is best accomplished by supplying what is deficient—viz., the natural secretion—mucin—in the elegant and pleasant tasting preparation, namely, elixoid mucin (Burroughs, Wellcome and Co.) first introduced by myself in treatment of such deficient mucus-secreting surfaces. It is perfectly safe if given in large quantities—a tablespoonful before meals, with meals to sip, and after food. A large soft oesophageal bougie should be passed once every day or two thoroughly smeared with the mucin elixoid.

I can usually hand the patient over to the general practitioner to pursue this treatment, and it becomes only necessary to pass the bougie once a month or once every six weeks, if the persistent lubrication of the interior of the oesophagus is kept up and the asepsis of the mouth is carefully carried out.

**Mr. W. M. Mollison.**

I have seen some cases of the condition and have found them improved if not cured by the regular passage of the mercury-filled tube. There must be a large functional element in the cases; the neuromuscular mechanism of the oesophagus being affected. The interesting findings of Dr. Brown Kelly would not negative this suggestion but rather support it since an organic basis is common to all functional arrangements at one time or another.
The last two cases that have come under my care were of the "nervous" temperament or had had nerve strain: the first was that of a bank clerk, aged 39, who had been working very hard during the War, from 9 a.m. to 11 p.m. at times; he began to have difficulty in swallowing in October, 1914, but his condition improved; the symptoms recurred with much flatulence and slight vomiting in 1916: X-ray examination showed much dilatation of the lower part of the oesophagus. After the use of the mercury tube for a few days the patient wrote to say that the food passed well and that he no longer had flatulence.

The second case was that of a nun, also aged 39; she was seen in April of this year. For about nine years she had had the sensation that food did not pass down; sometimes small amounts of food would come up suddenly during a meal; sometimes she had the sensation that food was coming up and this suddenly passed off. She had only lost a little weight; for four years she had had spasmodic cough which at times made her sick. X-ray examination showed dilatation of the lower part of the oesophagus and the passage of a bougie led to a large amount of food being vomited. The passage of the mercury tube was easy and the symptoms greatly abated; the patient has written to say that she is better but that she has to pass the tube at times.

The War has shown what an extraordinary number of conditions may be functional, so that there seems no reason why the condition under discussion should not fall into the same category. Perhaps the nearest parallels are to be found in the cases of persistent vomiting of months' duration following gassing, which have been cured by simple persuasion.

Dr. Dan McKenzie.

I have had experience of two of these cases of dilatation of the oesophagus and so-called cardiospasm. One of them occurred many years ago, before special attention had begun to be drawn to this very interesting subject; and it proved fatal. The patient, a man of middle age, came to the Central London Ear and Throat Hospital with the story which most of these patients give, of the arrest and regurgitation of food, and when he arrived at the hospital he had had absolute obstruction to food and liquids for three days, so that he was in a condition of water-hunger, although he did not seem to be very ill. When I passed the long tube, it plunged into a well filled with food and liquid, the result of his having tried for three days to force food down.
It took me two hours to empty this: though if it occurred now I should invert the patient as a quick means of evacuation. The emptying was a difficult process, because pieces of meat got into the mouth of the aspirator, and each time the instrument got blocked it had to be removed and cleared. When the oesophagus was empty, I was able to get a Hill's feeding tube into the stomach and to feed the patient. I left the tube in, and, unfortunately, the house-surgeon, at the man's request, removed it, and the patient strenuously objected to its replacement. Next day he left the hospital, but returned later saying he could not swallow. As he refused to have a tube passed again, gastrostomy was performed. The surgeon's finger was inserted from below, but he could not detect any spasm or indication of obstruction. The man died from shock.

My second case was a much more fortunate one: he is still under my care and has been shown before this Section.\(^1\) He is now 30 years of age, and has had symptoms of obstruction of the gullet for seventeen years. He keeps fairly well by means of occasional dilatations and he can now swallow comfortably.

**Dr. Dundas Grant.**

My actual experience in dealing with this type of case has been almost entirely limited to one I saw with Dr. Batty Shaw some time ago, in which I had the opportunity of passing a long bougie through the cardia under the guidance of X-rays, a very desirable precaution because there was a sacculated right half, the patient having a flask-shaped oesophagus. Dr. Batty Shaw found there was some digestive disturbance in two of his cases, the exact nature of which I do not know.

I have recently read of an interesting point in relation to the physiology of the lower end of the oesophagus—namely, that there are rhythmical gapings of its lower orifice as long as the food has not become acid, but that as soon as it becomes acid the cardia closes up at once. It is reasonable to suppose that if there is hyperacidity, dilatation will cease, and that the contraction will be more acute than normally. Probably in many cases of extreme pyrosis (waterbrash) there is absence of dilatation of the lower orifice, and some dilatation of the tube, so that very large quantities of saliva collect—really pharyngeal secretions—

\(^1\) *Proceedings*, 1915-16, ix (Sect. Laryng.), p. 51.
these constituting the so-called waterbrash. I used to treat these with bicarbonate of soda and belladonna, and in one case I passed an œsophageal bougie, with relief resulting. Apparently, in the cases under consideration there is an idiopathic tendency to dilatation, analogous to that of extraordinarily dilated intestines, or to the peculiar general condition which gives rise to ptoses, such as gastroptosis and enteroptosis.

Sir StClair Thomson.

Cardiac spasm and spasm of the hiatal orifice appear to be a little mixed and I should be glad if we could clear up some of the "terminological inexactitudes" of this valuable debate.

Mr. Douglas Harmer.

Without doubt reflex plays an important part in the production of this condition. Some years ago, I had under my care a colonel who had a dilated œsophageus, and who had at times so much spasm that he was unable to swallow anything. On one occasion he said that while taking his soup a small piece of carrot which it contained was felt to pass with great difficulty along the whole course of the œsophageus and caused considerable pain. When it reached the cardiac orifice, it stuck, and since then, namely for ten days, he had been unable to swallow anything, either fluid or solid. I took him at once to Dr. Hurst, who gave him some bismuth, which dilated his œsophageus considerably, but did not enter his stomach. Upon my passing a bougie an obstruction was encountered at the cardiac orifice. Pressure was maintained, and the bougie suddenly slipped into the stomach, so easily that I feared that I might have perforated the œsophageal wall. As soon as it was removed he said: "That has cured me; now I shall be able to swallow." He wrote me later that he went to his club, ordered a beef-steak, and ate it with the greatest ease. For a long time afterwards he had no further trouble. I had not used cocaine.

It is remarkable how suddenly the spasm may disappear in these old-standing cases. Some years ago, Sir Anthony Bowlby had a man under his care who had suffered from this condition for seven years, and was steadily getting worse. There came a time when he was unable to swallow anything and, even with anaesthesia, efforts to pass a bougie
failed: this was before I practised oesophagoscopy. As the man's condition was so bad, gastrostomy was performed. The moment he recovered from the anaesthetic in the ward he said to the sister: "May I have a drink?" She said: "But you can't swallow anything." He replied: "Yes, the obstruction is relieved." He was given water to drink, and it was found that he could swallow it perfectly. It was decided not to remove his tube as he was in such a bad condition, but that it should not be used. He was sent to the Swanley convalescent home, where he developed pneumonia. He was, therefore, re-transferred to St. Bartholomew's, and he died some time afterwards. The post-mortem examination showed that, apart from slight dilatation of the oesophagus, there was no hypertrophy in any part of its course.

Mr. W. Howarth.

It is very difficult in these cases to make out, by oesophagoscopy examination, where the obstruction really lies, whether it is at the hiatal level or a little below. Again, though I have looked at many dozens of skiagrams, I find the same difficulty in locating the obstruction exactly. I cannot see any particular reason for seeking an extra-oesophageal cause for this obstruction, and I am content to believe that it may lie in the wall of the oesophagus itself. What may cause the inhibition of the relaxation that normally precedes the peristaltic wave one is not prepared to say, but we know that variation in the acidity, or in the chemical composition of the stomach content can in some way produce this. I can recall two cases in which the oesophagus was distinctly dilated and held more than a pint of fluid, but in which I could find no obstruction whatever above the diaphragm, and the oesophagoscope passed easily into the stomach. I had the abdomen opened at the same time, and just below the diaphragm in the wall of the abdominal oesophagus was a small carcinoma: it was not in the lumen of the gullet, and was not causing obstruction, but the irritation set up in the neuro-muscular mechanism evidently sufficed to produce excessive dilatation of the gullet above. Dr. Hill's theory may be a good one, but it does not appeal to me. I am very interested in Dr. Brown Kelly's views with regard to hyperaesthesia as normally the lower end of the oesophagus is singularly insensitive.
The possibility of the faulty co-ordination theory as a likely explanation of this dysphagia does not seem to have been exhausted. Will Dr. Hill reconsider whether this theory of faulty co-ordination does not meet the case?

I was specially interested in his diagram (1) showing the action of the tongue muscles in straightening as they pulled the oesophagus forward and upward, so undoing the "repose kinks"; and (2) showing the bands anchoring the oesophagus to the crura. As Dr. Hill has shown, the oesophagus is a potential tube in repose with a tendency to sag or kink at certain points. The first part of the act of swallowing converts this into a patent tube, more or less, in proportion as the tongue muscles elevate the oesophagus into position, and in proportion as the anchoring bands hold. Faulty co-ordination and inequality in the pull of either side will exaggerate the repose position, create a feeling of difficulty in swallowing, and so establish something of the nature of a "swallow stammer." These cases resemble functional aphonia: some are cured quickly, others are very obstinate.

Mr. H. L. Whale.

One of Dr. Brown Kelly's diagrams (p. 53) gives a list of the foods taken by a patient at his four daily meals, and the quantity which was retained and regurgitated respectively. I notice that the proportion retained increased as the day wore on. This does not correspond with any periodicity in the secretion of the gastric juice; does Dr. Brown Kelly consider that this fact throws any light on the causation, or suggests a functional element in these cases?

The President.

Before Dr. Brown Kelly replies, will Dr. Hill say what is the value of the researches which have been made on the innervation of the oesophagus? Seven or eight years ago I heard Dr. Guisez deal with this subject at the Hôtel Dieu, and he mentioned that the dilatation of the cardiac end of the oesophagus was due to the special dilator branch of the vagus. On referring to Guisez's book I find that it was Oppen-chowski, of Dorpat, who demonstrated the special dilator nerves of the
cardiac end, and it was he also who showed the action in constricting
the opening of the branches of the sympathetic through the great
splanchnic. That seems to be the mechanism which is irritated and
which produces the closure of the orifice in these cases. In Mr.
Harmer's case we saw that the presence of a very small foreign body
in the oesophagus will excite spasm quite out of proportion to the size
of that body. In this discussion there has been no allusion to the
superior constrictor of the oesophagus, I mean the circular fibres around
its upper end, and their share in the mechanism, which is only second
in importance to the closure of the lower end in these dilatations.
Nothing was said as to the mode of opening of the mouth of the
oesophagus, except that it was passive to boluses of food, whereas
it has now been demonstrated that an active dilatation takes
place in anticipation of the bolus through the action of dilator
filaments.

Dr. Hill (in reply).

Mr. Shattock, Mr. Howarth and others have failed to grasp my
meaning as to what takes place at the hiatus of the diaphragm during
the last stage of the deglutitory act. In the same way that the opening
up of the upper orifice of the gullet is dependent not on any intrinsic
oesophageal action but on the pulling away of the larynx and cricoid
plate from the spine by the hyoid and tongue muscles, so the opening
up of the lower region of the gullet—the phreno-cardiac portion—is not
mainly brought about by the intrinsic oesophageal musculature, but is
mainly actuated by the musculature of the crura forming the margins
of the hiatus. I draw on the blackboard an inferior view of the hiatal
region at rest when it appears as a long lanceolate slit enclosing the
collapsed laterally compressed gullet. I maintain that whether or not
there are inhibitory nerves which bring about dilatation of the cardia,
the intrinsic gullet musculature is powerless to make the margins
of the hiatus in the diahragam assume a circular or elliptical form,
which is a necessary antecedent to the expansion or patency of the
previously collapsed and compressed gullet. If this phrenic mechanism
is at fault it would explain functional stenosis at the hiatal level. There
is no proof that hypertonic contraction or spasm occurs in these cases,
for the endoscopic appearances are normal. As regards the drawings
of Starck and Guisez, their pictures when they are not within the
limits of normality are purely fanciful. Achalasia may possibly be a
factor in functional stenosis at the hiatal level, but in my view the phrenic factor is necessarily of far greater importance, and has been too long ignored.

Professor Shattock (in further comment).

One can imagine, indeed one is disposed to believe, that those phrenic fibres which surround the opening are inhibited in their contraction as the œsophageal contents pass. It is so in the case of the urethra. When the bladder contracts to empty itself, the compressor urethra relaxes, but the urethra does not expand, as Dr. Hill would suppose in the case of the œsophagus; it merely relaxes. But given such an inhibition, there is no reason why the aperture should be made round instead of remaining as an inert slit.

Dr. Brown Kelly (in reply).

I have endeavoured to deal with the subject from a practical standpoint. In 1912, at Liverpool, I discussed the various theories that had been brought forward with regard to the aetiology of this affection. The opinions I have expressed to-day are founded on observation, and it remains for you to put them to the test.

The Pathology of Øsophagectasia (Dilatation of the Øsophagus without Anatomic Stenosis at the Cardiac Orifice).

Remarks by Irwin Moore, M.B.

I have compiled the following notes and references relating to the unique series of specimens shown at this meeting. With one exception the drawings have been carefully executed by Mr. Thornton Shiells, under my immediate supervision, with the collaboration of Professor Shattock, and in order to obviate the magnification caused by the cylindrical jars in which the majority of them are preserved, the preparations were submerged in a water bath with flat sides. Special care has been taken, in each case, to depict the thickness of the œsophageal wall accurately, especially at the cardiac orifice. Owing to
the fact that nearly all the specimens have been preserved in spirit, some shrinkage will have taken place, but this would be uniform in each and of no considerable degree; and it must be borne in mind that the subphrenic portion of the gullet always appears shorter and thicker than when recently removed. I am greatly indebted to Professor Shattock for having helped me to draw up the additional notes (included in brackets) appended to some of the specimens.

I might add to the observations made by the different speakers, by drawing attention to the interesting fact that in a certain number of these cases it is reported that the contents of the dilated œsophagus remained in situ after death. As it is obvious that all active muscular contraction had then ceased, the obstruction at the lower end can only be attributed to the mechanical pressure of the surrounding parts, as in the retention of urine, e.g., in the bladder after death. Another matter to which attention may be drawn is the association of neuroses with the condition under discussion. This has been fully referred to by Dr. Batty Shaw,\(^1\) in the three of these cases, e.g., Specimens I, IX, and X, and receives some confirmation from a further case also recorded in the present series, e.g., Specimen II, in which the family history showed a marked susceptibility to vomiting.

The original drawings of these specimens, with their references, will be bound and catalogued under the title of "Œsophagectasia (Dilatation of the Œsophagus without Anatomic Stenosis)," and placed in the library of the Royal Society of Medicine.\(^2\)

**Specimens exhibited from the London Museums, including a Specimen from the Victoria Infirmary, Glasgow.**

(I) **Unmounted Specimen from the Museum of St. George's Hospital\(^3\)** (fig. 1, p. 79).

**Shown by Mr. Barwell.**

*Description.*—"Removed from a female, aged 34, showing great fusiform dilatation of the œsophagus, extending from the level of the cricoid cartilage to the cardia. Measuring 10 in. in length and 4½ in. in its maximum width.

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\(^2\) In view of the rarity of these cases, and the importance in connexion with the causative factor of a thorough examination of the parts in situ, Professor Shattock would be pleased if anyone, holding a post-mortem on one of these cases, would kindly notify him at the Royal College of Surgeons.

\(^3\) Not yet catalogued and numbered.
When opened it contained fully a pint of milk. There is muscular hypertrophy, ulceration of the mucosa with small white patches (epithelial islands) scattered about. The stomach was small and catarrhal."

[The right vagus in this case is reported to have been dissected out and "appeared to be normal." The dilatation involves the whole of the oesophagus as far as the actual orifice of the stomach. This is the case also in specimens Nos. 8 and 10. The cardiac orifice was described as narrowed but dilatable. If narrowed it is not technically strictured so as to produce a mechanical obstruction.]

The diagnosis during life was cerebral tumour or toxaemia.

History of the Case.1—Patient had suffered from asthma [for how long not stated] and always required to swallow slowly, and this difficulty had increased during the last ten years. She became pregnant in July, 1914, and remained well until January, 1915, when constant vomiting after food began, for the relief of which induction of labour was performed at the thirty-fourth week. Vomiting continued, very often associated with an act of coughing, followed by delirium, nystagmus, deafness, and dimness of vision. Patient was much emaciated. When admitted to St. George's Hospital on February 27, 1918, well-marked bilateral optic neuritis was present. The lungs were distinctly emphysematous, the deep reflexes diminished, and the urine contained a trace of albumin. Patient died thirty-six hours after admission.

(This case was under the care of Dr. Arthur Latham.)

(II) Specimen, Series IX, No. 21a, from the Museum of St. George's Hospital2 (fig. 2, p. 80).

Shown by Mr. Barwell.

Description.—Moderate oesophagectasia. "Removed from a boy, aged 8. The commencement of the oesophagus is of normal size. Below this point the oesophagus is dilated, its circumference when distended measuring 3½ in., and at the post-mortem examination it contained liquid food. It was 9½ in. in length. The muscular coat was hypertrophied. The mucosa was normal. The lower end of the oesophagus was of normal size—a finger easily passing into the oesophagus from the stomach. There was no sign of stricture."

History of Case.3—After an attack of whooping-cough patient began to suffer from choking fits; succeeded by vomiting which resisted all treatment. Death occurred six weeks later. The family history showed a marked susceptibility to vomiting. One child died of vomiting during scarlet fever, whilst another had an attack of vomiting from which it recovered. An uncle died of vomiting believed to be of neurotic origin.

2 Descriptive Catalogue of the Pathological Museum of St. George's Hospital, London.
(This case was under the care of Sir (then Mr.) Edgcombe Venning.)

The segmented appearance of the oesophagus is obviously due to its having been filled out with a series of pledgets of cotton wool before being placed in alcohol. It has been supposed that the foregut is segmented in correspondence with the metameres of the trunk. Fleiner\(^1\) looks upon the dilatation in these cases as congenital, and calls it the preventriculus (Vormagen), and following Luschka has adopted Mehnert’s\(^2\) enteromere theory to explain this. He says “the primary foregut from which the oesophagus is derived is segmented like a rosary, and the musculature may be looked upon as consisting of a number of segments, which Mehnert calls enteromeres, separated from one another by a narrow zone or constriction. Each segment corresponds to a vertebra, and each narrow zone to an intervertebral disk. There are twelve segments and thirteen narrow zones or constrictions. One segment is below the diaphragm and the rest are above.” Fleiner thinks “that from an anomaly in development one or more of these enteromeres may remain, and the muscular fibres, being weak, will give way.” This conception does not rest upon observation; and at the present time embryologists are agreed that the primitive gut is never at any time more than a simple tube. The constrictions shown in the specimen cannot, for this reason, be attributed to such a primitive disposition.]

H. D. Rolleston,\(^3\) who made the post-mortem on this case, has fully described the specimen and discussed the possible causative factor under the title of “Simple Dilatation of the Oesophagus.” He points out that in this specimen there is “no sign of any special hypertrophy of the muscular fibres forming the cardiac sphincter.” To this author\(^4\) has been given the credit of having first enunciated the inco-ordination theory of the disease, for in referring to this case he suggested that “possibly a failure in the co-ordinating mechanism by which the cardiac sphincter is relaxed during swallowing—a kind of stammering as in speech—might give rise to both hypertrophy and dilatation of the oesophagus.” “Paralysis or continued inhibition of the longitudinal muscular fibres of the oesophagus,” he remarks, “would allow dilatation of the tube to occur, and at the same time by interfering with the opening of the cardiac sphincter, would induce hypertrophy of the circular muscular coat.”

A study, however, of the literature reveals the fact that Einhorn\(^5\) (New York) had made this suggestion eight years previously (in 1888). In referring

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\(^1\) Fleiner, “Neue Beiträge zur Pathologie der Speiseröhre,” Munch. med. Wochenschr., 1900, xlvi, pp. 529, 578.


\(^3\) Trans. Path. Soc. Lond., 1896, xlvi, p. 37.


to a case which he recorded of "Dysphagia with Dilatation of the Esophagus" he expressed the opinion that "a lack in the reflex relaxation or opening of the cardia during the act of swallowing" would account for the condition.

(III) Specimen. Series IX, No. 21B, from the Museum of St. George's Hospital ¹ (fig. 3, p. 81).

Shown by Mr. Barwell.

Description.—"Removed from a man, aged 28 years. Esophagus dilated in fusiform fashion over its middle five-sevenths, and contained much modified food. Circumference at widest, 3½ in., corresponding with a circular tube 1 in. in diameter. Mucous membrane shredding and muscle greatly hypertrophied, but, microscopically, no other change visible. At the cardia end it was narrower than elsewhere, but there is nothing approaching to a stricture. There is no abnormal predominance of circular fibres or inferiority in numbers of longitudinal fibres. The stomach is small and contracted, generalized miliary tuberculosis was found, but the total amount of tubercular mischief was not great; the chief seat being in the lungs, especially the left upper lobe."

[In the specimen the lower end of the esophagus is somewhat sharply bent on the adjoining portion of the stomach; presumably from the parts having been hardened in a misplaced condition after death.]

History of Case.—Admitted December 28, 1898, suffering from extreme emaciation, with epigastric pain and tenderness. Twelve months before had had difficulty in swallowing with frequent vomiting, and "tubes were passed" down the gullet. Ingested food was returned immediately and unchanged; vomit acid and containing mucus. Bougies passed as far as 17 in., but no obstruction was encountered. On the fifth day after admission, laparotomy was performed, but nothing abnormal was found in the stomach or elsewhere. Death nine days after admission.

This case was under the care of Dr. Penrose and Mr. Dent.

(IV) Specimen No. 912, from the Museum of St. Thomas's Hospital ² (fig. 4, p. 82).

Shown by Mr. Howarth.

Description.—An extremely dilated esophagus from a woman, aged 60. "The dilatation increases from the upper end to the junction of the middle and lower thirds, where the canal measures 5½ in. in circumference; beyond this it diminishes, until, at the cardiac orifice, the esophagus regains its normal dimensions. The mucous membrane is superficially ulcerated for

¹ Descriptive Catalogue of the Pathological Museum of St. George's Hospital, London.
² Descriptive Catalogue of the Pathological Collection in the Museum of St. Thomas's Hospital, 1892, part ii, p. 10.
considerable areas: the muscular coat, although increased in area by the dilatation, is as thick as natural, from compensatory hypertrophy. The stomach was of the ordinary size and perfectly healthy, and its cardiac and pyloric orifices in every way normal. There was tubercular disease of the liver and bronchial glands. A rod of whalebone has been passed through the cardia."

History of Case.—Death from peritonitis following perforating ulcer of the duodenum.

[Further clinical notes of this case are not obtainable.]

(V) Specimen No. 912A, from the Museum of St. Thomas's Hospital
(fig. 5, p. 83).

Shown by Mr. Howarth.

Description.—"Removed from a male. The lower 8 in. of an œsophagus (unopened) with the cardiac end of the stomach. The œsophagus is dilated throughout—measuring, where most so, 2 in. in diameter—but without any organic obstruction. The muscular wall of the dilated canal presents no signs of atrophy." A stout glass rod has been passed through the cardiac orifice.

History of Case.—Patient suffered from œsophageal obstruction for thirteen years, and ultimately died of inanition.

[Further clinical notes unobtainable.]

(VI) Specimen No. 552, from the Museum of Guy's Hospital
(fig. 6, p. 84).

(This is Sir Samuel Wilks's well-known case.)

Shown by Mr. Mollison.

Description.—"An œsophagus and stomach removed from a patient, aged 74. The œsophagus is greatly dilated. Its walls are of great thickness from the increased muscular development of its coats. It is 11 in. long, and measures 6½ in. in external circumference at its widest part. It is of nearly uniform size throughout, although slightly bulging in two parts. Towards the stomach it suddenly contracts. It is here constricted to half its usual diameter. The cardiac orifice is laid open and measures only 1¾ in. transversely. There is no appearance of fibroid thickening or cicatrization of this orifice."

1 Descriptive Catalogue of the Pathological Collection in the Museum of St. Thomas's Hospital, 1892, part ii, p. 10.

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[The stomach (which has been looped up by the side of the oesophagus and is not shown in the drawing) is much dilated and its walls abnormally thin; the pylorus is quite free from disease. Although, in the original account of this specimen (Wilks, loc. cit.) there is an allusion to a scar, it must be admitted, as stated in the more recent description above cited, that there is “no appearance of fibroid thickening or cicatrization of the cardiac orifice.” In the light of knowledge since gained with regard to dilatation of the oesophagus, there can be no hesitation in placing this case into the group of oesophagectasia of neuro-muscular origin. Both the muscular wall and the mucosa of the cardiac canal are natural in appearance, the longitudinal folds of the membrane being of the kind regularly seen at this spot. It may be pointed out that the prominent horizontal fold at the lower end of the oesophagus is due solely to the latter having been artificially misplaced so as to lie parallel with the long axis of the stomach, the direction of which has been reversed.]

History of the Case.—“Since boyhood, patient had difficulty in swallowing solid food, small quantities of which were regurgitated after meals, so that he might have been regarded as a ruminating animal. He never could take any meal without eating very slowly and masticating very carefully, and at the same time taking a draught of fluid to wash down every bolus of food as soon as it entered the gullet. He always brought back some portion by ‘an act of coughing’ and not vomiting. The passage of bougies relieved the symptoms. The complaint caused no inanition and the patient died of pneumonia.”

(VII) Specimen No. 550, from the Museum of Guy’s Hospital
(fig. 7, p. 85).

Shown by Mr. Mollison.

Description.—“Local hypertrophy of the mucous membrane of the oesophagus. An oesophagus laid open to show upon its mucous membrane numerous raised patches, round and oval in shape, and varying in size from a pin’s head to a split pea. The nodules have a smooth convex surface, and histologically are seen to be due to local thickenings of the epithelium. The muscular coat at the lower end of the oesophagus is seen to be hypertrophied.”

The following additional description has been obtained from the post-mortem notes, No. 160, 1866: The oesophagus was in a very remarkable state, its muscular tissue being very much hypertrophied, especially opposite the enlarged heart; there was, moreover, a warty condition of the mucous membrane.

History of Case.—Patient, aged 62, was admitted to hospital in May, 1866, for albuminuria and dilated heart, and lay in a dying state for many days. At

the autopsy the heart was found to weigh 18½ oz., and the kidneys were granular.

(This case was under the care of Dr. Pavy.)

The muscular hypertrophy in the specimen involves the circular fibres at and above the cardia, for a distance of 5 cm. (2 in.). In the divided edge on the opposite side there is a track of similar thickening, but extending upwards for twice the distance; and even above this the muscular wall is unusually well developed. (In the specimen, the muscle, where thickest, has been cut obliquely, and appears thicker than it actually was.) In this case there is no dilatation. A microscopic examination of the elevations referred to shows that they consist of hypertrophic papillae, with a correspondingly increased production of interpapillary epithelium; the summits of the papillae project slightly from the free surface. The lesions may be classed as sessile papillomata. There is in the Museum of St. Thomas's Hospital a specimen (No. 917) consisting of the lowest 4 in. of an œsophagus, the mucosa of which presents many small circular elevations of similar structure. The whole of the œsophagus was affected. There is no dilatation of the canal, and no hypertrophy of the muscular coat.]

(VIII) Specimen No. 1838, from the Museum of St. Bartholomew's Hospital (fig. 8, p. 86).

Shown by Mr. Rose.

Description.—"An œsophagus with a portion of the stomach, removed from a male, aged 20. A dilatation of the œsophagus commences immediately below the pharynx, and gradually increases to its termination in the stomach. In its lower half it measured nearly 6 in. in circumference. In the upper half of the dilated œsophagus the lining membrane is sound; in its lower half the greater part of this membrane is superficially ulcerated and shreds of it hang in the interior of the tube. Just above the stomach the complete removal of the lining membrane exposes the muscular fibres of the œsophagus, which are here, and on every part of the canal, hypertrophied. The cardiac orifice was free and the stomach was healthy."

(The rugae of the gastric mucosa adjoining the cardiac orifice are obliterated from oedema due to an extension of the infective process which has led to the ulceration of the lower end of the œsophagus. The cardiac orifice (as stated in the description cited) is not strictured; and its mucous lining presents the longitudinal folds natural in the resting condition of the canal.)

History of Case.—Patient had had signs of this disease for about eighteen

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1 Specimen No. 917, Descriptive Catalogue of the Pathological Collection in the Museum of St. Thomas's Hospital, 1892, part ii; Trans. Path. Soc. Lond., 1885, xxxvi, p. 189.

2 Descriptive Catalogue of the Anatomical and Pathological Museum of St. Bartholomew's Hospital, 1882, i, p. 281.
months before his death, with frequent sickness about two hours after taking food, pain and tenderness in the epigastric region, and a feeling as if his food stopped at the lower part of the oesophagus. He died from peritonitis.

(IX) Specimen No. 1522, from the Museum of University College Hospital¹ (fig. 9, p. 87).

Shown by Mr. Herbert Tilley.

Description. — Fusiform oesophagectasia of the whole thoracic gullet. "Removed from a female, aged 35. The oesophagus was greatly dilated and was filled with undigested food, and no stricture could be made out. The oesophagus measured 24 cm. (9\(\frac{1}{2}\) in.) in length. At the upper end the internal circumference measured 4\(\frac{1}{2}\) cm. (1\(\frac{3}{4}\) in.). At the cardia 5 cm. (2 in.). Between these points involving the whole oesophagus there is a fusiform dilatation, which reaches its maximum at about an inch below the middle, where the internal circumference is 13 cm. (5\(\frac{1}{4}\) in.). The wall is slightly increased in thickness at the lower end and becomes somewhat gradually thinner towards the upper part. In the main part of the dilatation the longitudinal rugae are smoothed out, but are present at the upper and lower portion of the tube. In the latter position the mucous membrane is swollen, resembling somewhat the condition in chronic gastritis. A finger could be passed through the cardiac opening of the stomach into the oesophagus, and no resistance could be felt."

[As in specimens Nos. 8 and 10 the dilatation extends to the gastric orifice. In the middle portion of the dilatation there is some loss of mucous membrane, leading to exposure of the circular muscle fibres.]

History of Case.²—Duration of illness two years. Began with attacks of pain in the upper abdomen, with vomiting: such attacks recurring periodically, the pain finally becoming continuous. No hematemesis. Last six weeks: Attacks more violent, and dyspnœa superadded, occurring in paroxysms of half an hour's duration. Patient thin: inspiratory stridor, especially on exertion. Cough. Difficulty in taking solid food. The vomiting was generally brought on by an attack of coughing. Death from broncho-pneumonia.

(This case was under the care of Dr. Martin.)

(X) Specimen No. 4004, from the Museum of University College Hospital³ (fig. 10, p. 88).

Shown by Mr. Herbert Tilley.

Description.—"Removed from a male, aged 55. The oesophagus measured 28 cm. (11 in.) in length. For 6 cm. (2\(\frac{1}{2}\) in.), as far down as the bifurcation of the trachea, there is a slight cylindrical dilatation and marked hypertrophy.

¹ Supplementary Catalogue of the Pathological Museum of University College Hospital.
³ Supplementary Catalogue of the Pathological Museum of University College Hospital.
For 17 cm. (6½ in.) below the bifurcation there is a large fusiform dilatation with circumference of 16 cm. (6½ in.), with marked hypertrophy of the walls in the upper third. For 4 cm. (1½ in.) and below the fusiform dilatation there is a slight cylindrical dilatation with marked hypertrophy. The lowest 1½ cm. (¾ in.) of the tube is normal in calibre. There are no ulcers in the lower part of the œsophagus or cardiac end of the stomach."

[This specimen is of particular interest and value in that it shows a localized overgrowth of circular muscle corresponding with the cardiac canal—a condition present, but to a much less marked degree, in only one other, viz., that in the London Hospital Museum, which is figured by Sir Hugh Rigby in Choyce's "System of Surgery." The longitudinal rugae in the cardiac canal are very pronounced.]

**History of Case.**—Duration of illness eighteen years. Began with a fit of unconsciousness preceded by coughing (occurring immediately after a meal). Complete recovery from this, but, three days later, patient was unable to swallow properly. Had difficulty in swallowing and retaining food off and on ever since, with pain in the chest, not related to meals. Last six weeks general weakness, dysphagia more severe, the food being returned directly after swallowing; pain in chest worse. Last two weeks shortness of breath and cough. Increasing weakness. Patient wasted, rather short of breath, some pain in upper abdomen, with tenderness, cough and bronchitis. Bougie passed into stomach without difficulty; a smaller one subsequently passed caused discomfort and retching. No free HCl in vomit: lactic acid present. Post-mortem: Coronary disease of heart. No other abnormality was present.

(This case was under the care of Dr. Batty Shaw.)

**(XI) Specimen No. 4004A, from the Museum of University College Hospital**

Shown by Mr. Herbert Tilley.

**Description.**—"Removed from a female, aged 51. The œsophagus is 23½ cm. in length (9¾ in.). Its upper and cardiac orifices are of normal size, between these the tube is dilated throughout, its internal measurements being from 6 cm. (2½ in.) circumference at the level of the cricoid to 13½ cm. (5½ in.) circumference at the level of 17 cm. (6½ in.) below the cricoid. At the lower end, just above the cardia, is an oval ulcer about 1 in. in length. The muscle in the lower half of the dilatation is distinctly hypertrophied, but at and for a short distance above the cardia it is normal. The lower tracheal and bronchial glands are enlarged and caseous and adherent to œsophagus. The œsophagus is ulcerated through in two places owing to breaking down of the glands."

[As in specimens Nos. 1 and 8, the dilatation of the œsophagus involves the whole of the canal as far as the gastric orifice.]

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2 Supplementary Catalogue of the Pathological Museum of University College Hospital.
History of Case. 1—Duration of illness, four years. Began with attacks of pain in the chest and vomiting, lasting about a fortnight and intermitting for about two months. The pain started about twenty minutes after a meal and vomiting occurred a few minutes later, with relief of the pain. No haematemesis. Last five months, attacks much more frequent, with irregular fever. Patient wasted and anaemic; tenderness in epigastrium and slight rigidity of muscles here; there was proptosis of stomach, which reached well below the umbilicus. No free HCl in stomach. Death occurred from exhaustion.

(This case was under the care of Dr. Bolton.)

XII) Reserved Specimen, No. H. 49, Post-mortem 487, 1908, from the Pathological Institute of the London Hospital 2 (fig. 12, p. 90).

(This specimen has been traced and included in the Series through the courtesy of Sir Hugh Rigby and Dr. Turnbull.)

Description.—"The oesophagus with a portion of the stomach removed from a female, aged 42. About 10 oz. of grey-brown fluid containing grape stones was present in the ulcerated, dilated, and hypertrophied oesophagus at the post-mortem examination. No primary cause was found in the body for the condition of the oesophagus. No microscopic section was made from the oesophagus."

History of Case.—Patient was admitted to hospital on May 19, 1908, complaining of vomiting and pain after food for many years; worse for the last six months. Death occurred on May 23, 1908, from "marasmus."

(The case was under the care of Dr. Francis Warner.)

Under the title of "Diffuse Dilatation of the Oesophagus with Cardiospasm," Sir Hugh Rigby 3 further describes this specimen: "The muscular coat of the entire wall of the oesophagus is hypertrophied, whilst in the lower three-fourths of its extent marked dilatation has taken place. The mucous coat is also greatly thickened, and scattered over the surface are numerous shallow ulcers. At the cardiac orifice the muscular hypertrophy is most noticeable; the lumen is here greatly diminished, and the mucous membrane is thrown into longitudinal folds. There is, however, no ulceration in this part."

In this specimen the circular muscle of the lower portion of the dilated oesophagus is notably hypertrophied (3 mm.), the thickening reaching a maximum (4.5 mm.) near the lower end of the dilatation. It is to be noted, however, that the thickened wall is involved in the dilatation, and that the thickness of the circular coat at the actual site of narrowing is not obviously increased. In the illustration of the specimen in Choyce's "Surgery," the figure given is somewhat misleading, since it was drawn with the parts in a somewhat unnatural position.

2 This specimen has not yet been catalogued, but has been provisionally indexed as "Idiopathic Hypertrophy of the Oesophagus."
Unmounted Specimen not yet catalogued, from the Pathological Department, Victoria Infirmary, Glasgow (fig. 13, p. 91).

Shown by Dr. Brown Kelly.

Description.—This drawing was made largely from a reconstruction of the parts after they had been damaged in the removal, and is therefore somewhat approximate. A re-examination of the specimen shows that a small portion of the left crus of the diaphragm remains connected with the lower end of the œsophagus; and this serves to locate the phreno-cardiac segment of the canal. The thinness of the muscular wall in this case, as compared with that in others, may be drawn attention to. Slightly larger than half size.

History of Case.—The patient was a woman, aged 56, who had suffered from dysphagia for twenty years. Four weeks before death the obstruction at the cardia had suddenly become almost complete. She was first examined by exhibitor five days before her death, when on introducing the œsophagoscope a large quantity of bismuth in suspension was drained off: this had been swallowed six days previously for X-ray purposes. A tube was passed into the stomach under direct observation, and left in situ. After having been fed through this for two days she withdrew it. She was then able to swallow a little fluid naturally. The same evening she died of heart failure.

The Normal Cardiac Orifice and Canal (figs. 14A, 14B, 14C, p. 92).

Shown by Professor S. G. Shatlock, F.R.S.

Specimens (a), (b), and (c), specially dissected and prepared by Professor Shatlock to show the normal cardiac orifice and canal for comparison with the previous specimens.

Hypertrophic Stenosis of the Pyloric Canal (fig. 15, p. 93).

Shown by Professor S. G. Shatlock, F.R.S.

Specimen No. 943F, from the Museum of St. Thomas's Hospital, for comparison with the cardiac orifice and canal of the œsophagus in Dr. Batty Shaw's specimen.

Specimen from the Royal College of Surgeons.

Shown by Professor Shatlock, F.R.S.

Dissection showing intra-abdominal segment of the œsophagus and crura of the diaphragm. Showing the decussation of the crura above the œsophagus. (A drawing of this specimen was not made.)
Fig. 1.
Unmounted specimen from the Museum of St. George's Hospital.
Fig. 2.
Specimen Series IX, No. 21a, from the Museum of St. George's Hospital.
Specimen Series IX, No. 21a, from the Museum of St. George's Hospital.

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FIG. 4.
Specimen No. 912 from the Museum of St. Thomas's Hospital.
Specimen No. 912a from the Museum of St. Thomas's Hospital.
Fig. 6.
Specimen No. 552 from the Museum of Guy’s Hospital.
Specimen No. 550 from the Museum of Guy's Hospital.
Fig. 8.
Specimen No. 1833 from the Museum of St. Bartholomew's Hospital.
Specimen No. 1522 from the Museum of University College Hospital
Fig. 10.
Specimen No. 4004 from the Museum of University College Hospital.
Specimen No. 4001A from the Museum of University College Hospital.
Fig. 12.
Reserved specimen No. H 49 from the Pathological Institute of the London Hospital.
Fig. 13.
Unmounted specimen, not yet catalogued, from the Pathological Department, Victoria Infirmary, Glasgow.
COMPARISON BETWEEN THE NORMAL PYLORIC AND CARDIAC CANALS.

Fig. 14A.
Normal cardia from a well-developed young male adult (the same subject as fig. 14c. The crura of the diaphragm are shown in their position; they lie immediately above the sphincter.

Fig. 14B.
Normal cardia from a well-developed female of middle age. The white oesophageal epithelium is sharply demarcated from the gastric; the sphincter embraces portions of both areas.

Fig. 14C.
Normal pyloric canal from a well-developed male adult.

Natural-sized drawings from specimens specially dissected and prepared by Professor S. G. Shattock, F.R.S.
Fig. 15.

Specimen No. 943 F. Hypertrophic stenosis of the pyloric canal, from an adult. (In the Museum of St. Thomas's Hospital.) Natural size.
Skiagram showing Reflex Achalasia of the Œsophagus due to the Irritation of a Gastric Ulcer.

By Professor S. G. Shattock, F.R.S.

A skiagram taken after a bismuth meal, from a case of gastric ulcer. The stomach has been moderately filled; the irritation thereupon produced at the site of the ulcer has led either to a reflex achalasia or to cardiospasm. The further entry of the meal is, as a consequence, held up in the œsophagus, the lower end of which is dilated as far as the level of the diaphragm. From a case under the care of Dr. H. G. Turney, in St. Thomas’s Hospital. As the reproduction is from a print made from a negative reduced by photography, and so
converted into a positive, the bismuth is shown white: the normal position of the parts is reversed. On the left side of the dilated end of the oesophagus there appears a portion of the spinal column.  
(Skiagram by Mr. C. L. Wrinch.)

Dilatation of the Oesophagus without Stenosis.

By Robert Worthington, F.R.C.S.

Patient, a Belgian lady, aged 34, was first seen in 1915. She had suffered from difficulty in swallowing for two years, for which she had been treated medicinally in Belgium. She lived almost entirely on milk, but occasionally was able to swallow more solid food.

Oesophagoscopy showed that the gullet was dilated and contained milk curds, together with a piece of macerated meat (about 1 in. in length), which had the appearance of having been in situ for some considerable time.

On evacuating the curds and piece of meat, it was surprising to find how easily a large sized bougie could be passed into the stomach. The following day a great improvement in swallowing was noticed, though it was not by any means normal. Patient unfortunately left the hospital and declined to have any further treatment.

Further news of her shows that she speedily relapsed into her previous condition of chronic dysphagia. She occasionally has crises, when for a few days she is practically unable to swallow anything at all, whilst during the intervals she lives entirely on milk.

This case, in my opinion, entirely disposes of the contention put forward a few years ago that there was no such condition as spasmodic stricture of the oesophagus.

Remarks by Professor S. G. Shatlock, F.R.S.

The following comments, which have received the approval of the two openers of the discussion on oesophagectasia, have been drawn up with the object of indicating what conclusions are suggested from a study of the specimens exhibited at the meeting.
The Thickening of the Muscular Coat.

As shown in the specimens, this varies in degree in different cases; and even in the same specimen, in different zones, or at different levels of the dilatation. There is none in which the muscular wall is atrophic—i.e., absolutely thinner than the wall of the normal œsophagus. In some, the amount of muscular thickening is not merely proportional to the increased capacity of the tube, but is absolutely above the normal. The overgrowth of muscle is to be viewed as of a secondary or compensatory kind, brought about by the obstruction below. In all, therefore, a paralytic condition of the tube may be excluded as an explanation of the dilatation; although, of course, such an ætiology might obtain from disease, either peripheral or central, of its vagal supply; for it may be so produced experimentally. That an atrophic condition, however, might be merely a secondary phenomenon is not to be ignored in this connexion. As long as the blood supply of the dilating tube remains good, the muscular overgrowth would be maintained: when this fails, as from disease of the supplying arteries, immediate or remote, the dilatation would proceed with atrophy. These results would be strictly comparable with those which occur in the muscular wall of the bladder in obstruction due to enlargement of the prostate.

One of the interesting things arising out of the examination of the specimens is, that in one there is a well defined and pronounced hypertrophy around the terminal part of the œsophagus; viz., in one of the three from University College Hospital. The overgrowth is confined to the circular fibres.

[On the ground that there is evident hypertrophy of the phrenocardiac portion, Dr. Hill would not include this as a case of functional stenosis without anatomic lesion.]

The only other specimen suggesting an overgrowth of the cardiac sphincter is one from the London Hospital, figured by Sir Hugh Rigby in Choyce’s “System of Surgery.” The figure there given is somewhat misleading, however, in that the parts have not been straightened out to show their proper relations. When this is done (as shown in the drawing, fig. 12, p. 90), it is seen that the thickened circular muscle lies almost entirely above the actual constriction, and in the wall of the lowest part of the dilated œsophagus. In this specimen, the thickening of the circular muscle, if regarded as a sphincter hyper-
trophied in thickness and in length, is now incorporated in the dilated termination of the oesophagus. This apparent anomaly can only be explained by supposing that the chief part of the sphincter, which may at one time have closed the cardiac orifice or canal, has slowly given way under the distending force from above. It is therefore remarkable that the musculature of the terminal portion of the canal below has sufficed to maintain the obstruction.

[It may be observed, in passing, that the sphincter vesicæ in the cat's bladder, although insignificant in size, is sufficient to prevent the distended bladder being emptied by firm pressure made with the hand, when the extrinsic action of the urethral muscle in front is excluded by the passage of a catheter through the urethra but not actually into the bladder.]

As the exact position and dimensions of the cardiac sphincter are matters of importance in connexion with the subject of oesophagectasia, two anatomical specimens have been specially prepared, male and female, and accurately drawn in order to elucidate the subject. Fig. 14A (p. 92) is from a well developed young adult male, who died after an operation for cerebellar tumour. The parts were carefully dissected after removal; a narrow cylinder of wet cotton wool was lightly drawn through the lower part of the oesophagus from the stomach; and the specimen, laid on cotton wool, was hardened in formol solution, and afterwards bisected in the coronal plane. Fig. 14B is a similarly prepared specimen from a well-developed female of middle age, who died suddenly from pulmonary embolism after hysterectomy.

One thing noticeable in making the preparations was the ease with which the cardia could be displaced through the opening between the crura, the connexion of the structures concerned being of the most delicate kind. Attention may be drawn to the thickness of the crura as well as to their close apposition to the lower end of the oesophagus, the relative positions of the different structures being carefully preserved. A study of the drawings will show that the thickening of the circular fibres forming the sphincter embraces part of the stomach as well as the end of the oesophagus, and that it lies quite below the diaphragm. In fig. 14B the dividing line between the oesophagus and the stomach is sharply brought out by the greater opacity of the thicker, squamous-celled epithelium of the former. The sphincter, moreover, is not a simple ring, but a fusiform thickening of sufficient length to justify the use of such a term as the cardiac canal rather than that of cardiac orifice. If compared with the pyloric sphincter (fig. 14c),
the latter has an abrupt termination on the distal aspect, where it produces an annular elevation within the lumen; but on the proximal side the thickening of the circular muscle tails off around the pyloric canal, much as in the case of the cardiac sphincter.

If overgrowth of unstriped muscle be taken as an indication of abnormally forcible contraction, the dilatation in the case from University College referred to (p. 76) may be ascribed to cardiospasm [in the sense, Dr. Hill remarks, in which Mikulicz employed the term—viz., hypertrophic stenosis following on a primary functional spasm].

The hypertrophy in this case is remarkably like that met with in hypertrophic stenosis of the pyloric canal, as seen in infants; and more so when this stenosis is found in the adult. Of the last there are two examples in St. Thomas's Hospital Museum (Nos. 967, 943f). In both, the mucosa is intact; the muscular thickening is confined to the circular fibres; and in one, certainly (967), the stomach has been dilated from pyloric obstruction. Of No. 943f a drawing has been introduced (fig. 15). It came from a man, aged 61, admitted for mitral stenosis; he had had malaria, and there was a history of rheumatic fever; nothing in regard to gastric trouble is mentioned in the notes. In the case of the University College specimen, the patient was 55 years of age; symptoms had existed for eighteen years. In Sir Hugh Rigby's case, the patient was 42, and symptoms had been present "for many years." Whether hypertrophic stenosis of the pyloric canal may not arise at a later date than usually assumed is a matter needing further observation; if so, the two conditions would be still more parallel. No similar overgrowth of the cardiac sphincter is present in any of the other specimens. In explanation of these, a want of co-ordination between the propulsive action above and the relaxation of the sphincter below may be the more correct explanation; and it is this which the term "achalasia" is designed to connote: for, clinically, there is no palpable hindrance to the passage of the bougie through the cardia. When the two phenomena are compared, there is, perhaps, no very great difference between them. In the second case, the obstruction is due to the tonic contraction of the cardiac sphincter; and in the first, the contraction is augmented, or hypertonic—i.e., it is a proper cardiospasm. Nevertheless it conduces to accuracy to draw the distinction if there are grounds for making it.

Next, in regard to the part played by the diaphragm. Whatever can be distinctly conceived is possible, and it may be that the neuro-
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muscular fault in certain cases lies in the diaphragm and that the obstruction is extrinsic. The skiagrams undoubtedly demonstrate the presence of an undilated segment of the oesophagus above the stomach, the upper limit of which segment corresponds with the superior surface of the diaphragm. This, it may be submitted, however, does not prove that the diaphragm is the cause of the obstruction. The obstruction, one may still think, is below at the cardia; the tone of the diaphragm disallowing the dilatation of the included part of the tube. When, in the course of time, the increasing pressure of the accumulation above overcomes this extrinsic support, then, as shown by the skiagrams and specimens, the dilatation involves the whole of the canal as far as the sphincter, which nevertheless remains capable of holding up the oesophageal contents. The term "phreno-cardiac segment," as used by Dr. William Hill, is very appropriate as applied anatomically in the present discussion. That an inhibitory relaxation of the diaphragm immediately around the oesophagus accompanies the dilatation of the cardiac sphincter is not improbable, judging by analogy from what occurs in other positions; and it may prove that failure in the second is accompanied with failure in the first, although, per se, the tone of the diaphragm would be ineffective as a cause of obstruction.

That a reflex may arise in the stomach, which will prevent the entry of ingesta, may be taken as established, and is illustrated by such a case as the following: A patient was in St. Thomas's Hospital suffering from a gastric ulcer. Following a bismuth meal a skiagram showed that after much had passed into the stomach, the material was held up in the oesophagus, which became dilated, the dilatation terminating inferiorly immediately above the diaphragm. Here the achalasia arose as a reflex transmitted from the stomach. There is no evidence, however, that gastric conditions, either organic or functional, are necessary aetiological factors in producing neuro-muscular oesophagectasia. On the contrary, the direct observation made by Dr. Brown Kelly proves that the reflex fault may lie in the oesophagus, since the experimental irritation of the mucosa above the cardia, in place of being followed by relaxation of the sphincter, as it is normally, was followed by its closure.

Lastly, amongst the drawings there is inserted one showing a distinct and widespread muscular hypertrophy, especially of the circular layer, but unaccompanied with dilatation; the heart was much dilated, and weighed 18½ oz. A similar condition is shown in a second specimen from Guy's Hospital Museum, described by Dr.
Newton Pitt. Here the heart was likewise enlarged. The muscular thickening of the oesophagus was most marked an inch above the cardiac orifice. Such results have been attributed to obstruction caused by the pressure of the enlarged heart. Aortic aneurysm may undoubtedly produce oesophageal obstruction and some local dilatation of the canal above.

In connexion with muscular hypertrophy of the oesophagus without dilatation it is enough to point out that it may represent a completely compensated achalasia, or even cardiospasm—a possibility already conceived by Dr. Brown Kelly. This could only be established by finding such hypertrophy unassociated with aortic aneurysm, enlargement of the heart, or other extrinsic organic obstruction.


Section of Laryngology.

President—Dr. James Donelan.

Endothelioma of Left Tonsil; Operation performed. ¹

By Andrew Wylie, M.D.

Patient, female, aged 56, was shown at the November meeting with large swelling of left tonsil, which exhibitor considered to be sarcoma, but the pathologist reports after the growth was examined that it is a rapidly growing tumour of the class sarcoma and the variety endothelioma.

On Tuesday, November 5, laryngotomy was performed and the anaesthetic administered by Dr. Kingsford through the tube. The pharynx was plugged with two sponges and the growth removed in two large pieces and several smaller ones, chiefly with a snare. It shelled out fairly easily. The pillars of the fauces were drawn together with sutures which controlled the haemorrhage. The laryngotomy tube was removed at the finish of the operation, and the patient discharged cured in ten days.

The result is satisfactory, and the patient has increased in weight.

Dr. Wyatt Wingrave reports:—

Macroscopical.—Tumour consisting of one chief mass and several small ragged portions, the whole weighing 50 grm. In consistence it was somewhat soft and friable, resembling tonsil tissue.

Microscopical.—A true endothelioma originating in the cells, lining the blood-vessels and capillaries. The earlier groups are arranged as hollow cylinders supported by an abundant stroma of fibroid or cellular elements. In parts the stroma shows true embryonic cartilage somewhat resembling the mixed tumours of the parotid. There is an ill-defined boundary of fibroid tissue, but scarcely worthy of being termed a capsule.

¹ At a meeting of the Section, held February 7, 1919.
DISCUSSION.

Mr. Dawson: I had what appeared to be a similar case a year ago. The tumour was so large that it protruded to the other tonsil and half filled the mouth. The general practitioner thought it was an abscess of the tonsil, and attempted to open it, but without any result. I was obliged to do a tracheotomy hurriedly. Later, I removed it in the same way as Dr. Wylie has done here. There was surprisingly little bleeding. There was no capsule, but the tumour was very friable and soft, and it shelled out. Six months later the patient seemed to be all right.

Dr. Douglas Guthrie: In the Journal of Laryngology last October¹ I reported a similar case, together with two early cases which were under the care of Dr. Logan Turner. Mention was made of seventy-seven cases of sarcoma of the tonsil, collected from literature, in which the diagnosis had been verified by microscopic examination. My case was more advanced than Dr. Wylie's; the growth had invaded the posterior pillar and involved the glands, so that prognosis was bad. I have here a water-colour drawing of the pharyngeal appearance. (Drawing shown.)

Dr. William Hill: Mr. Norman Patterson and I reported a case some time ago—an endothelioma in this region. We applied diathermy to the base, and as that coagulated much outlying tissue, we did not feel inclined to follow up that procedure with the knife. We first tied the carotid, in order to diminish the haemorrhage. Still, diathermy after removal is a good precautionary measure in cases in which a return of the growth is probable, which is especially the case when they are of such large size.

Mr. Norman Patterson: The case referred to by Dr. Hill was an epithelioma. In the case of a mesoblastic tumour, I prefer to use radium before trying anything else.

Dermoid Fistula of Nose.

By W. Stuart-Low, F.R.C.S.

A female who for years has complained of a periodical discharge from a minute fistula over the middle of the dorsum of the nose. This seems to come from a medianly situated retention cyst, most probably a dermoid judging from the long history and situation. It is proposed to excise the cyst through a longitudinal incision.

DISCUSSION.

Dr. Brown Kelly: I showed three cases of this sort at the Scottish Society of Otology and Laryngology, and they were reported in the Journal of Laryngology, 1913, p. 498. One of the chief differences between Mr. Stuart-Low's case and mine is the low situation of the orifice in the former. In this patient another congenital defect is present, namely, insufficiency of the palate. My attention was directed to this by the patient's open nasal speech. There is no submucous cleavage of the hard palate: her insufficiency is due to defective muscular action.

Dr. Perry Goldsmith: I suggest that in attacking this apparently very simple condition the operator does so with much hesitation, so far as expectation of cure is concerned. It seems to be a simple matter to cut down and remove the sac, and if it is very limited and directed a short distance under the skin, well and good: but years ago I had to operate on a similar case, and found that it extended along the side of the nose. Three fistula resulted altogether, and I did not cure my patient.

Dr. Kelson: I have had several similar cases. They appear to result from incomplete fusion of the mesial-nasal processes in the embryo. Curing them is sometimes quite easy, sometimes very difficult. Two of mine presented no difficulty, but in a third, a girl, three operations were necessary before a cure resulted. You follow up and excise the sinus and the wound heals. You may think you have cured it, but after a time it breaks out again. A definite dermoid cyst may or may not be present. Most cases occur in women and you are "between the devil and the deep sea," as you must make a sufficiently free dissection to cure the affection yet not disfigure the patient.

Mr. Frank Rose: I, too, have had a similar case, in which, when I first saw the patient, the external nose was considerably swollen: it was the size of a large cherry, and very red. On making the incision, clear fluid and some pus came away, and then, as in the present case, a small fistula appeared. I made several efforts to obliterate it, but I did not succeed in getting rid of the lining entirely: enough always seemed to remain for another collection of fluid to form. One device, which I thought would succeed, was to open the cyst internally, so that it should drain into the nose. This was not a success: the opening closed, and the cyst again re-formed.

Mr. W. Stuart-Low (in reply): I shall make a point of operating very carefully in this case so as to endeavour to obtain a good result. Some years ago I showed a case here in which the dermoid cyst was a large, long one attached to the bone and dura mater in the middle frontal region. I dissected it off with a very successful result as there was almost no disfigurement and no recurrence. The late Mr. Betham Robinson showed a case about the same time in which the cyst was attached to the longitudinal sinus—he had dissected it off with great skill and success.
Epithelioma of the Epiglottis.

By W. Stuart-Low, F.R.C.S.

A man, aged 45, with extensive epithelioma of the epiglottis extending to the base of the tongue and sides of the pharynx. He has suffered from increased pain on swallowing during the last three months. His trade, that of a boiler-cleaner, is dusty, and he is a mouth-breather owing to nasal obstruction—namely, a deviation of the septum and hypertrophic rhinitis. When he first came to the hospital his mouth was very septic from carious teeth and pyorrhoea alveolaris, and the secretions were found to be very acid. The pain has been much relieved since he has used antiseptic mouth-washes and gargles, and especially relieved from the use of mucin elixoid (Burroughs Wellcome and Co.) as a mouth-wash, gargle, and to sip. He has been a great smoker all his life, and has been addicted to very hot and very salt food. The Wassermann reaction was found to be positive.

DISCUSSION.

Mr. Whale: What was the condition when Mr. Stuart-Low first saw the patient? He says there was dysphagia for three months, and the mouth was septic: he does not say how big the growth was when he first saw it, or whether there were any enlarged glands, so that we do not know whether this growth was ever considered operable.

Mr. W. Stuart-Low (in reply): In the early stages there were no enlarged glands at all. I have at the present time eight cases of epithelioma of the pharynx under treatment, and it is an undoubted fact that there is an alarming increase of this affection in this particular region. It might be helpful and enlightening to have a discussion on this subject.

Recurring Spheno-choanal Polypus in a Child.

By Irwin Moore, M.B.

Patient, a little girl, aged 8, was previously shown at the meeting of this Section on February 2, 1917, when 6 years of age, following the removal of a post-nasal polypus (microscopically shown to be a soft fibroma), having its origin in the left sphenoidal sinus. The case gave

rise to considerable discussion on account of its exceptional interest and rarity, and the exhibitor reported that he had been able to trace the record of two cases of similar growths in adults, but none during childhood.

The patient is again shown (two years later) after the removal of a recurring growth from the same sinus.

It is interesting to note that whereas on the previous occasion the growth consisted of two polypi (a nasal and post-nasal portion) attached by separate pedicles to a common stalk, which originated in the left sphenoid sinus, on this occasion the recurring growth consisted of two polypi on separate pedicles, as follows:

**Report on Specimens by Professor S. G. Shattock, F.R.S.**

"(1) The larger growth which is somewhat oval in form, measures 26 mm. (1 in.) by 22 mm. (7/8 in.) in diameter, and is furnished with a short, somewhat broad pedicle, at one corner of the divided end of which there is a minute scale of bone.

(2) The second tumour is much smaller (12 mm. × 8 mm.) and has a remarkably long cord-like pedicle, 30 mm. (1\(\frac{3}{16}\) in.) in length."

On this occasion it has been possible to locate the origin of the pedicle attached to the larger growth—to the lower margin of the sphenoidal ostium—the piece of bone attached to the pedicle consisting of a portion of the anterior-inferior wall of the sinus.

It is obvious, from this case, that the additional term "solitary" cannot properly be applied to these growths.

Further research of the literature shows that a total of only nine cases of spheno-choanal polypi have been recorded, and these were all in adults between the age of 20 and 30 years—viz., Kubo (Japan), four cases; Moure (Bordeaux), two cases; Massei (Naples), Sippel (Würzburg), and Syme (Glasgow) one case each.

This case exemplifies the common recurrence of these antro-choanal polypi in cases in which radical operation upon the antrum is not performed and the polypoid mucosa lining the cavity is not removed.

This specimen is now in the Museum of the Royal College of Surgeons.\(^1\)

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\(^1\) It was exhibited at the Museum of the First Annual Summer Congress of the Section of Laryngology at the Royal Society of Medicine, May 2 and 3, 1919.
Irwin Moore: *Recurring Spheno-Choanal Polypus*

**Fig. 1.**
Spheno-choanal polypus. Shows a sagittal section through the left naris with the growths placed in position.

**Fig. 2.**
Shows attachment of the flattened pedicle to the lower margin of the sphenoidal ostium. (Dotted line represents upper margin of the ostium.)
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(Original drawings of the recurring growth (figs. 1, 2) exhibited on the epidiascope and compared with the previous growth removed, also an X-ray photograph exhibited, showing the opaque condition of the left ethmoid region before removal of the growth.)

DISCUSSION.

Dr. Syme: I showed a case of spheno-choanal polypus before the Scottish Ear, Nose and Throat Society. In that case I traced the origin to the lining of the sphenoidal sinus. In regard to the general question of choanal polypus, I think we have been too ready to follow a lead. In my experience of antro-choanal polypus there is concomitant (more or less general) disease of the lining membrane in most cases. On another point I am at variance with the generally accepted view. Certainly there is not in all cases an accessory ostium. Indeed I should say that in the majority there is not. On the other hand I have found an accessory ostium in ordinary antral disease without a choanal polypus. I advise that the antrum should be opened in all cases of antro-choanal polypus.

Dr. Dan McKenzie: With regard to the evolution of these polypi, obviously their shape and their position is postulated by the fact that they are edematous mucosa tumours which have grown out from the cavity round a sharp edge. It is the combination of the sharp edge, which constricts the neck of the tumour, and the fact that gravity acts upon it: so one gets a balloon-shaped tumour, with a long pedicle. I do not think there is any mystery in regard to their production. In the case of the antrum, the polypus obtains its entrance into the free cavity of the nose through the accessory opening, and into the sphenoid sinus, through the ostium. One does not find a polypus of this shape coming from the ethmoidal region for two reasons: first, polypi growing there grow more freely, they are not constricted; secondly, even if they were constricted at their origin, the growth of the tumour would be sufficient to destroy the cell walls, while in the ostium of the sphenoidal sinus and in the accessory opening of the antrum you have thick bone too hard and too dense for the polypus to disrupt.

Dr. Brown Kelly: I think that an ostium accessorium is usually present in cases of nasal-antral polypus, because a cystic lining membrane from which polypi of this kind develop is often associated with an accessory opening.

Dr. Irwin Moore (in reply): I am glad that in cases of recurrence of antro-choanal polypi Dr. Syme performs the radical operation on the antrum. Some members are of the opinion you can go on dealing with them by merely pulling them out. I hope to keep in touch with this little girl to see how many recurrences she has, before more fully opening the sphenoidal sinus and gently curetting the lining membrane. I agree with Dr. Dan McKenzie
Displacement of the Lateral Cartilages of the Nose.

By Irwin Moore, M.B.

Patient, a youth, aged 17, has suffered from enlargement of the extremity of the nose for four years, accompanied by periodic attacks of localized hyperhidrosis and numbness. This condition is supposed to have followed the removal of a growth from the root of the nose. Marked hypertrophy of both middle turbinals was present, but this has recently been removed with considerable improvement in the hyperhidrosis. Opinions are invited as to the best method of improving the appearance of the nose.

Case of Long Frænum Linguae.

By Irwin Moore, M.B.

Boy, aged 13, with an excessively long frænum linguae, which enables him to roll back and insert the tongue into the post-nasal space. Patient has a large pad of adenoids.

So-called Malignant Mixed-parotid Tumour.

By W. S. Syme, M.D.

The patient is a female, aged 50. She was first seen on May 20, 1915, when she gave a history of sore throat for about three years, with a slowly increasing swelling on the left side. On examination a swelling was seen involving the left side of the soft palate and bulging that side
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of the palate well towards the middle line. There was no ulceration; apparently the tonsil was not invaded. On palpation it appeared firm in places and soft and almost fluctuant in others. On May 24, 1915, it was removed under chloroform. It was found to be encapsulated. The deeper surface of the capsule was rather firmly adherent to the underlying muscle and the capsule gave way at this part. There has been no recurrence. The tumour, about the size of a hen's egg, is shown.

Dr. Syme: I do not know whether there is any cartilage. Dr. Wilson, the pathologist at the Western Infirmary, said it was malignant, a mixed chondro-endothelioma. Probably most of these cases go to the general surgeon, which accounts for the rarity with which we see them.

Removal of the Larynx for Malignant Disease.

By W. S. Syme, M.D.

Male, aged 48. The patient was first seen at the Glasgow Ear, Nose and Throat Hospital two years ago, when he complained of hoarseness. This was due to some thickening in the region of the cords and in the subglottic region. With the aid of the suspension apparatus a portion was removed which was reported by the pathologist to be simple overgrowth or pachydermia. He was seen once or twice afterwards, and then disappeared. A year later, at the beginning of February, 1918, he was admitted to the Paisley Royal Infirmary suffering from severe dyspnoea. Tracheotomy was performed at once by Dr. Tellet. I was asked to go to Paisley to see him. It was difficult to get a good view by the indirect method, so he was again examined with the help of the suspension apparatus. The larynx appeared to be completely filled with new growth. A portion was removed and examined by Dr. Haswell Wilson, who reported it to be squamous epithelioma. There was no glandular involvement. On February 21, with the help of Dr. Gibb and Dr. Tellet, the whole larynx was removed under chloroform. The growth had just broken through the cricothyroid membrane. It appeared to have originated in the subglottic region, and it extended well down into the trachea especially posteriorly, so much so that it seemed doubtful if it would be possible to get below the disease and yet leave sufficient trachea to be brought forward. This was, however, accomplished. No enlarged glands were found. The cut
end of the pharynx was stitched to the root of the tongue and the floor of the mouth. The patient was fed by a tube, passed through the nose for five weeks. At first there was some leakage from the pharyngeal wound. Except for this he made an uninterrupted recovery, in great part due to the very careful nursing. There has been no recurrence. He is now in very good health, is very cheerful, and works regularly at his trade as a skilled mechanic. He has developed a good pharyngeal voice, has no difficulty, he says, in making himself understood by his family, and even strangers can make out what he says.

Case showing Method of Repair of Right Side of Nose.

By Somerville Hastings, M.S.

Private H. W. L. The repair of the right side of the nose has been carried out by turning forward flap formed of cartilage and bone of septum and mucous membrane of the right side only, the left remaining intact as in the operation of submucous resection, and at once covering the exposed cartilage and bone by a pedicled flap from the forehead. At a subsequent operation the base of the pedicle was divided and the patency of the nose restored by dividing the septal mucous membrane of the right side in the line of its reflection. (See opposite page, figs. 1-4.)

A Fork accidentally Swallowed and Impacted in the Pylorus.

By William Hill, M.D., and K. A. Lees, F.R.C.S.

Remarks by Mr. Lees.

This Irish girl, a domestic servant, aged 25, had been in England only a week when this happened, last Christmas Day. She had recently lost most of her upper teeth, and had not yet had a plate put in, so her powers of mastication were very poor. After eating a portion of giblets she was seized with a violent feeling of indigestion and she vomited, and the undigested portion came up and stuck in her throat, leading to some dyspnoea. She therefore hastily seized the nearest object, which was this fork, and, holding it by the prongs, she pushed the handle behind her tongue and down her throat. She succeeded in moving the piece of meat and withdrew the fork,
Fig. 1.
Before operation, full face.

Fig. 2.
After operation, full face.

Fig. 3.
Before operation, side face.

Fig. 4.
After operation, side face.
but as she felt it was still sticking in her gullet, she proceeded to try the same manoeuvre a second time, and this time she got the fork, according to her own description, a long way down. Then, to her surprise, the handle of the fork was seized by an "unseen power"—probably the constrictor muscles—and having only a slimy hold on the fork, she lost grip of it and it went down. She went to the hospital on Christmas afternoon, where her story was received with diffidence. Unfortunately, the X-ray department was not working on that day, and it was two days before a plate could be got to prove that she had swallowed a fork. The plate shows the fork in the stomach, with the handle resting near the pylorus, and the prongs towards the cardia. I operated upon her fifty-four hours after she swallowed the fork, making a small incision in the epigastric region to the right of the mid-line, and making a \( \frac{3}{4} \) in. incision in the anterior wall of the stomach, near the pylorus. I seized the prongs of the fork and extracted it. The stomach had made an attempt to pass it on: the handle was in the duodenum, and the hilt of the fork in the pylorus, the prongs in the stomach. Suture was done in the ordinary way, and she made a very successful recovery, going out in a fortnight. Later she said she felt no ill-effects and had no indigestion.
Case of Infiltration and Ulceration of the Vocal Cords
(previously shown).

By W. H. Jewell, M.D.

E. H. W., male, aged 56. This case was shown at this Society in 1917, under the title, “Infiltration and Ulceration of the Vocal Cords. Malignant?” when there was a division of opinion as to malignancy and advisability of operating. When first seen the left cord and the anterior one-third of the right cord were covered with a yellowish glazed deposit; this cleared away from the left cord leaving a raw surface. The right cord has remained much the same as when first seen. During the last two months the movement of the left cord has become much impaired. The patient has been hoarse for about three years. The Wassermann test was negative on two occasions about twelve months ago. He has been a heavy smoker and drinker for more than twenty years.

DISCUSSION.

Mr. Whale: Dr. Jewell has had to leave. He has shown the case before, and he inclined to the view that it was malignant. Sir StClair Thomson is uncertain as to its nature. I think it is a gumma.

The President: I had an opportunity of seeing this case before the patient came here. He was sent to me by another doctor, who did not know he was under Dr. Jewell, and this doctor reported a positive Wassermann on January 28. It looks malignant: the age of the man, the fixation of the cord, and the general appearance, favour such a diagnosis.

Dr. Syme: With the aid of the suspension apparatus, a piece of the tissue might be removed for microscopical examination.

Postscript.—Since last meeting the patient has had pain in swallowing owing to a small ulcer in the party wall. A third Wassermann test has proved negative.

Perithelioma of the Right Maxillary Antrum; Radium Treatment.

By J. Gay French, F.R.C.S.

F. W., a female, aged 43, whilst residing in Russia, in November, 1915, consulted a specialist for a small growth in the right nostril. Radium treatment was advised, and carried out daily for two months. On her return to this country in August, 1916, she was seen by Mr. Tilley, who removed a "polypus" from the same nostril.

In September, 1916, when she was in Paris, the "polypus" began to grow rapidly, and patient attended the British Hospital for two years, and during this period twelve operations were performed, the last being done in 1918.

In September, 1918, the "polypus" became inflamed, and was treated by radium continuously for forty-eight hours, with some improvement for a month, followed by increased growth. At this time the patient complained of headache, a continuous nasal discharge, with complete loss of smell.

On January 23, 1919, she was first seen by the exhibitor at the Royal Free Hospital, when a large amount of polypoid-looking material was removed from the right naris and antrum.

Patient is now under the care of Dr. Knox at the Cancer Hospital, where she is being treated by a series of exposures to radium.

Pathological Report.—"Sections show a densely cellular mass—rapidly growing and certainly malignant. In parts the growth consists of a dense collection of round cells, resembling an atypical sarcoma. In other parts the cells are oval and form spaces filled with blood—suggesting that the growth arises from some tooth structure and is really epithelial in origin."—M. Schofield.
Section of Laryngology.

President—Dr. James Donelan.

Cases, Casts, Photographs, and Diagrams, illustrating some Methods of Repair of Wounds of the Nasal Cavities and Nasal Accessory Sinuses.¹

By G. Seccombe Hett, F.R.C.S.

Case I.—This case shows the method of support of the new tip and alæ by means of skin only. The injury was one of the Indian mutilation type. The ordinary Keegan operation was performed. The support of the tip depends entirely on the spring of the turned down skin flaps. A feature of the forehead flap was that a very broad columnella was cut and turned upon itself so that the sides of the columnella form the anterior part of the new septum. Subsequently a cartilaginous tip was inserted. Note the full thickness of skin-graft covering the area on forehead from which the flap was taken.

Case II.—Illustrating a method of supporting the new tip and alæ by means of a submucous advancement of the cartilaginous septum. This case was one of the Indian mutilation type, but with less loss of septum and alæ than in Case I. The bisected triangle of skin was turned down after the Keegan-Smith method. The anterior edge of the remains of the septum was incised and a submucous resection of the cartilage performed. The cartilage remained attached below at its anterior inferior portion. It was advanced and swung forward. The reversed skin flaps from the bridge were attached to the edges of

¹ At meetings of the Section, held February 7 and March 7, 1919.
the septal mucous membrane. A forehead flap was brought down. Subsequently a cartilaginous tip was inserted.

**Case III.**—(a) Support for the new nose formed by an upper septal swing; (b) support and mucous lining for the side of the nose formed by swinging forward the inner wall of the antrum; (c) opening into antral cavity closed by means of a reversed pedicle flap. An L-shaped septal swing was first done. The inner wall of the antrum was rotated inwards and forwards so that the antral mucous membrane formed the mucous lining for the side of the nose. The outer surface of the supports were denuded of mucous membrane at the next operation and a forehead flap brought down. A lateral flap was turned up on the right side to form the lining of the new right ala. An opening from the front of the cheek into the antrum remained. This was filled up by turning in scar tissue to form a lining and then instead of returning the pedicle its proximal end was cut free and the end of the reversed pedicle brought down to cover over the deformity in the cheek. Subsequently the intermediate portion of the pedicle was removed. Finally a cartilage graft was inserted to give more prominence to bridge and tip.

**Case IV.**—Support and mucous lining for the rhinoplastic closure of a cavity in the side of the nose formed by a lateral septal swing. A piece of metal, the size of the opening in the side of the nose, was cut and placed against the left side of the septum. This being used as a guide, the septum was incised and swung laterally, remaining attached only at its anterior portions. It was sutured to the freshened edges of the wound. This case is shown before the skin flap is brought down, so that it may be noticed how healthy the mucous membrane remains although the septal swing has been almost severed from its attachments.

**Case V.**—Result of plastic operation for deformity caused by gunshot wound involving loss of anterior wall of frontal sinus, root of nose and supra-orbital ridge.

**Case VI.**—Repair of wound of side of nose, orbit and antrum. The three cavities were thrown into one, owing to loss of floor of orbit, antral walls and side of nose. The plastic operation consisted in filling up the cavity by folding in an epithelialized forehead flap.
Methods of Repair of Wounds of the Nose and Nasal Accessory Sinuses.

[PART I.]

By G. SECCOMBE HETT, F.R.C.S.

Restoration of an Ala.

There are many methods of restoring a lost ala nasi. A flap may be brought down from the side of the nose or one may be turned in horizontally from the cheek. The flap may contain a previously imbedded piece of cartilage or be attached over an inferior turbinal which has been swung across the gap. Skin may be turned down from the
nose to form the lining of the new vestibule. These methods are designed to give support to and form the lining of the ala.

Although it is easy to make an ala it is not so easy to make a really satisfactory or natural one. The outer portion of the ala often tend to contract upwards, so that the contour is spoilt. A method of forming the ala skin flap which I have found satisfactory is the one depicted in fig. 1. The flap has its base above, and does not tend to contract upwards. After being cut, it is rotated inwards and attached to the nose over turned down skin or turbinal, and may, if necessary, contain imbedded cartilage.

Figs. 2 and 3 show a case before operation, and the result obtained. A small piece of skin has usually to be removed subsequently, at the base of the flap where there is a redundancy.
Restoration of Alæ, Tip and Columnella.

Where alæ, tip and columnella have to be formed, an extension of the last flap may be employed. The flap is cut in a Y-shaped form, the upper limb of the Y making the columnella and support for the tip. The lower limb forms the ala of the opposite side. The portion between the short limbs of the Y is made to include some fatty tissue, and forms the tip—the pedicle of the flap forms the ala of the same side.

Fig. 4 shows the incision employed, and figs. 5 and 6 the case before and after operation. This method was designed by Captain Aymard, previously attached to Queen's Hospital.

Indian Mutilation Type.

Where there is still greater loss of alæ, tip and columnella as in the Indian mutilation type, a forehead flap is indicated. This type (so-called because the cutting off of the nose of their wives by
jealous Indians generally results in this deformity) has been a not uncommon result of a wound of the nose in the present war.

Keegan, who operated on over sixty cases in India, used a very ingenious method of forming a support and lining for the lost parts. He turns down a bisected triangle of skin from the bridge of the nose and sutures it to a small flap in the region of the nasal spine; a forehead flap is then brought down to cover this.

The case shown is one I did by the Keegan method. A modification which I used was to cut a very broad forehead columnella folded on itself, so that the latter forms the sides of the anterior portion of the new septum. The redundant tissue of the columnella was subsequently cut away by a median incision in order to improve the profile (see figs. 7, 8, and 9).
Smith, who worked with Keegan, devised a useful modification of the original Keegan operation by twisting round the edges of the turned down skin flap and attaching them to the mucous membrane of the remains of the septum, which he split for this purpose. I have employed a still further addition to the Keegan-Smith operation. It is as follows: The mucous membrane over the septum is divided, and the mucous membrane is raised as in a submucous resection. The septal cartilage is then divided submucously, except at its anterior inferior angle, and swung forwards—in fact what I call a submucous lower septal swing is adopted. The turned down skin flaps are then attached to the septal mucous membrane as in the Keegan-Smith operation. Finally a forehead flap is brought down with a very wide columnella, which wraps round and covers the projecting piece of septal cartilage (see figs. 10, 11 and 12).
The next type is one of extensive loss of the nose with an opening between the antrum and the cheek. The method of repair adopted illustrates several principles (see fig. 13). The support of the bridge was formed by an upper septal swing.

Stage 1.—An L-shaped piece of septum covered with mucous membrane was cut free, except above, where it remained attached. It was swung forwards, and the end of the short limb of the L was attached to a groove cut in the region of the nasal spine (see fig. 14). A full thickness flap was cut out of the nasal cavity so that it swung forwards like a door; the hinge corresponding to the anterior edge. Its free edge was attached to the septal swing, and filled up the right half of the gap in the nose. The lining of the right half of the external nose was thus formed by antral mucous membrane. The left side was covered in by an inturned skin flap.
Stage 2.—At the next operation the remains of the left ala were adjusted and used for the lining of the left vestibule. The lining of the right vestibule was formed by a turned up labial flap which was attached to the lateral wall swing and to the septal swing. A forehead flap was brought down over the whole, after removal of the mucous membrane from the superficial surface of the lateral nasal wall swing (fig. 15).

Stage 3.—Subsequently, instead of the pedicle being returned or removed, the proximal end of the pedicle was cut loose from the forehead with a flap, which was designed to cover the opening into the antrum from the cheek. Scar tissue was turned in to line the hole, and the reversed pedicle was sutured over this (see fig. 16).
Stage 4.—Lastly, the portion of pedicle between the nose and cheek flaps was cut away (see fig. 17).

I have frequently employed this principle of the reversed forehead pedicle, which I believe is original, for various devices, such as the formation of nose and eyelid, or to give an additional prominence to the tip of a nose.

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Fig. 16.

Fig. 17.

LATERAL SEPTAL SWING.

In cases of loss of portion of one side of the nose, a skin flap may be turned in to cover the opening, either from the opposite side of the nose or from the same side of the cheek. Another method which I have devised is what I call the lateral septal swing. The case shown (fig. 18) is one of considerable loss of the left side of the nose. The septum was cut through to form a flap, consisting of cartilage covered
on both sides by mucous membrane. This flap was cut to fit the gap, and was turned up and sutured to the freshened edges of the opening. The only attachment left to the septum was at the anterior border. Despite this, the colour of the mucous membrane remained excellent, and its vitality was never in question (see fig. 19). The next stage is to remove the mucous membrane from the outer surface of the septal swing, and to bring down a forehead skin flap to cover it.

Fig. 18.

Fig. 19.

WOUNDS CAUSING LOSS OF BRIDGE OF NOSE AND NASAL AND SEPTAL SUPPORTS (PUG-NOSE TYPE).

In cases in which a wound of the nose causes loss of the bridge with destruction of the nasal and septal supports, a very typical deformity is produced. As healing takes place, the alae and tip of the nose sink in, and are drawn up by contraction of scar tissue, so that
the openings of the nostrils assume the vertical position. The photographs (figs. 20 and 21) show the early and late stages of such a wound. This I call the pug-nose type. If the case is seen soon after the injury, the deformity of the alae and tip can be to some extent prevented by sewing the lining skin of the vestibules to the skin of the alae, and leaving the nasal cavity open on the surface. This method allows free drainage of the cavity and may avert a chronic sinusitis.

The operation consists in cutting free the alae and tip and bringing them down into the normal position. This leaves a gap in the nose which has to be covered over by lining flaps, somewhat after the fashion of the Keegan lining flaps. These are made from the skin of the sides of the upper part of the nose, somewhat after the fashion of the Keegan lining flaps.
A very useful method for forming a new bridge and holding the tip in correct position is one devised by Major Gillies. This consists of a previously implanted rod of cartilage which is turned down on a hinge of skin and soft parts and attached to the tip. The sides of the opening are covered in by skin flaps as above. A forehead flap is then brought down. Fig. 22 shows the flaps employed, and fig. 23 the result of the operation.

**Fig. 22.**

**Fig. 23.**

**Wounds causing Loss of Bridge of Nose and Nasal Supports, without marked Deformity of the Tip (Bird-beak Type).**

There is a well-defined deformity of the nose due to gunshot wounds in which the nasal bones and upper part of the bridge are destroyed, but the tip and alae do not become greatly displaced owing to the fact that sufficient of the septal support remains to maintain their correct position. This I call the bird-beak type which, like the pug-nose type,
I have described in former publications. A forehead flap is necessary in these cases to fill up the gap, and if the nasal cavities are open on the surface a turned in skin flap has to be used to cover the gap. In the case where photographs are shown, the cavity had become closed over and the only problem was how to get a better contour. This has been attempted by a flap which covers over the nose from root to tip but leaves the alæ free. No cartilage has been implanted in the bridge in the present case and I think this shows that cartilaginous support is not by any means always necessary to form a bridge. Photographs of the cast show the flap employed (see fig. 24) and figs. 25 and 26 show the case before and after operation.
WOUNDS OF THE NOSE CAUSING DEPRESSED BRIDGE AND NASAL OBSTRUCTION.

After a gunshot wound or any other injury such as a crash in an aeroplane or severe blow on the nose, a depressed bridge often occurs, with flattening and fracture of the nasal bones, and nasal processes of the superior maxilla, and there is often nasal obstruction from a co-existing traumatic septal deflection. These injuries are especially interesting to the rhinologist in that the experience gained from the war has opened up a considerable field in the treatment of civil cases.

Fig. 27 shows the case of an officer, who as the result of a gunshot wound had a depressed bridge causing an unsightly deformity and severe nasal obstruction. He also had a thickened deflected septum.
A submucous resection was done first, but although the operation which was performed by Captain Whale was a perfect surgical result, he was still unable to breathe through the nose. I then implanted a piece of his costal cartilage in the nose to form a new bridge by an incision at the root of the nose. He was very pleased with the improvement in his appearance, but was still better pleased to find that he could breathe freely through his nose (for the first time since his injury) (fig. 28).

I have since then noticed the relief of nasal obstruction through raising a depressed bridge, in other cases. The nasal supports of the bridge having given way while the soft tissues remain, the soft tissues with the lateral and the alve cartilage sag down and obstruct the passage. When a cartilaginous bridge is inserted the bridge cannot sink down because it is rigid and is supported at each end, i.e., at the
root of nose and at the tip of nose. Also, by means of its bulk it stretches the skin of the nose and this pulls up the cartilages into position. If the figs. 29 and 30 showing the nasal openings before and after operation be examined, it will be seen how much longer they are after-than before. This is probably due to the opening out of the angle between the median and lateral crura of the alar cartilage, the opening out being due to the pressure of the cartilage on the angle.

![Fig 29.](image1)
![Fig 30.](image2)

**DISCUSSION.**

The President: The termination of the war will probably not allow of frequent opportunities for plastic surgery of the kind witnessed in this most interesting demonstration. Accidents to the nose in civil life will occasionally require similar treatment, and I have no doubt that many people, encouraged by such successes as these, will want their natural noses to be improved according to taste.
Mr. Somerville Hastings: These results are excellent. I have treated many such cases myself. Mr. Hett spoke about septal cartilage, and using it as a bridge and for similar purposes. I have found it useful to adopt the following method: To make up the nose with wax to its required shape, or rather I have got Captain Derwent Wood, the sculptor, to do it for me. The wax is then cast in metal by the dental department. This metal cast gives me the exact shape of the piece of cartilage I have to put under the skin to remedy the deformity. I have it changed from wax to metal because I can then boil and handle it. In many cases I have found this method answer very well.

Mr. Herbert Tilley: I have only had experience in rectifying the smaller types of nasal deformity, and for these I have used portions of septal or of rib cartilage removed from another patient. In one instance I used septal cartilage which had been removed from the first patient on the day before it was implanted in the second patient. The piece thus employed was kept in sterile normal saline solution during the intervening night; no local reaction occurred after implantation and a perfect profile was attained in the patient, whose nose had been badly flattened as a result of a "crash" of his aeroplane.

Mr. Whale: In these cases it is striking to see how the scar tissue softens and the colour and texture of the skin approximates to that of the surrounding skin. The most uniformly good results are obtained by those who always insist on lining the nose as Mr. Hett has done in the case of these patients. I made some noses in France earlier in the war, and those which are not lined do not do so well. At Sidcup it is a rule that everything shall be lined by skin. In reference to what Mr. Tilley says, I have several times taken a piece of cartilage from one man and put it into two others on the same day. Nowadays those who do this work frequently put cartilage under the patient's skin: it seems a more convenient way of carrying it about than preserving it in saline.

Mr. J. F. O'Malley: I have had some cases of this kind, but far fewer than Mr. Hett, whose results are excellent. In regard to the advancement of the turbinates and the swinging of the septum, has Major Hett, months afterwards, found that there is a little septic trouble which is associated with the difficulty of getting the nose ventilated where there are dead spaces? Cartilage has always had the reputation of being very deficient in vitality, but it seems to be able to live in practically every living tissue into which it is implanted. With regard to the transplantation of one man's cartilage into another man, is there any danger of infection being transmitted from one patient to another? Possibly syphilis, for instance, may be present in the donor.

The President: With respect to Mr. Tilley's remarks about the preservation outside the body of cartilage which it is intended to transplant, I am a
little surprised that no more direct reference has been made to the Carrel method, which has shown that it is rather an advantage than otherwise to keep cartilage, tendons and other tissues in saline or alcohol for a considerable time before they are grafted. It will doubtless be remembered that the idea is to get rid of the normal cells of the graft leaving only the matrix, as the matrix more quickly picks up new cells from the body into which it has been transplanted than if it first had to get rid of its own. I think that may have occurred in Mr. Tilley’s specimen.

Mr. Hett (in reply): With regard to Mr. Somerville Hastings’ question as to the method adopted for measuring and regulating the size and shape of lost parts of the face which have to be replaced—the following is the procedure adopted: A cast is taken of the face and a model of the missing portion in plasticine is made on the cast by the sculptor. This reconstructed face is then made as a model on which the flaps are measured and the supports determined. These flaps are first cut in wash-leather or elastic composition and from them tin-foil flaps are made. These tin-foil flaps can then be boiled and used at the operation. In the case of nasal supports, such as the support for a new bridge, the requisite contour having been obtained in plasticine (allowing for the thickness of tissue under which the cartilage is to be placed) a mould is made, and a metal cast obtained. This is then used as a model which is first inserted; it is followed by the piece of cartilage which has been cut the same size and shape. With regard to cartilage being kept in stock, this, as Mr. Whale says, has not been necessary; if not used at the time it is removed, it is kept imbedded in the owner’s abdominal wall or in someone else’s, ready for use. Another reason is that cartilage remains as such without any obvious change when imbedded in the body. According to Professor Keith, this is because it lives by itself bathed in the fluids of the body. It does not become infiltrated by other tissues nor undergo growth—at any rate for the time (a year or more) during which one has been able to observe it. A piece was boiled after being removed and was then replaced in the body by Major Gillies without any macroscopic change on subsequent examination. I have soaked a piece in tincture of iodine and replaced it in the body with the same result. Mr. O’Malley has asked whether trouble has arisen from dead spaces or from subsequent sepsis inside a new nose. One is very careful to line a new nose with hairless skin or if possible with mucous membrane; also, to effect a cure of any intranasal trouble, such as a sinusitis, before doing a rhinoplasty, and I think the absence of any subsequent intranasal trouble which one has observed is due to these facts. I agree with Mr. O’Malley as to the necessity of doing a Wassermann reaction before transferring cartilage from one patient to another. Experience has shown that it is possible to convey infection through two patients to a third, by neglect of this precaution. Cartilage is certainly ideal for nasal supports as it retains its original size and shape and is in every way superior to hydro-carbon injections. The latter tend to spread in unexpected directions, after causing deformities more
unsightly than those which they are designed to remedy. My experience of this method has been confined to the removal of paraffin from the nose and face, for the deformity or other complication which its introduction has caused. In conclusion I should like to take the opportunity to thank Mr. Edwards, the sculptor, at the Queen’s Hospital, Sidcup; Mr. Scott, the artist, and Corporal Warbridge, photographer to the hospital, for their skill and work in producing lasting records in the form of casts, wax reproductions and photographic records of facial injuries and their methods of repair.

Epidiascopic Demonstration of Methods of Treatment of Gun-shot Wounds of the Nose and Nasal Accessory Sinuses.

[Continued from the February meeting.]

By G. Seccombe Hett, F.R.C.S.

(Cases shown by Dr. Douglas Guthrie.)

Private B.—Insertion of autogenous cartilage graft for contour. Cast before operation and patient shown.

Private M.—Rhinoplasty; reversed pedicle flap for lower lid. Cast before operation and patient shown.

Private H.—Total rhinoplasty. Cast before operation and patient shown. Note apparatus to hold tip forward.

Private Br.—Antral suppuration in case of partial loss of nose. Operation performed through traumatic opening. The case is shown from the point of view of the direct examination of the right antral cavity during the healing process.

DISCUSSION.

Mr. R. A. Worthington: Are these tubes which are made drained in any way, or are they entirely closed at both ends? I have not seen any of these operations.

Dr. Douglas Guthrie (in reply): These “tube-flaps,” so-called, are constructed by suturing at the back, and the skin from each side is also slid in and sutured. Gauze is placed between the “tube” and the incisions at the back, and the wounds heal by first intention.

1 At a meeting of the Section, held March 7, 1919.
Methods of Repair of Wounds of the Nose and Nasal Accessory Sinuses.

(PART II.)

By G. Seccombe Hett, F.R.C.S.

I discussed some of the type methods of rhinoplasty in the first part of my paper, and in this, the second part, I will endeavour to sketch out certain further principles which I have not yet touched upon. One cannot hope to cover the whole subject, as the scope for improvement and extension of methods is almost unlimited. Every case presents special features which require some alteration or modification, and hardly two flaps are identical.

Case I.—The first case which I show illustrates a method of overcoming the deformity left after a wound of and operation on the frontal sinus region. In this case, a piece of high explosive shell carried away the anterior wall of the inner portion of both frontal sinuses. In addition the eye was destroyed and the tip, columnella and part of the alæ nasi were blown away. Both frontal sinuses suppurated and it was necessary to have oblitative operations performed on them. After complete healing had taken place, a forehead flap was slid down over the depression to restore the contour. In such cases a piece of costal cartilage may be inserted subcutaneously to form a new brow ridge, but this was not found necessary in the present case. A plastic operation has also been performed on the nose. Figs. 1, 2, 3, show the condition before and after treatment.

Case II.—The next case is one of total loss of nose. The problem of a total rhinoplasty is always a difficult one, owing to the loss of the bed of the nose and the nasal supports. The nasal bones and anterior portion of the septum are lacking and owing to the severity of the injury a portion of the superior maxilla is generally missing. The new nose therefore starts from a depression on the face and it is difficult to obtain sufficient prominence of the organ. Either the nose has to be made of preposterous size or else it is unduly sunk into the face. In the case illustrated, pieces of cartilage were implanted into the sides

1 At a meeting of the Section, held March 7, 1919.
Wounds of Nose and Nasal Accessory Sinuses

Fig. 4.

Fig. 5.

Fig. 6.

Fig. 7.
of the nose to reproduce the supports which were lacking, i.e., the nasal processes of the superior maxilla. Lining flaps of skin containing the cartilage were turned inwards with lateral prolongations, which, when twisted into position, formed the lining of the vestibule of the new nose. A very large forehead flap was then brought down and subsequently cartilage was implanted subcutaneously in order to reproduce the prominence which should be formed by the nasal bones and cartilaginous bridge (see figs. 4, 5, 6, 7, and 8).

Fig. 11.

Case III (figs. 9 and 10) illustrates a case of total loss of nose in one of our British soldiers while a prisoner of war in Germany: the operation was done by a German surgeon. The principles of epithelial lining and nasal supports have not been carried out, with the result that the forehead flap which had been brought down to form the new nose has shrivelled, shrunk, suppurated and lost its contour and attachments. The case is now under my care for reconstruction and I hope to be able to show the result at a later date.

Case IV illustrates the use of my reversed pedicle forehead flap. In this case it was used to form a new lower eyelid in addition to repairing a deficiency of the bridge of the nose. A gunshot wound had
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Fig. 13.

Fig. 14.

Fig. 15.

Fig. 16.
carried away the upper part of the bridge of the nose and damaged the
left eye, so that the eye had to be enucleated. The lower lid was
also destroyed. Figs. 11 and 12 show the case before operation.
Fig. 13 shows the forehead flap brought down. Fig. 14 shows the
reversed pedicle flap for the formation of the lower eyelid. In order
to obtain the eyelashes for the new lower lid a few hairs from the
eyebrow were included in the reversed pedicle flap. Figs. 15 and 16
show the case shortly after the removal of the intermediate portion
of the pedicle.

Case V illustrates the employment of a forehead flap containing
the anterior division of the superficial temporal artery. This flap has
an excellent blood supply, and is useful in many cases, especially scarring
of the forehead, and renders the use of the ordinary forehead flap
containing the angular and supraorbital arteries impossible. Fig. 17
shows the case shortly after the gunshot wound. Fig. 18 shows the
same case after healing had taken place. Fig. 19 shows the pedicle
flap in position. Like all pedicle flaps, it is tubed by suturing the
skin margins of the pedicle together. This device prevents sepsis or
sloughing of the pedicle and improves the blood supply of the flap.

Case VI illustrates the principle of the chest flap rhinoplasty. In
this case a severe gunshot wound of the face resulted in the damage
depicted in fig. 20. When the patient arrived at the Queen's Hospital,
Sidcup, a forehead flap had already been used for a rhinoplasty so
that a flap for the restoration of the lower eyelid, cheek and nose
had to be made from the chest. Fig. 21 shows the condition on
arrival at Sidcup. Fig. 22 shows the chest flap with its tubed
pedicle in position. A word of warning is necessary as regards chest
flaps. In this case a portion of the new nose sloughed and I have
found that it is difficult to get a chest flap to live more than 3½ in.
beyond the tube of the pedicle.

The principle of the tubed chest flap in plastic surgery of the
face was worked out by my colleague Major Gillies and my late
colleague Captain Aymard, and I believe that to Major Gillies we owe
the original conception of this new and valuable principle.

Case VII is that of a soldier shot through the base of the nose
by a bullet which entered through the right nasal process and found
its exit through the left cheek, leaving the antral cavity open on
the surface (see fig. 23). The nasal supports were partially destroyed
and there were external intranasal adhesions as well as chronic suppuration of the left antrum. The nasal obstruction and sepsis was dealt with, and subsequently a small plastic operation closed the communication between the antrum and the cheek. After this the condition was as is shown in fig. 24. The bridge was much depressed and, although the nasal passages were clear of adhesions, the patient had marked nasal obstruction. An autogenous costal graft was inserted into the bridge for the restoration of contour of the nose, and the result is shown in fig. 25. The result of the plastic closure of the antrum can be seen in fig. 26, which is a front view taken at the same time as fig. 25. The patient stated that after the insertion of the cartilage into the bridge of the nose, he could breathe freely through the latter for the first time since his wound. This subject has already been referred to in Part I (p. 131).

Case VIII is another case of sunken bridge due to a gunshot wound of the nose, resulting in destruction of the upper nasal supports. Figs. 27 and 28 show the profile before and after the insertion of a costal cartilage graft for the restoration of the contour of the nose.
METHODS OF REPAIR OF WOUNDS OF THE NOSE AND NASAL ACCESSORY SINUSES.

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By A. L. MacLeod, M.B.

Patient, a male, aged 57, was first seen on October 1, 1917. He had suffered from hoarseness for five months, accompanied by cough and mucous expectoration. The left vocal cord was inflamed, fixed, and its central portion occupied by a cauliflower-like growth. No history of tubercle or syphilis. The Wassermann reaction was negative. Rest to the voice, with potass. iodide, was prescribed for some weeks without improvement.

On May 13, 1918, thyro-fissure was performed and the left vocal cord removed. Patient unfortunately died six months later from broncho-pneumonia.

Microscopical Report.—Two sections were cut: (1) A small piece which shows a large mucus-secreting gland which appears quite normal. (2) A large piece which shows the squamous mucous membrane, and the submucous tissues full of round cells, and deep in a large mucous gland.

There is nothing to indicate the cause of the inflammation and no evidence in the section of any malignant neoplasm.

The two microscopic sections were shown.

DISCUSSION.

Dr. Wylie: How can this be an adenoma of the vocal cord, as there is no gland tissue in the vocal cord? The growth must be from the ventricle. Or it might be a malignant growth.

Dr. W. Hill: I suggest the title should be altered to adenoma arising in close proximity to the cord.
Dr. Irwin Moore: I am glad to hear Dr. Wylie's opinion because Dr. Macleod wished the specimen referred from the last meeting in order to ascertain the views of members of this Section. Dr. Wylie would maintain that there is no such thing as an adenoma of the vocal cord. It is entirely a question of what structure the vocal cord is considered to include. I referred the specimen to Professor Shattock and he kindly prepared some beautiful sections through the normal vocal cords and ventricles showing their minute structure. These normal sections show not only a large number of mucous glands in the superior and lateral walls of the ventricles, but also an isolated gland in close proximity to the elastic fibres of the vocal cords. The latter are well seen in one of the sections exhibited by Dr. Macleod—which shows a portion of the normal vocal cord with a small amount of ventricular tissue attached. With respect to the two microscopic sections exhibited by Dr. Macleod, the following report furnished by Professor Shattock corroborates that originally supplied by the exhibitor.

Further Report on Dr. Macleod's Specimen by Professor Shattock, F.R.S.

Section (1).—This consists of mucous glands lying in adipose tissue with a striated muscle fibre here and there. The glandular tissue corresponds in character with that of the normal larynx, in the immediate neighbourhood of the elastic tissue of the vocal cord—i.e., with the glands lying on the outer or ventricular side of the latter. The volume of the gland tissue and its compact disposition justify the classification of the lesion as an adenoma.

Section (2).—This consists of a portion of the cord, infiltrated with round cells; the preparation includes a minute fragment of glandular tissue of the same kind as that in the other section.

Remarks by Dr. Irwin Moore.

I have looked up the literature, and find there are but three cases recorded which can be definitely regarded as adenoma of the true vocal cord.

Mr. Tilley has reported a case of adenoma of the right ventricular band at a meeting of this Section in March, 1914—viz., that of a male, aged 52, who suffered from hoarseness for from ten to fifteen years. There was a smooth globular swelling covering five-sixths of the anterior surface of the true cord. It was apparently an adenoma originating in connexion with the ventricle, although in the

discussion which followed, Mr. Rose suggested that the swelling was of an inflammatory nature, and Sir Felix Semon considered it was tubercular.

**Angioma of the Left Arytenoid.**

**By G. W. Dawson, F.R.C.S.I.**

**Patient,** a female, aged 50, who felt something stick in her throat whilst at breakfast one day in October, 1918. After this she spat a little blood. **Patient** first consulted the exhibitor in December, 1918, when a round bluish tumour, the size of a small nut, was seen on the left arytenoid. The galvano-cautery was applied on February 7, 1919, with the result that the size of the tumour was considerably reduced.

**DISCUSSION.**

**Sir StClair Thomson:** Should this be called angioma or should it be called telangiectasis, as there is much difference between the two? If there is such a thing as an angiomatous tumour, it is generally an angio-fibromatous tumour. It is possible the patient has had this many years, and we know that unless symptoms are caused by it, it is rather dangerous to tackle. Angiomatous tumours, so-called, when they do occur, are generally more or less pedunculated and definite, and as this is indefinite, I suggest it be called telangiectasis.

**Mr. J. F. O'Malley:** This does not seem to me to be a growth, but rather a permanent hyperaemic condition—a group of vessels on the arytenoid.

**Dr. H. J. Banks-Davis:** There must have been a tumour present of some kind in the arytenoid region, but since it has been cauterized it has shrunk up, so that it is now scarcely visible. I thought there was telangiectasis at the apex of the cords in addition to the cyanosed appearance in the arytenoid, which is hardly now observable.

**Adherent Palate.**

**By G. W. Dawson, F.R.C.S.I.**

**Patient,** a male, aged 26, was previously shown at the meeting of this Section in May, 1917. When aged 12 he suffered from sore throat, followed by nasal obstruction, for the relief of which, at the age of 16,

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four operations were performed. In October, 1916, patient consulted
the exhibitor for increasing deafness. The nasopharynx was found to
be completely occluded by a dense mass of fibrous tissue, through which
an opening was made and a rubber tube inserted. After the tube had
been kept in situ for several weeks, it was replaced by an obturator
(made by Mr. Turner), which patient is now wearing. The Wassermann reaction is positive.

DISCUSSION.

Dr. Perry Goldsmith: I have had a similar result after an adenoid
operation a number of years ago. The case was evidently syphilitic. The
end was exactly like this. Everything I did to keep the palate forward failed.
The point to which I want to draw attention is this: Here is a man with a very
small opening in the nasopharynx, but he complains of no nasal obstruction,
and, if a man with such a small aperture can get along without discomfort in
breathing, it should make some of us hesitate to operate on the front part
of the nose as often as we do.

Mr. O'Malley: I showed a similar case before the Section, with the
history of the patient having had a very bad throat a few years ago. It
was not definitely proved that it was from scarlet fever, but it dated from
that illness. There was nothing to suggest it was syphilitic, and the Wassermann test proved negative on more than one occasion. I concluded it was
ulceration. The way in which these cases arise is interesting. There
is an intense inflammatory condition on the mucous membrane owing
to a very virulent infection, and deep to that there is a myositis, which
paralyses the movements of the faucial pillars; and as there is oedema,
the soft tissues are all brought into contact, and this with the denudation
of the epithelium causes the opposing surfaces to become adherent to
one another. Before the myositis has completely disappeared permanent
adhesions have formed, and subsequently contractions occur.

Dr. Kelson: Mr. Dawson has improved this patient's condition. But he
still gets a little food and drink through his nose; cannot some better arrange-
ment be made to prevent that, some valvular contrivance for instance? There
is an open rigid tube at the back of the mouth, connecting it with the post-
nasal space, consequently during mastication some food must get forced up
into the nose.

Dr. Dan McKenzie: I have recently had a case which has given me much
trouble on account of adhesions of the soft palate. The patient is a girl, aged
17, and she has never remembered being able to breathe through her nose. On
inspection nothing abnormal was to be seen, but posterior rhinoscopy showed
an absolute adhesion between the posterior part of the palate and the posterior
pharyngeal wall. Though she has had five operations, she is now no better than when we started. First, we broke it down with diathermy, and for a fortnight she could breathe through the nose, but it closed up again. Then we incised the adhesions with the diathermy knife and put in tubes. She wore these for a month in a most persevering way, but when we took them out it closed up again. Then I tried Mr. Mark Hovell's suggestion of grafting: it was difficult, but I managed to get a graft in after breaking down the adhesions once more, and anchored it by two threads through the nose and two through the mouth. After ten days the threads came away, and again the palate slowly closed up. We tried another time with a graft, but with no better result. I shall be glad to show the patient, in order to get suggestions. I have even gone so far as to suggest the removal of the soft palate, which would enable her to obtain nasal breathing, but at the cost of nasal speech. I cut the graft from the thigh.

The President: I showed a case here, two or three years ago, and after trying three different methods I broke down the adhesions fully with a blunt dissector and my fingers, and then sutured the upper surface of the palate to the lower, round the posterior edge. First I used catgut sutures, but they were absorbed too soon; next time I used silk and it did very well. Much depends on the thickness of the adhesions. In this case there was recurrence on the right side, where the adhesions were thicker. I have not seen the patient for a year, but I hope to bring her to the Section at an early meeting.

Mr. Dawson (in reply): I agree with Sir StClair Thomson that the first case is a fibro-angioma. When I saw the growth it was of the characteristic blue colour, the size of a small cherry. I applied the cautery very carefully because I did not know what might happen. The growth seemed fairly firm, and the application of the cautery made it shrink a good deal. With regard to the second case, there were two cases exactly alike, but the other could not come. The one shown had been operated upon four times by Dr. Lack. The man came to me because of increasing deafness. They both had a positive Wassermann reaction, and both began at the age of twelve to have sore throats, which eventually became closed up. I think there is great difficulty in forming flaps for these cases, because you cannot fashion a flap from material, the thickness of which is as great as the length or breadth of the tissue at our disposal. No doubt there are some cases of adherent palate which are quite thin, but those I have seen have always been very dense. In this case it was probably half an inch thick. Both patients expressed themselves as very much better: the treatment has stopped the increasing deafness, of which both complained, and the dripping from the nose has ceased. The amount of food which passes into the nose is very little.
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Mucocele of the Frontal Sinus.

By G. W. Dawson, F.R.C.S.I.

Patient, a female, aged 65, who complained of a swelling over the left eye for twelve months, which was incised at an eye hospital, on December 19, 1918. When seen by the exhibitor on January 3, 1919, there was a foul wound with much pus. Operation performed on January 10: The eyebrow was reflected upwards, disclosing a large opening, through which the finger could be introduced into the frontal sinus, which was large and deep. There was much erosion of the nasal process of the frontal bone. Owing to the foul condition of the wound none of the necrosed bone was removed. The cavity was packed with gauze saturated in iodoform emulsion, and rapidly closed up. There was no history of pain either before or after the operation.

Choanal Polypi in Children: (1) A Boy, aged 9; and (2) a Girl, aged 12.

By Douglas Guthrie, M.D.

Case I. illustrates the rare condition of nasal polypus in children under 10 years of age. Out of 116 cases, recorded in literature, of nasal polypus in patients under the age of 16, only fourteen were found in children under 10 years. A boy, aged 9, suffered from right-sided nasal obstruction for a year. No discharge, headache or epistaxis. A bluish-grey growth, smooth and elastic, filled the nasopharynx and blocked the right nasal cavity, projecting downwards below the palate. A skiagram was taken and showed a shadow in the right antral region. Both antra were clear on transillumination. The polypus was removed by snaring and an intranasal operation was performed on the right antrum, which contained no pus. There was no sign of recurrence two years later.

Case II is that of a choanal polypus which had "herniated" into the opposite choana and had there become nipped and strangulated. The patient is a girl, aged 12, whose left nasal passage had been blocked
for six months. The right side had also become obstructed about a month before admission to hospital. Posterior rhinoscopy showed a dark purple swelling in the right choana. Anterior rhinoscopy shows the left nasal cavity to be blocked by polypus. The skiagram taken shows blurring of both choane and of the left antrum. The left antrum is opaque to transillumination. On palpating the nasopharynx in order to pass the snare, the strangulated lobule became dislodged. A radical operation done on the left antrum showed that the growth arose from the posterior part of the antral floor. On inquiry eighteen months later, the child was reported to be well and free from any nasal trouble. Water-colour drawings from the cases are demonstrated on the epidiascope, also a table showing the age-incidence of nasal and nasochoanal polypi according to various writers.

DISCUSSION.

The President: The second specimen appeared to me to be an example of a solitary antral polypus.

Mr. Tilley: It is a pity to do anything in the first instance in the nature of a radical operation for the removal of these growths. I have removed many of them satisfactorily by seizing the main mass of the polypus in a pair of ordinary adenoid forceps passed into the nasopharynx and pulling the polypus away. Often the pedicle will be removed at the same time. Many do not recur, and hence I think every patient should be given the chance of removal by such a simple method. The operation can be done with a very light, or even without any, anaesthetic. On the other hand, to enter the canine fossa of a young patient means destroying some of the dental nerves, and so setting up conditions disadvantageous to the patient’s after-career.

Mr. Dawson: A few years ago I had under my care a child, aged 12, with a large choanal polypus. It filled up the post-nasal space and bulged the soft palate downwards. Under cocaine I succeeded in passing a small blunt steel hook under the middle turbinate so as to engage the pedicle of the polypus. By making traction forwards the pedicle was torn away at its attachment, and the growth with its long stalk fell into the mouth.

Dr. H. J. Banks-Davis: In 1908 I showed a specimen of a large pedunculated nasopharyngeal polypus which I had removed from a boy, aged 9. I also showed a specimen from a boy, aged 8; both these cases were at the West London Hospital, and the specimens were mounted for the Museum, and are there still.

Dr. William Hill: The whole point lies in that to which attention has been drawn by Mr. Tilley—do the polypi recur? Judging from my own
practice, I think they do not, but I have heard of them recurring. Even if they recur once in ten times, opening the antrum is hardly justifiable, especially in a child.

Mr. Waggett: There is a little practical point. The way to get rid of them, in the hope of getting the base away radically, is to push the snare backwards, not to pull it out of the nose. By pulling outwards you cut through the polypus, and the base is not eradicated.

Mr. W. Stuart-Low: I have had a number of these cases under my care, and have successfully treated them by inserting through the nasal passage a pair of long, strong, flat fenestrated forceps, grasping the swelling, twisting it, and slowly pulling it away. It is rare for a recrudescence to take place if the removal is done in this manner.

Mr. Rose: The question of age has been mentioned. Polypi are not common in children under 10 years of age. The point especially to be noted is, that if a child under 10 has a polypus, it is almost always of the antrochoanal type. I agree with Mr. Tilley that radical operation on the antrum of a child for such a condition is too severe.

Dr. Douglas Guthrie (in reply): The discussion seems to have hinged mainly on treatment. I should have been interested to know whether others had seen any cases like the second I described. What would those members do who are averse to opening the antrum in such cases, if they found pus on proof-puncture? Would they perform a radical operation, or an intranasal operation, or simply remove the polypus? In dealing with the accessory sinuses of children one naturally leans towards conservatism.

Further Notes of a Case of Diverticulo-pexy.¹

By William Hill, M.D.

I heard that Dr. Grant was going to show a case in which he had done the operation I had suggested. When I set out, eighteen months ago, to operate on the pouch of a man, I intended to do the two-stage operation which had been recommended by Goldmann and Halstead. I meant to stitch up the pouch into another position and wait a considerable time before removing it. Halstead did it in ten days. Neumann strangulated it at the time of operation by putting a ligature round the neck, and it came away as a slough. There was a

¹ Shown at the meeting of this Section on December 7, 1917: Proc. Roy. Soc. Med. (Sect. Laryng.), 1918, xi, p. 60.
sloughing wound in the neck and a big cavity into the mediastinum. But a month afterwards the X-ray appearance was so satisfactory that I thought I would do it, and I was content with diverticulo-pexy. I was surprised there was no recurrence when I showed the case three months after operation. The case is still doing well: the patient was passed A1 into the Army last July, and I have recently heard from him that he is as well as ever he was. If there is recurrence I shall report it. In order to avoid leakage and the danger of mediastinitis, it is well to do the operation in this first stage, and if sufficient, leave it at that. If not, you can do a subsequent operation without danger. The technique of freeing the diverticulum is not difficult. I put a bougie into the pouch, and another into the oesophagus. The wall is sometimes very thin, and care has to be exercised in dissecting out. Lower down, the finger is a great aid in breaking down adhesions in the mediastinum. After sewing up the pouch, I put stitches under the sternomastoid, and I relied on the adhesions keeping it in position. It has been kept in position, and the swallowing is correct. I put a ligature round the neck, not enough to strangle it, as Marshall, and, I think, Goldmann did, but to prevent the food ascending in the early stages. By the time adhesions had formed, the pouch was more or less flattened. At all events, the man is very satisfied.

Case of Pharyngeal Diverticulum treated by Dislocation and Fixation in the Upper Part of the Neck.

By J. Dundas Grant, M.D.

The patient had had considerable difficulty in swallowing for over eighteen months, being unable to eat anything stringy; the least tickling of the throat caused return of the food, and food thrown up some hours or even as long as a day and a half after it had been swallowed frequently returned unchanged; on most nights he brought up a quantity of watery fluid, often containing particles of food. On examination there was no definite swelling on either side of the neck and the hyoid fossa were absolutely clear of liquid. An X-ray examination showed a very large pouch extending below the level of the episternal notch (see fig. 1, p. 166).

Operation: An incision was made along the anterior border of the
sternomastoid, which was retracted inside its sheath; it was then slit up so as to expose the deep parts of the neck; the omo-hyoid muscle was then divided and the great vessels drawn outwards while the larynx and trachea were drawn inwards. The thyroid gland was thus exposed and close behind it was a swelling consisting of the pharyngeal sac. This dipped very deeply down behind the manubrium sterni and was adherent to the surrounding tissues; it was detached, however, by means of the finger and pulled up into the wound; it was about the size of a very large human tongue and was apparently thick-walled; it was then stitched into the upper part of the operation cavity without puncture of its walls; the wound was closed except at the lower part where a rolled india-rubber drain was introduced; this was kept in for two days, there being very little discharge; the wound was then allowed to heal. A small tube was kept in the stomach for twenty-four hours and after its removal the patient could swallow with perfect ease and has continued to do so ever since (see fig. 2, p. 166).

Some adrenalin had been injected along the proposed line of incision and at the end of the operation the patient's pulse was so extremely tense as to occasion anxiety as to its effect on the vessels of the brain. From 3 oz. to 4 oz. of blood were, therefore, drawn off from the vein at the elbow by venesection. I am greatly indebted to Dr. William Hill for his co-operation.

When seen on February 10 he was able to swallow quite well, but after swallowing a fullness in the upper part of the left side of the neck sometimes persisted; this disappeared again very rapidly on each occasion; he was instructed to keep pressure on it. At the present time the discomfort from it is extremely slight, swallowing being normal.

Dr. W. Hill: This diverticulum was a long way down, but there was no great difficulty in the operation. The patient swallowed that night, and has done so well ever since. There is a pharyngocele, and air is getting into it, and if it causes trouble Dr. Grant will do the two-stage operation and cut it out. The other operation has shown a large mortality, in the hands of Rigby 18 per cent. And it must be remembered all deaths are not recorded, whereas successes are. I helped Dr. Grant with the operation.
Case of Epithelioma of the Epiglottis treated by Diathermy.

By R. A. Worthington, F.R.C.S.

The patient, a male, aged 44, had an ulcerating epithelioma involving the right half of the epiglottis and extending well down on to the right ary-epiglottic fold. The epiglottis and the growth at its base on the right side were removed in November last by diathermy. Healing was rapid, and the dysphagia was completely relieved. It is of course early days for the discussion of the possibility of recurrence or otherwise. A section is shown.

DISCUSSION.

Mr. W. M. Mollison: I have recently had what appears to be a similar case, in which I excised the growth by the actual cautery. There was an epithelioma of the epiglottis, involving the back of the tongue, in the middle line. I had to remove the whole of the epiglottis and a triangular wedge out of the back of the tongue. I set out to do it by means of the knife and forceps, by suspension laryngoscopy, but found it was impossible and so used the cautery. This was nine months ago, and there is no local recurrence. The patient had at the time an enlarged gland in the neck, which he neglected, and when subsequently removed it proved to be carcinomatous.

Dr. Dan McKenzie: Within limits one's experience with diathermy may be wonderful. I recently had a case of what appeared to be inoperable epithelioma at the base of the tongue, extending on to the faucial pillar. There was a fungation from the base of the tongue as large as a tonsil. There were also secondary glands in the neck. I attacked this growth with diathermy, and the effect has been amazing. After three or four applications the patient's condition is one of absolute comfort. He is well, he eats and sleeps normally, and he has no pain or swelling. Nor is there much scarring left. Of course, I removed the glands by external operation. Before the days of diathermy, such a patient would have been dead by now, and his death would have been a painful and miserable one. I have only come across one case in my own practice in which there has not been recurrence, but diathermy makes a wonderful difference to these patients' lives.

Dr. W. Hill: The point raised by Mr. Mollison is very important—namely, whether, with the choice of going to a nursing home fitted with diathermy apparatus, you are wise in choosing Paquelin's cautery for use in this class of case. I have seen the cautery applied to a big growth in the pharynx, and the large amount of heat generated in the neighbourhood is a
serious disability. With the use of diathermy, on the other hand, the heat is so localized that there is no scorching effect on the immediate tissues, and in the hands of an expert operator there is no sparking.

Mr. Howarth: A point of technique needs explanation, and that is, in what way does Mr. Worthington use diathermy? Personally, I use the electrode as a knife, that is to say, the spark takes the place of the cutting edge, and one can make a trench round the growth and excise it. I have found this method very successful. Does Mr. Worthington do that, or does he plunge the electrode into the growth and destroy it that way?

Mr. Mollison (in explanation): I did not mean I preferred the cautery to diathermy. I have never used the cautery before for such a condition, but I did in this case because I was driven to use the means immediately available. I agree with what has been said about diathermy.

Mr. Worthington (in reply): In answer to Mr. Mollison, I think a great deal depends on the healing afterwards. After diathermy I have found extraordinary rapidity of healing. I have now used it in many cases which one must regard as absolutely inoperable in the ordinary way. After its use, even if the patient has a recurrence and dies, the comfort is much greater than it would have been otherwise. I have not shown this case because I regard the patient as cured: it is too soon for one to be able to say that. But I think this is a method of surgery which should be much more widely used for cases which may even be regarded as operable by other methods. It seals the blood-vessels and lymphatics as it goes on. In certain cases I would prefer it to the knife. In answer to Mr. Howarth, I use it secundum artem, that is to say, in each case differently. Where there is a fairly large projecting growth, if I can save time by doing so, I cut and coagulate round it and then pull it away, because then it is unnecessary to coagulate the whole mass. But when the growth is superficial and spreading, it is necessary to go over the whole of it.

Foreign Body removed from the Nose after Thirteen Years.

By R. A. Worthington, F.R.C.S.

The patient was a boy, aged 15, who had had a fetid discharge from the right nostril since the age of 2, according to his mother. The boy himself was convinced he was born with it! A foreign body—apparently a piece of stick with bark on it—was removed from the right nostril; it was lying in the posterior part of the middle fossa. There was a condition of atrophic rhinitis. The septum was pushed over to the left, and the anterior surface of the sphenoid widely exposed to view.
DISCUSSION.

Mr. MOLLISON: It is extraordinary how rapidly these people recover after removal of a foreign body. In the first year of the war a man was sent to me because he was an offence to his companions, owing to a fetid smell coming from his nose. I extracted from it a foreign body which had been there fifteen years, and two days later I heard that the smell had quite gone.

Dr. JOBSOX HORNE: In the days before rhinology received the attention it does now, little or no nasal inspection being carried out, it was taught that discharges and bad smells from the nose were to be regarded generally as syphilitic, and were treated accordingly. Some few years ago such a case came under my care. The cause of the discharge and factor was a rhinolith, the nucleus of the rhinolith was cotton-wool, left in the nose thirty years previously. The case under discussion, therefore, cannot be regarded as a record in reference to the length of time a foreign body had stayed in the nose. In reference to a foreign body not undergoing any change or decomposition after a long sojourn in the nose, I can call to mind a bean taken from a boy's nose: the bean had not only retained its normal appearance but had commenced to sprout.

Dr. H. J. BANKS-DAVIS: I have shown a case here in which a grain of Indian corn was retained in the nose and expelled after fourteen years. No disintegration had occurred. I agree with Mr. Worthington than this fact is worthy of note.

Mr. WORTHINGTON (in reply): The condition of the foreign body was astonishing. One would have expected it to have been disintegrated by now. The fact that it is not is a great tribute to the antiseptic power of the nasal secretions.

Bilateral Ankylosis of the Vocal Cords; Case for Diagnosis.

By ANDREW WYLIE, M.D.

The patient, an old soldier, aged 53, consulted me on Monday, February 24, complaining of hoarseness, cough, difficulty in swallowing, and inability to sleep, of not more than seven weeks' duration. There is an old history of specific disease and rheumatism, but he is otherwise healthy.

Examination shows complete fixation of both vocal cords, nearer the middle line than the cadaveric position, with a swelling or
oedema of both arytenoids, but owing to a considerable amount of
mucus in the throat it is difficult to get a good view. There are no
enlarged glands, no symptoms of aneurysm, and as far as can be
diagnosed no mediastinal growth. The patient considers the disease
was caused by gas at the front.

An X-ray photograph gives no information.
The exhibitor considers that tracheotomy is necessary, and after-
wards potassium iodide and mercury administered in increasing doses.

Postscript.—Since the notes were published in the Agenda Dr. Iron-
side Bruce has taken a screen of this patient, and reports that “there
is an increased opacity of the posterior mediastinum at the level of
the aortic arch. Food impacts at this level and suggests a definite
obstruction, the nature of which is not definitely indicated . . . prob-
ably a growth.”

DISCUSSION.

Dr. W. Hill: I gather that Dr. Wylie does not intend to stick to the title
he has put down: probably he means ankylosis of the crico-arytenoid joint.

Mr. Whale: I thought those cords were movable. Dr. Wylie says the
patient considers the disease is due to gas at the Front. I saw many cases of
gassed troops after the Somme fighting, in France, as many as sixty in a week,
and there was nothing in them to suggest that there was a deep lesion of
the crico-arytenoid joint: the ulceration was superficial. I made several
examinations of each case. The injury caused by the gas tended to be limited
to the posterior part of the larynx and the posterior pharyngeal wall. I
thought this distribution of the lesions must have relation to the usual dorsal
decubitus posture.

Dr. Perry Goldsmith: It is important to note that this man is losing
weight, and that his laryngo-pharynx is full of mucus. Taken together with
the remarks in the notes, this points to it being malignant disease of the
oesophagus. The use of the oesophagoscope would be very helpful in definitely
clearing up the diagnosis.

Mr. J. F. O'Malley: Did Dr. Wylie use the oesophagoscope, because there
is, I believe, an epitheliomatous growth near the cricoid, and I think the
spread of it is fixing the arytenoid. The mucus and the patient’s wasting are
confirmatory of that view.

Mr. Mollison: This patient has no tendo-Achillls jerk, and I think that
fact, plus the bilateral lesion, is suggestive that it is syphilitic. It is most un-
usual in cases of hypopharyngeal epithelioma to find fixation of both cords.
Dr. H. J. Banks-Davis: I thought the case was one of oesophageal carcinoma implicating both recurrent laryngeal nerves, hence the double abductor paralysis: this is not uncommon, and double abductor paralysis when no obvious signs are present should always suggest the possibility of an oesophageal growth before dysphagia is marked. This patient has the increased salivation and mucus in the pharynx, which is always suggestive of this condition as a cause of double paralysis.

Sir StClair Thomson: Dr. Wylie showed me a skiagram indicating stenosis in the cricoid region, and that would confirm the view which has been expressed: malignant disease of the retro-cricoid region, implicating the recurrent laryngeal nerve.

Dr. Wylie (in reply): The notes were written in a hurry: I only saw the case for the first time last week. A skiagram was taken, but it showed nothing definite. I sent him yesterday to Dr. Ironside Bruce, and on the screen he states that food became impacted at the level of the aorta, and drew a diagram of the position. Therefore, in my opinion, the case is one of malignant growth of the oesophagus. I cannot see in any literature a case in which malignant disease, even of the oesophagus, affected both vocal cords.

Sarcoma of the Nose; Modified External Operation
(Moure's Lateral Rhinotomy).

By Andrew Wylie, M.D.

Patient, a male, aged 43, has attended my clinic at the Central London Ear, Nose and Throat Hospital for several years, suffering from sarcoma of the ethmoidal region of the nose, which was removed several times with forceps and curette.

In November, 1918, after a prolonged absence, he came to the hospital with the left eye somewhat bulging, the left cheek inflamed, and the nose swollen and red on the left side. The swelling in the left nostril was like an abscess. I admitted him as an in-patient, and a deep incision was made from the inner canthus of the eye down to the alae of the nostril, and a horizontal incision along the infra-orbital ridge. This triangular piece of skin and cellular tissue were dissected from the bone, and a good view obtained of the growth. A large quantity of sarcomatous tissue and carious bone were removed, the piece of skin stitched back into place, and a large tube put in the nose.

The result is satisfactory so far.
DISCUSSION.

Dr. Perry Goldsmith: Dr. Wylie has obtained an excellent result. It is four months after the operation, but I cannot see anything in the nose to excite remark.

Dr. W. Hill: These cases of sarcoma do so very well under radium that I doubt whether we are justified in resorting to surgical operation for them without having first tried radium.

Mr. O’Malley: I have had cases which have given me considerable difficulty, but after witnessing this successful issue of Dr. Wylie’s case, I am doubtful whether one ought to waste time with radium, or incur the expense of it.

Dr. Dan McKenzie: Here you are dealing with growth in the ethmoidal region, where there is much bone, and you are not sure where the growth is, or how far it extends, and in that region there are some important structures. I do not know what the effect of radium would be on the optic nerve or retina, but perhaps I may hear something which will calm these apprehensions.

Dr. W. Hill: I can confirm Dr. McKenzie’s apprehensions. I had a case of malignant disease in which the patient came from Australia to have radium applied. It was a sarcoma high in the ethmoidal region. After three or four doses of radium the whole growth, apparently, had disappeared. But on the sixth day the patient became ill and died, from what we believed to have been thrombosis of the cavernous sinus, possibly there was thrombosis of vessels inside the brain. Death from thrombosis following operations in this region is not unknown. It never occurred to me to attribute the patient’s death directly to the radium treatment, but one cannot be too positive that it was not accelerated by it. He had ocular but no mastoid symptoms.

Sir StClair Thomson: I am very glad to see that Mouré’s operation is becoming popularized, because I was one of the first in this country to recommend it. I have recorded two cases, in which the patients are still alive, one nine and the other either seven or eight years since the operation. In one the growth was sarcoma, in the other epithelioma. There was malignant disease in the ethmoid region of the nose, completely filling the antrum in one of the cases. The ease with which the operation is carried out and the field obtained surprise everybody. It should be still more popularized, because either Mouré’s or Rouge’s operation, or a combination of the two, would practically abolish the fearful operation of removal of the upper jaw. No doubt Dr. Wylie found at the operation that one incision is enough from the inner canthus, going down just to the edge: this I did not discover until I had done a few cases. I used to make an incision under the eye, but the skin is so friable that it is not wise, and you can turn back the cheek as far as the malar eminence.
Mr. Mollison: At Guy's Hospital I had a case of sarcoma of the right upper jaw, which was referred to the dentist, as it was thought to be a dental cyst. I found it was a sarcoma, and I thought radium would be better than removal by the knife. I sent him to the Radium Institute, and they replied they would rather the growth were removed in the ordinary way, and radium applied subsequently if necessary.

Mr. Worthington: I have had a case similar to Mr. Mollison's, but with the opposite experience. It was diagnosed by the dentist and by another surgeon and myself as a dental cyst. As the man had some work to do, I told him there was no hurry and advised him returning in about two months for the operation. In three weeks he came back with a swollen cheek. I cut into the swelling and found it was sarcoma. I have a section of it. I sent the case to Dr. Hill and asked him to apply radium. That was done and the patient returned in a few days to Exeter. In a week the growth had vanished. That was five years ago, and to this day there is no sign of recurrence.

Chronic Unilateral Laryngitis for Diagnosis.

By Andrew Wylie, M.D., and Archer Ryland, F.R.C.S.

Patient, a soldier, aged 39, was admitted to the Seventy-third General Hospital in France, on November 24, 1918, for "boils," and was referred to the throat department, on December 2, for pain in the larynx.

December 15: The appearance of the larynx was very suggestive of early carcinoma of the right true cord, and he was recommended for evacuation to England for laryngo-fissure, if the diagnosis of carcinoma was confirmed.

December 18: The patient was admitted to the Central London Ear and Throat Hospital, with thickened and inflamed true cord. Its movement was impaired. It does not adduct to the middle line. There is thickness of the right arytenoid region of the vocal process. The voice is hoarse. There is no enlargement of the cervical glands. No definite diagnosis was made, but pot. iod. and mercury was administered.

March 3, 1919: There is still a marked unilateral laryngitis of the right cord, which is reddened, swollen and thickened along its whole length. The vessels are injected. There is no evidence of ulceration, nor of subglottic growth. There is no cough, spit, nor loss of weight.

Mr. Ryland states, on careful examination, that there is some
reddening and inflammation of the anterior one-third of the left cord, as if an extension of the inflammation had taken place by the anterior commissure. There is no pain. The voice is improved, and the movement of the right cord has recovered considerably since he saw him in France at the end of the year.

DISCUSSION.

Dr. H. J. Banks-Davis: This is either a very unusual case or a very simple one. Either he has laryngitis which has recovered on one side before the other, or he has early tuberculosis or carcinoma, which may begin with redness on one side only before any other signs are observable to suggest the condition.

The President: This is a very interesting case in view of the possibility of malignancy. It did not seem to me, however, that the lagging of the cord was sufficiently marked to warrant our taking it as a sign of cancer. I have seen cases of subacute laryngitis persisting after an acute attack in which one vocal cord remained congested for a long time after the other had cleared up. I have also seen some of these cases associated with discharge from the nasal accessory sinuses on the corresponding side. One of them did develop an epithelioma of the cord three or four years after the sinus discharge had been cured by operation.

Mr. Mollison: I think the case is one of tuberculous laryngitis.

Dr. Wylie (in reply): The case was sent from France as one of malignant disease. There is a history of specific disease. Iodide of potassium seemed to improve him for a time, but the condition is becoming worse, and the cord is not so movable as it was two weeks ago.

Case of Polypus of the Larynx removed with Snare.

By J. Dundas Grant, M.D.

The patient had been hoarse for nine months. On examination there was seen in the larynx a polypus growing from the anterior commissure, lying above the cords with its pedicle below them. The polypus, which is shown, was removed with a snare. The anterior commissure is the seat of election for the snare, and the snare is the ideal instrument for the removal of pedunculated growths in this situation.
Fig. 1.
Case of pharyngeal diverticulum (before operation). (See p. 156.)

Fig. 2.
Case of pharyngeal diverticulum (after operation). (See p. 157.)
Large Antral Polyp.

By James Donelan, M.B.

A large polypus with two pedicles removed from a man aged 24. Is this an instance of fusion of two polypi from inflammation or other cause? One pedicle appeared to come from the hiatus and the other posteriorly from an accessory ostium.

DISCUSSION.

The President: I pulled one pedicle out with forceps but finding the growth did not come away I slipped a snare over it far back bringing away the second pedicle. I think that one polypus grew from the ostium and the second through an accessory maxillary ostium and subsequently became fused. The antrum was operated on by the naso-antral method a few days afterwards and though it was found full of polypi the patient made a very good recovery and there has been no recurrence.

Dr. Watson-Williams: I think this specimen is more interesting than at first sight it might appear, and it might very well be referred to the Pathological Sub-committee. It is extraordinary that a polypus should have two pedicles, or that there should be fusion of two polypi. It seems to upset my conception as to the genesis of nasal polypi.

The President (in reply): We have heard of polypi becoming ulcerated, and there seems no reason why one polypus should not be adherent to another under those circumstances. I am quite willing to have it referred to the Morbid Growths Committee.

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1 At a meeting of the Section, held April 4, 1919.
Radiograms showing Absence of Right Frontal Sinus.

By A. J. Hutchison, M.B.

The patient complained of severe pain limited to the right frontal region. The roof of the right orbit was tender. Nothing wrong was detected in the right side of the nose, but pus and polypi were found in the left. A postero-anterior radiogram showed a fairly clear left frontal sinus and a dark shadow on the right side. Two further plates were taken in a slanting direction, the one to show the right and the other to show the left sinus. The plates were placed in front of the forehead, while the tube was placed near the right and the left ears respectively. These plates show a large left frontal sinus and no frontal sinus at all on the right side. After removal of polypi and of the anterior end of the left middle turbinal the pain on the right side of the forehead ceased and had not returned two months after the operation.

DISCUSSION.

The President: The radiograms shown have been taken at somewhat unusual angles, and there is a question whether that is a legitimate way of showing the frontal sinus.

Dr. P. Watson-Williams: If we are using skiagrams for determining the existence or condition of the frontal sinus, it is of the utmost importance to take an antero-posterior view, and not to be content with a lateral one. I had a case in which a lateral skiagram seemed to indicate a very large frontal sinus, but when one came to look at it antero-posteriorly the sinus was large on one side, but absent on the other. But when looked at laterally, the large frontal sinus, in its outline and shadow, so dominated the absence of the other, that if you had only the lateral view you would imagine there were two well-developed frontal sinuses. Recently I had a lady patient who had given me great trouble. Apparently, both frontal sinuses were absent. I had operated upon her maxillary antrum and sphenoidal sinuses, and, as far as I could see, the upper fronto-ethmoidal cells, because she had all the symptoms of frontal sinus infection. Others had seen her before. I contented myself with a per-nasal operation, and I got into the frontal sinus, which was small. Feeling I had done all I could do, I hoped she would get quite well. However, the symptoms persisted. When I came to open by an external operation, I found the frontal sinus flattened out and entirely infra-orbital. It must not be assumed, because the skiagram does not show you a frontal sinus, that it is non-existent. Skiagrams are very useful, as far as they go, but we should not attach too great importance to them.
Sir StClair Thomson: This, I understand to be a case in which there were discharge and symptoms on one side, on which there was no frontal sinus as shown by the radiograph. I will bring to the next meeting a skull I dissected many years ago and still possess, in which the frontal sinuses are both entirely on one side, one superimposed on the other. Each sinus, however, opens into its respective nostril. Without complete examination, one might have opened the sinus on the right side of the head, and found that it opened into the left nose.

Mr. Faulder: Did Dr. Hutchison try to pass sounds into the sinuses, and if so, with what result?

The President: Dr. Abererombie and Dr. Dan McKenzie have shown cases in which the symptoms were on the left side, whereas the affected frontal sinus was on the right. There was no other abnormality.

Mr. W. Stuart-Low: Does not this case illustrate the danger of what I have characterized as blind surgery, burrowing up through the anterior ethmoid cells, and attempting in this way to enter the frontal sinus? This method has been much vaunted, but I am certain that it should never be employed: the vagaries of the anatomy, as this case, where no frontal sinus existed, well illustrates, are so extreme that one can never be certain of taking a definitely safe route into the sinus—whereas the open, sound surgical procedure of Ogston's operation is always safe and successful.

Case of Pituitary Tumour; Trans-sphenoidal Operation; Great Improvement in Pressure-symptoms.

By H. Lawson Whale, F.R.C.S.

The operation being of recent date, the completed notes were not available in time for printing in these précis.

DISCUSSION.

The President: This patient is too ill to be here to-day, but members will be anxious to hear from Mr. Whale what was done.

Mr. Clayton Fox: How much of the septum adjacent to the anterior surface of the sphenoidal sinus was removed by Mr. Whale?

Mr. Lawson Whale (in reply): There was very little bleeding, and what there was was stopped easily by adrenalin. The after-treatment consisted in closing the outer incision below the eye, except for half an inch where I left a tube in the antrum. From the nostril I had a single wick of gauze, which was renewed daily after irrigation with 1 in 2,000 mercury perchloride. After I thought granulations had formed I used hydrogen peroxide. The tube was
taken out on the fifth day. I made a lateral cut as well as that on the side of the nose: this lateral cut differed from the stereotyped incision, in that it lay in the infra-orbital skin only as far as just outside the punctum lachrymale; from here it passed through the tarsal plate, and was continued in the conjunctival fornix. The result is that there is no scar on that half of the cheek, and there was no oedema. With regard to the septum, I went prepared to separate the septum from the cribriform plate, so as to dislocate it over to the other side, but this proved to be unnecessary. After I had cut away all the sphenoidal cell on that side I had decent access, without fracturing the septum. Having scraped out the endothelioma, I went along until I came to intact dura. The patient is prevented from attending to-day by a transient "pyrexia of unknown origin."

Case of Epithelioma of Tonsil two years and nine months after Diathermy.

By Frank Rose, F.R.C.S.

W. S., aged 67, was treated by diathermy for a growth in the left tonsillar region on June 6, 1916. At the present time there is an ulcer in the right tonsillar region and a hard swelling in the right side of the neck, but the left side appears to be free from growth.

DISCUSSION.

Mr. Douglas Harmer: This is a very interesting case for us to see now. Those who have operated on many of these cases realize that it does not matter how early is the disease, or how widely it is excised, there is often a rapid recurrence in the mouth and in the glands on both sides of the neck, and the result is a dismal failure. In the last few years we have treated many malignant ulcers by diathermy, and my impression is that they are not so liable to recur if they are burnt away as when removed with the knife. One thing is certain. In the old days we made our cut very wide of the disease, whereas, in the present day, the diseased tissue is removed much more locally. I think that members should now show their cases treated by diathermy, so that we may prove whether they have done well or not.

Mr. Norman Patterson: A point of special interest in that case is the period at which the recurrence took place. I had an old lady patient whose upper jaw I removed on account of malignant disease of the palate, which had extended into the antrum and ethmoid. I excised the growth, and a fortnight later I made a diathermy application to the whole surface of the wound. The usual incision was made, with the exception of that under the orbit, which is
unnecessary in excision of the upper jaw. Two years and nine months after the operation she had a recurrence of button-like form in the lip scar. That has now been freely removed. The recurrence in my case was really an implantation epithelioma: the original growth did not approach the lip. Some stray cancer cell must have lodged there at the time of the original operation, and it had taken two years and nine months to develop into the secondary nodule.

Mr. Frank Rose (in reply): I agree with Mr. Harmer, that these cases which have been treated by diathermy should be shown from time to time, so that members can judge for themselves what are the results of treatment.

Removal by the Indirect Method of a Jagged Piece of Bone impacted in the Óesophagus.

By W. Jobson Horne, M.D.

The patient, a woman, aged 32, whilst taking broth for dinner at 12 o'clock, swallowed what appeared to be a piece of bread in the broth; when it stuck in the throat she realized that it was a piece of chopped up beef bone. I saw her about 3 o'clock the same afternoon. The parts immediately behind the arytenoids and at the mouth of the Óesophagus were already so swollen that it was not possible to see the foreign body. With a probe it could be detected, and after an application of cocaine the uppermost point of the piece of bone could be seen in the mirror. With a pair of Morell Mackenzie's forceps I removed the piece of bone intact. The foreign body, in its greatest width, measured exactly one inch.

Later in the day the patient was none the worse for the accident, and on the following day was able to complete the unfinished broth without the bone. Tute, cito, et jucunde.

DISCUSSION.

The President: I am glad to see there is still a field open for indirect laryngoscopy, and that it has not become an altogether lost art. It is still possible to do good work with the mirror, without putting patients under chloroform.

Dr. Brown Kelly: I think that the term "hypopharynx" should have been used in the title of this communication.
Mr. Norman Patterson: In these cases it is preferable to use the suspension method. Of course the foreign body must be visible. There is a danger in cocainizing the larynx in the erect posture as the foreign body may be pushed down. Spraying the larynx with cocaine is risky, as it is liable to cause cocaine poisoning.

Dr. Irwin Moore: I suppose Dr. Jobson Horne means the hypopharynx, and not the œsophagus. I agree with the remarks of Dr. Brown Kelly. I congratulate Dr. Horne on the success of his manipulation. While we work on the newer methods now, we can still practise the older ones, and to the older one we can contribute this success.

Mr. Clayton Fox: I think it most probable that the foreign body was in the laryngopharynx, and not in the œsophagus or its upper sphincter.

Mr. Whale: This matter of the position of the foreign body I regard as important, because it is of no use to say the older methods will do what they are not capable of doing. If a foreign body is impacted in the œsophagus itself, it is not possible to see it by the indirect method, whereas in this case the body was seen, at any rate one end of it.

Dr. Jobson Horne: In reply to the inquiry whether the foreign body was in the hypopharynx or in the œsophagus, from my own observations I should say it was gripped by the œsophagus. However, the all-important point is that it was removed by the indirect method. The object in bringing the case forward was to assist in checking the growing tendency to carry out all work in connexion with the larynx or œsophagus by the direct method, with the result that the beneficent art of laryngoscopy through the too frequent use of endoscopy and laryngofissure is becoming lost, in the same way that the art of miniature painting was killed by the development of photography. One speaker advocated suspension laryngoscopy in such a case. [Mr. Norman Patterson: In these cases one can seize the foreign body from behind the arytaenoid, and I think suspension laryngoscopy is an excellent method.] The suggestion reminds me of the very amusing drawings which for some time appeared in a weekly illustrated paper, depicting a large staff going in a round-about way with elaborate devices to do a most simple thing, such as erecting a scaffold and a system of pulleys to light a pipe. If by five minutes' cocainizing the part a foreign body can be removed by the method I used, 
tute, cito, et jucunde, then why subject a patient to the discomfort and risk of endoscopy or suspension laryngoscopy?
Case of Laryngeal Whistling.

By L. H. Pegler, M.D.

The patient is a middle-aged man from Goring-on-Thames who attended the Fitzroy Square Hospital for severe tinnitus unaccompanied by deafness. It transpired on investigating the case that this man had the unusual faculty of whistling softly through the glottis.

Examination of the larynx, shows that the sound which ranges through an octave, and can represent any note quite audibly, is produced by the emission of air through a gap in the interarytenoid region, the imperfect apposition of the vocal processes creating an appearance much as one sees in a common type of functional aphonia. The difference is, that the speaking voice is good, both cords and vocal processes approximating normally on phonation, but an interarytenoid interval can be effected at will when the man is asked to whistle. Furthermore, in the former case the larynx is well exhibited, but whistling is assisted by depression of the epiglottis and some arching of the dorsum of the tongue.

These are all the observations I have been able to make in one brief inspection, but there seem to be some points of physiological interest here. The patient states that his four children all have the same faculty.

Additional Note.—Nineteen years ago, almost to the day (April 6, 1900), Sir Felix Semon exhibited a case of laryngeal whistling in a boy aged 13½ with a nervous cough. Particulars will be found in vol. vii of the Transactions of the Laryngological Society. The description given is precisely similar to that in my case, including the drawing down of the epiglottis, the obscuration of the cords in greater part anteriorly, and the chink formed by the arytenoids in order to produce whistling.

DISCUSSION.

Mr. Clayton Fox: The condition of this larynx is that the vocal cords are in the position met with in paralysis of the thyro-arytenoidicus, internus and transverse arytenoid muscles; there is an elliptical slit of the ligamentous glottis, and a triangular space of the cartilaginous glottis on phonation. I have never heard of such a condition in a laryngeal whistler, but remember a case in which the ventricular bands were opposed in a whistler.

Mr. F. D. D. Davis: This patient's voice is normal. He is able to whistle through the larynx and it is a trick which some people can perform. He closes
the whole of his larynx, with the exception of a small hole at the top of the larynx, as well as between the vocal cords. The opening occurs between the vocal processes. I have been watching him very carefully during whistling and think the whistling, in his case, is a trick. I knew a girl who could whistle beautifully through the larynx and could imitate almost any bird. His voice is good, he speaks well, and there is no paresis of the cords.

Dr. Jobson Horne: On phonation the posterior third of the glottis is not closed, a triangular space as in paresis of the interarytænoid muscle being left; this is met with so frequently in people who do not whistle through the larynx that it does not explain the whistling peculiar to this case. The same remark applies to the observation that the epiglottis comes down over the interarytænoid gap. On phonation I observed that the cords were not completely adducted in the middle third, so that two gaps were presented. The vocal phenomenon, I think, is best explained as an acquired habit or trick and not due to disease.

Dr. Kelson: The surprise is that we cannot all whistle through the larynx; it has a perfect mechanism for whistling. I think it is simply a trick we do not attempt because it is of no use. I am sure that if a thousand-pound prize were offered for laryngeal whistlers, cases such as this would spring up all over the place.

The President: The appearances seemed to me as described by Mr. Clayton Fox. I show a rough sketch I made to indicate the appearance of the glottis, both cartilaginous and ligamentous. The opening between the ligamentous cords may be due to the patient having acquired a trick of contracting the internal thyro-arytænoidens so as to leave the space one so often sees with the falsetto voice. The cartilaginous glottis also remains open constantly during the whistling and it is probably through there that the sound is made. No one has referred to the cause in the patient's four children except that it is a trick acquired by imitating the father. Perhaps this is the explanation of the so-called hereditary cases.

Dr. Pegler (in reply): I agree with a remark that has been made that the whistling is of the nature of a trick. While natural in the father, it has probably been acquired by his children through imitation.

Case of Naso-pharyngeal Angeio-Fibroma.

By G. W. Dawson, F.R.C.S.I.

Patient, a boy, aged 13½, was first seen by exhibitor in July, 1918, when he was found to be suffering from a growth originating by a broad attachment from the roof and left side of the nasopharynx. It had
caused the soft palate to bulge forward, and appeared at the left anterior naris, from which issued a foul discharge. The left cheek was also bulging. A preliminary laryngotomy was first performed, and the tumour removed by working through the mouth and nose, leaving rough bare bone in the roof of the nasopharynx, and posterior part of the septum. Patient made a rapid recovery. The pathologist’s report showed that the growth was an angeio-fibroma with large blood-vessels.

Patient was not seen again until a few weeks ago when recurrence was found to have taken place, the case presenting an almost identical picture with the addition that the left eye was now seen to be on a plane anterior to that of its fellow.

Suggestions as to treatment are invited.

DISCUSSION.

Mr. Harmer: I would operate on this case by the external route. Some years ago I reported to the Clinical Section a case of tumour of this kind in a man who came from New Zealand. The tumour was rather larger than the fist, and projected under the forehead; there was marked proptosis and the eyes looked in opposite directions. It pulsed violently, and was very vascular. He was sent to see Sir Henry Butlin, who said it should be removed, and that the patient should do well. He was admitted to St. Bartholomew’s Hospital under my care. The operation consisted, in the first place, in doing a preliminary laryngotomy. Then the external carotid arteries were exposed and compressed temporarily with Crile’s clamps. Each carotid took me twenty minutes to clamp, and the laryngotomy three minutes, so that up to that stage the time occupied was forty-three minutes. Then large incisions were made across the front of the tumour and laterally by the side of the nose, which was turned to the opposite side. The tumour was thus freely exposed on its anterior aspect, and rapidly evulsed. A patch, 3 in. by 2 in., of the base of the skull came away with it. The dura mater was intact. Haemorrhage was slight and the patient made an uninterrupted recovery. In forty-eight hours he was sitting up, and in about a week was well. He has written me regularly from New Zealand since, for seven years, and the last letter stated that there had been no sign of recurrence.

The President: The result in Mr. Harmer’s case has been excellent.

Mr. Norman Patterson: I agree that Mr. Harmer has had a brilliant result in the case he has related. I had a case of similar nature, in which a tumour was bulging into the temporal fossa. I did a very extensive operation on the boy which included the removal of the upper jaw. There was a great deal of haemorrhage. About an hour afterwards he died of shock. If I had clamped the external carotid that death might have been avoided. Recently
I have had two cases treated by radium. In one of them the tumour completely disappeared; in the other, all the swelling in the temporal fossa subsided, and the shrinking of the tumour, the origin of which was in the nasopharynx, was very rapid.

Dr. Irwin Moore: It is important that these cases should be diagnosed early, and that these growths should be very thoroughly removed. I had an interesting case which I operated upon and showed four years ago at a meeting of this Section, and so far as I know the growth has not recurred. In the pathological report of the case shown to-day there is a reference to the growth having large blood-vessels. These growths do not have blood-vessels, but very large cavernous spaces lined by a single layer of endothelial cells. There is consequently no artery wall to contract, and that is why these growths bleed so readily as soon as they are operated upon.

Mr. Dawson (in reply): I am much obliged by the suggestion as to Crile's clamp for the external carotids; it is perhaps particularly on the left side that clamping of the carotid will be required. I shall try radium first, leaving it in situ twenty-four hours. When I removed this tumour before it was very vascular; the bone was very bare, and it seemed to leave a depression in the roof of the pharynx. I thought I had gone into the sphenoidal sinus, but it must have been rather behind the sinus. I could get my finger into a depression; it was spiculated and rough, and alarmed me at the time. The patient made a good recovery.

Case of Perichondritis of Larynx.

By G. W. Dawson, F.R.C.S.I.

Patient, a male, aged 52, was admitted to hospital on March 8, 1919, suffering from urgent dyspnoea. He had had a previous attack early this year. Previous history: Laryngofissure had been performed by Mr. Tilley in August, 1918, for a small carcinoma of the posterior portion of the right vocal cord which was freely removed.

On examination there was marked oedema of the larynx. This has since improved, but considerable thickening remains. The Wassermann reaction is negative.


Section of Laryngology.

President—Dr. James Donelan.

Case of Sarcoma of Tonsil.

By G. W. Dawson, F.R.C.S.I.

This man, aged 38, came under my observation on December 14, 1917, suffering from dyspnœa and with a history of slight pain and swelling in the throat for five weeks. A large globular, smooth, pale tumour was seen to occupy the left tonsillar region. It was soft on palpation and stretched across to the opposite tonsil and extended below the epiglottis. A tracheotomy was performed the same evening owing to the increasing dyspnœa. This was followed by a profuse discharge of pus from the nose and tracheotomy wound for several days.

On December 23 I placed a loose ligature around the common carotid and removed the tumour per os. There was no capsule; the palate, base of tongue, and left side of pharynx were infiltrated by this very friable growth. The styloid process and neighbouring parts were laid bare. The bleeding was surprisingly small.

He made a good recovery, and on January 28, 1918, and again on February 18, radium was applied.

Pathologist's Report.—Round-celled sarcoma infiltrating muscle. Lymphatic glands (which were removed at the same time) show sarcomatous growth.

The case is shown to illustrate the exhibitor's opinion that too gloomy a view is much too frequently taken of these growths.

1 At a meeting of the Section, held May 2, 1919.
Pituitary Tumour; Sellar Decompression and Removal of Endotheliomatous Tissue; great Improvement.

By H. Lawson Whale, F.R.C.S.

The patient was prevented from appearing at the last sectional meeting by a transient pyrexia of unknown origin. The following is a synopsis of her case-notes, taken by Dr. C. O. Hawthorne (since September, 1917) and by the exhibitor:

Evidence of a pituitary tumour: Increasing lethargy, two and a half years; persistent headache, seven months; amenorrhoea, four years; Röntgenogram by Mr. Wigg, February, 1919 (exhibited), shows a very wide and deep pituitary fossa, with abnormalities of the anterior and posterior clinoid processes. Sugar tolerance (February, 1919): Five ounces of sugar were digested without any subsequent glycosuria. Optic disks, white, show optic atrophy. Vision: Perimetric charts (exhibited) show an increasing bi-temporal hemianopia. For the period preceding operation no chart could be taken, because the left eye had only perception of light. At this time also there was the divergent squint of a blind eye (left) and a slight exophthalmos.

Operation, March 8, 1919: Access by modified Moure's incision. Resection of naso-antral wall, ethmoidal gallery, and sphenoidal cell. Removal of endotheliomatous tissue (section by Dr. Wyatt Wingrave exhibited) from pituitary fossa.

Subsequent history: Entire freedom from headache. Recovery from lethargy. No change in optic disks; has not menstruated, and still has a high tolerance of sugar (3 oz.). Vision: Improvement in visual fields (charts exhibited), and acuity. Right $\frac{9}{6}$, and reads type J1; left $\frac{6}{10}$, and reads type J6; this eye had, seven weeks ago, only perception of light. The divergent squint persists.

DISCUSSION.

Mr. Tilley: The result in Mr. Lawson Whale's case is good, but the improvement appears to be temporary only. Whether there will be a recurrence of symptoms remains to be seen. He has adopted the method of approach which I think I was the first in this country to employ—viz., lateral rhinotomy, with removal of the nasal process of the superior maxillary bone. It gives easy access to the sphenoidal sinus. In my two cases I preserved the nasal bone, but Mr. Whale says he did not do so in this case and perhaps that accounts for some of the depression in the scar. In my
case there was no more disfigurement than in those operations of lateral rhinotomy done for malignant growths in the ethmoidal region. In my case of pituitary tumour, the operation amounted to removal of nasal process of superior maxilla, submucous resection of the posterior third of the septum, and entering the sphenoidal sinus by the interfental route. The sinus did not appear to be more than 1 in. to 1½ in. from the external wound. The patient, a schoolmaster, had partial bilateral blindness, and as a result of the operation he was able to return to his school duties. I have not heard from him for some months, so I do not know his present condition. I recommend lateral rhinotomy because of the advantage of bringing the field of operation so near to the surface.¹

Mr. Lawson Whale (in reply): With regard to the depression over the nose, I shall excise the sear and raise the inner canthus where it has dropped: I think I can entirely correct the slight facial deformity. There is a slight difference between the route I followed and Mr. Tilley's. The advantage of resection of the nasal bone is, that it gives a better access, and one does not have to resect any of the septum. In this case I was prepared to fracture the septum and push it to the opposite side, but I found that was not necessary, as I had enough room. I have minimized the statement as to the improvement in the sight. Her own statement is that before the operation she could not tell when the electric light was switched on: now she can read Jaeger 10. In France I had to remove from the pituitary fossa a piece of shrapnel as large as half a man's thumb, and I removed it so satisfactorily by the route I have mentioned that I tried the same route again in the present case.

A Lachrymal Sac removed Entire by the Intranasal Route.

By H. Lawson Whale, F.R.C.S.

The greater part of one canaliculus remains attached. The opening of the other is indicated by a bristle.

Unilateral Perithelioma of Maxillary Antrum, Upper Jaw, and Ethmoid, Removed at one Operation from a Woman aged 55, after Ligature of the External Carotid.

By H. Lawson Whale, F.R.C.S.

The patient made an uninterrupted recovery, but six to nine months later died of a secondary recurrence in the brain.

Adhesions and Contracture of the Fauclial Pillars following Complete Enucleation of the Tonsils.

By Irwin Moore, M.B.

Patient, a lady, aged 25, had suffered from septic tonsils for some years. They were carefully enucleated by the exhibitor in January, 1919, without any damage to the faucial pillars. The present condition of contracture and adhesions followed the operation and prevents her from singing. The voice easily gets tired, and she is unable to reach the upper notes. Her father, who is a physician, is anxious to know whether it is any use continuing her singing career. Opinions will be much appreciated.

This case exemplifies one of the disadvantages of complete enucleation of the tonsil with its capsule, causing interference with the action of the palatal and lingual muscles by contracture and adhesions, and is the chief objection to this procedure in the case of singers.

DISCUSSION.

Mr. Tilley: Can Dr. Irwin Moore give us a little more information about the case? We see the end result, but we do not know what were the difficulties, if any, in the operation. Was there any tearing, or accidental traumatism? Or was the anaesthetic faulty, thereby causing difficulty for the operator? I saw a case some three weeks ago in which the same condition existed: I do not think the ordinary cedar pencil could have been passed from the oral cavity into the nasopharynx, and portions of septic tonsils had been left behind.

Mr. Rose: Did any muscle come away at the time of the attempt to remove the tonsils?

The President: It seems to me that the left pillar is not complete. The patient complained that the sense of fatigue came on more on that side after singing than on the other. Training of this lady's voice can accomplish much, especially if there is a naturally good voice.

Mr. O'Malley: I think it is a good result of enucleation, with, perhaps, a small amount of contracture, and consider that the effects of the operation on her palate has not affected her voice at all.

Mr. Cyril Horsford: As a result it is quite good: one commonly sees contractions of that kind, and I have seen them in professional singers, whose voices have not been seriously injured thereby. Our first duty to the singer is to remove everything which is diseased and is likely to interfere with the general health, because anything which impairs the general health impairs the voice too. We should not blame the surgical operation. It takes two
things to make a good singer: a healthy organ and a healthy method. One cannot attach blame here unless one knows the method of training of the voice has been satisfactory. In this case it is very unlikely that it has been satisfactory. My custom, in the case of these people, is to impress upon them that the operation, which is necessary for their health, is done on condition that sound methods of voice training are indulged in afterwards. If one can recommend a sound method to her, I think her trouble will be cured. There is no limit to the voice possibilities when the diseased structure is removed. I think this patient should not regard the operation as being the cause of her vocal failure; that it is a vocal fault, not a physical fault.

The President: I have had the opportunity of seeing many singers during the last thirty years, from some of the first singers in the world to the worst; and my experience is, that the great singer has as nearly a perfectly healthy throat as a man or woman can have. They rarely require the services of a throat specialist, except when they are nervous, as about some slight "cold." It is the second-rate singers who clamour round throat specialists in the hope that they may be able to give them what God has not bestowed upon them.

Dr. Irwin Moore (in reply): There was no question of doubt in this case that it was necessary completely to enucleate the tonsils with their capsules, because they were deeply infected and their crypts filled with caseating material. I employed the method recommended by Mr. Waugh, i.e., dissection with forceps, the tonsil pedicle being clamped and twisted out. No difficulty was experienced during the operation. the patient being deeply anaesthetized with open ether by Dr. Rood, and in safe posture in case of bleeding—i.e., with the head well extended, so that any blood collected in the concavity of the hard palate, from which position it was easily sponged. The tonsils shelled out without any difficulty, no damage was done to the pillars, there was only slight bleeding, and one vessel only (in the upper angle of the left tonsillar fossa) was clamped and tied. The patient discontinued her singing lessons for three months after the operation, and it is a question whether it would not be advisable in such cases to re-start singing lessons sooner after enucleation of the tonsil, in order to exercise the palatal muscles, and so prevent adhesions or contractions.

Paralysis of Left Vocal Cord in a Woman, aged 49.

By James Donelan, M.B.

Patient, first seen at Italian Hospital on April 23, inst., complained that last November she began to feel a lump in her throat and then her voice gradually got weaker until it had almost disappeared at Christmas. She had some difficulty in swallowing but this seemed to be on the right side. About six weeks ago the tone of the voice began to recover but
with the return of voice a sense of suffocation was experienced and continues at the present time.

The left vocal cord lies in the cadaveric position and the voice is produced by the compensatory over-action of the right cord. There is a slight murmur to be heard over the aortic arch but it is not definite enough for any conclusion about it to be formed, and there has not been time for an X-ray to be taken. There is a curious inspiratory sound which is probably due to the air passing over the slack left cord. The patient was seen at a throat hospital a few weeks ago when the case was considered to be one of asthma dependent upon an intranasal condition. The septum is deflected to the right with much obstruction on that side, while there is considerable compensatory hypertrophy of the left middle turbinal.

DISCUSSION.

Dr. De Havillard Hall: The dyspnœa from which this patient is suffering gave me the impression that there is some direct pressure on the trachea, and this is probably the cause of the paralysis.

Dr. Dundas Grant: My opinion coincides with that expressed by Dr. de Havillard Hall, and it will be of the greatest interest to know what the skiagram shows. There must be, I think, a mediastinal growth of some sort pressing on the trachea. The stridor is double, it is not that either of laryngeal or of bronchial obstruction.

The President (in reply): There is a murmur, and I think the case may turn out to be one of aneurysm. The patient went to a throat hospital a few weeks ago, when her symptoms were considered to depend on the nasal condition. The description of the intranasal condition is given in order to complete the account of the case and not as implying that it has anything to do with the paralysis.

(Since the meeting this patient died and the post mortem showed a cancerous tumour of the gullet which, while but slightly narrowing its lumen, pressed on the aortic arch and left recurrent nerve).

Case of Diathermy for Epithelioma of Palate, Tonsil, Tongue, and Floor of Mouth; Patient well nearly Four Years after First Appearance of Disease.

By Norman Patterson, F.R.C.S.

The patient, aged 59, was first seen in June, 1915. Epithelioma of soft palate and right tonsil. Diathermy applied twice within a short period. In April, 1917, the patient presented himself with an extensive
growth involving the right side of the tongue and floor of mouth. There was also a large swelling in the submaxillary region. The growth was treated with diathermy and the electrode was plunged deeply into the floor of the mouth in many directions. Shortly after this the mass in the neck was removed by dissection. At one time the case looked quite hopeless, but now the patient is well and able to work, and there is no sign of the original disease.

Epithelioma of Palate and Anterior Fauacial Pillar; Enlarged Glands in the Neck.

By Norman Patterson, F.R.C.S.

Patient, a male, aged 59, presented a growth which involved the whole of the right half of the soft palate, base of uvula, and right anterior pillar. His chief trouble was pain on swallowing, which had been present for some months.

January 6, 1919: The whole growth was removed by diathermy.

February: Glands and fascia removed from right anterior triangle including all the fascial and glandular structures underneath the sternomastoid. Patient made a very good recovery, the only trouble being an attack of bronchitis which kept him in hospital for some time.

Mr. Tilley: Mr. Norman Patterson's results are extraordinarily good. I had not time to inspect the case, but the first—operated upon four years ago on account of extensive disease of palate, fauces and the base of the tongue, with glands in the neck—is a triumph of the method. This result can be produced by diathermy with less shock and loss of blood than by ordinary surgical methods and a better result could not be desired.

Tumour of the Base of the Tongue.

By G. W. Dawson, F.R.C.S.I.

Patient, a female, aged 56, complains of difficulty in swallowing for three years, and alteration of the voice for eighteen months. A large globular swelling is seen at the base of tongue. It is lobulated and hard anteriorly, whilst the base is constricted and not so hard. There is no ulceration. No lymphatic glands are detected, but there is slight induration of the submaxillary glands. The thyroid appears to be small or absent. Opinions as to treatment are invited.

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DISCUSSION.

Dr. Dundas Grant: The question is whether he has a "thyroid" tumour, or some form of malignant growth. It is rather irregular and rough on the surface for ordinary thyroid, and the position is one of the seats of election of endothelioma. The balance seems to favour it being a mild form of endothelioma. A portion should be removed for microscopical examination.

Mr. G. W. Dawson (in reply): I hope someone will suggest some form of treatment. I think it is an aberrant thyroid undergoing adenomatous degeneration, and that this accounts for the nodular part which is seen in the anterior part of the tumour. The posterior part is rather soft, and is consistent with the feel of a lingual tonsil. I will have a portion of it removed, and will show the case later.

Four Cases of Atrophic Rhinitis with Ozæna undergoing Treatment by the Glycophylic Method.

By C. H. Hayton, F.R.C.S.Ed.

Case I.—E. P., aged 15. Father a chemist. Four in family. History of tubercle bacilli on father's side. No venereal history. At the age of 11 her father noticed what he called large slugs in the child's nose, with a horrible odour. She complained of persistent headaches. There was no anosmia, and occasionally she could smell the crusts. The condition began in the right side, the left side being infected by douching the right side. She had been treated by the usual nasal douches—e.g., peroxide of hydrogen, protargol, silver nitrate, and tincture of iodine in various strengths. She was placed under glycophylic treatment in December, 1917. In two weeks the headaches had stopped, the odour had disappeared, and the crusts rapidly diminished. She began painting her nose five times a day, and now only requires to do so night and morning. At the last inspection the mucous membrane appeared quite normal, though slight atrophy was noticeable in the right nostril. If the treatment is neglected she can occasionally smell the scabs.

Case II.—Miss O., aged 24, domestic. History of tubercle bacilli in immediate family. Wassermann negative. The septum is deflected

1 A paper dealing with this treatment and its results was read by Mr. C. H. Hayton and Dr. F. Benians at the Summer Congress of the Section of Laryngology (vide p. 227).
to the right. Transillumination negative. Eighteen months ago patient first noticed scabs on blowing her nose. Her sister drew attention to a bad smell. Her sense of smell was slightly deficient. She suffered from frontal headaches. First placed under glycyphylic treatment twelve months ago. Headaches, foetor, and crusting have now disappeared. She paints her nose but once daily. The mucous membrane appears normal, though atrophy is still apparent in the left nostril.

*Case III.*—L. P., aged 43, housekeeper. Three sisters. No tubercle bacilli in family. Wassermann reaction negative. Transillumination negative. Twenty years ago patient's attention was first called to her nose by her sister, who complained of a bad smell. She soon noticed crust formations and complained of frontal headaches, which in time became persistent. There was complete anosmia. Crusts showed nearly a pure culture of Perez' bacilli. Many remedies—e.g., douching, lotions and ointment have been tried. She developed gastric trouble with declining health. Eight months ago patient was placed under the glycyphylic treatment, at first with pure glycerine (cultures showed no glycerine fermenters), next with pure glucose, and later with a 10 per cent. mixture of each. She began painting her nose six times a day, now she syringes once and paints once a day. She has now no foetor, and only a few crusts. The headaches have disappeared. The mucous membrane is somewhat dry, and atrophy is still apparent. Her general health has much improved.

*Case IV.*—Miss T., aged 17, domestic. Three brothers and two sisters. No history of tubercle bacilli in family. Wassermann reaction negative. Transillumination negative. Three years ago patient first noticed crusts, and shortly after was told by her mistress of the stench from her nose. Has persistently tried the more common remedies. No anosmia, no headaches. Patient was first placed under the glycyphylic treatment three months ago. She began by painting her nose five and six times a day, now requires to do so only twice a day. The foetor and crusts have disappeared. The mucous membrane appears normal, but much atrophy is present in both nostrils.

**DISCUSSION.**

Dr. Dundas Grant: It is an extraordinary group, and if I had a case to-morrow I should give this form of treatment a trial. There is an absolute absence of any unpleasant smell, and the only thing one notices is that the
mucous membrane has no brilliancy. That may not be due to the treatment, but to the disease, as the glands have atrophied.

Dr. Kelson: The question is, is the improvement permanent? I have not learned what was the interval of time elapsing since the last treatment. Obviously the patient cannot have her nose painted six times a day all her life, hence the entire merit of the method depends upon the permanency of the benefit. One constantly sees cases of atrophic rhinitis in which marked improvement takes place without apparent reason, and the patient seems to have got rid of the trouble, but it then reverts to the former condition. Packing the nose with gauze produces a similar improvement in these cases, but it is only temporary.

Captain E. F. Risdon, C.A.M.C.: I have had Italians under my care with this condition, and have usually treated them by the application of ichthyol, 40 gr. to the ounce of vaseline. Within a week patients can carry out the treatment themselves, and the nose becomes quite normal. I have carried this out for six or eight weeks, and I think they are all improved.

Mr. E. M. Woodman: I, too, have found that it is unadvisable to flush the nose with an alkaline lotion in this disease. I have been acting on the lines of Bier's negative pressure, with a view to flushing out the nose and establishing phagocytic action. I have had an evacuating apparatus invented for the nose. I first remove the crusts, then apply the suction method. Patients are always much more comfortable afterwards, and they can breathe through the nose freely. The treatment is carried out once every seven or eight days.

Mr. Benians: The position as regards permanent cure is this. If anatomical destruction of tissues has lead to a cessation of the normal self-cleansing functions of the nose, the nose cannot become normal again, and artificial cleansing of the nose will probably have to be carried on permanently. The point at issue is more as to whether we are able to replace an obnoxious by a less unpleasant bacterial growth, for it is likely that a considerable permanent bacterial growth will continue on these damaged tissues. We do not claim that our treatment will restore to patients the tissues they have lost.

Mr. Hayton (in reply): Our chief claim as regards this method is that it eliminates the most objectionable features of the disease—namely, the stench, the headaches, and the crusts. It consists in keeping the nose copiously supplied with pure glycerine, with the addition of 25 per cent. liquid glucose. The nasal cavities are swabbed with the solution, and the patients can help in the treatment themselves. At first they are required to apply it five or six times daily, but in diminishing frequency later. Our indications are the characters of the flora in the nose. We gradually lessen the frequency of the application as the condition improves, until one application a day is sufficient.
Specimens of *Aspergillus fumigatus* from Nasal Sinuses.

Exhibited by Douglas Harmer, M.Ch., and T. Jockes, M.B.

(A) Primary cultures from muco-pus from antrum: (a) in glucose broth; (b) on Sabouraud's glucose-agar (two days old, fixed by formalin vapour).

(B) Secondary growths: (a) on Sabouraud's agar from one of the primary colonies (two days old, fixed by formalin vapour); (b) sub-culture from one of the abscesses in rabbit's kidney.

(C) Slides with *Aspergillus fumigatus* in different stages of development.

(D) Different organs of a rabbit injected with *Aspergillus fumigatus* on April 15, 1919. An emulsion of a small quantity of spores of *Aspergillus fumigatus*, in 5 c.c. broth, was injected in the ear vein. On this or the next day the rabbit did not show any abnormality. On the third day the rabbit appeared to be languid and slow in its movements; it seemed to waste. These symptoms gradually increased and the hairs became dry and lost their glossy appearance. On the fifth day there was great weakness, especially of the hind legs, which became much worse on the sixth day, when it could only walk with great difficulty. On the morning of the seventh day it was found lying on the left side, was unable to stand, was very much wasted and its respirations were quick and superficial. As it was in a dying condition it was killed with chloroform on April 22, 1919.

At the post-mortem examination practically all the organs were found to be affected. The kidneys showed both numerous cortical and medullary abscesses. The lungs, liver, spleen, heart, many of the muscles and the mid-brain show numerous small tubercles. The right heart was much distended and on cutting the vena cavae much blood escaped, containing many whitish specks, which appear to be little lumps of mycelium. Subcultures from the heart's blood and from one of the abscesses in the kidney yield pure growth of *Aspergillus fumigatus*.

(E) Sections of kidney, lung, liver, spleen, suprarenal, psoas muscle, stained with haematoxylin and with carbolthionin, showing numerous tubercles all with definite zones. (a) The innermost, where the parenchyma has entirely disappeared and where a mycelium
can be seen surrounded by (b) the middle zone where the parenchyma is much degenerated and (c) an outer zone, with a well marked round cell infiltration, containing mainly polymorphonuclear cells. This outer zone is seen to contain a moderate number of plasma cells as seen in the sections stained with Pappenheim's stain.

Note.—The patient was a school mistress aged 30; she had had heavy colds in the head for eighteen months with profuse discharge of mucus, and occasionally some cheesy secretion. On washing out her antrum a mass of thick brown jelly was displaced similar to that discovered in the previous case reported by the author. Cultures showed aspergillus.

REFERENCES.

Combined Tongue Forceps and Depressor for use in Enucleation of Tonsils.

Exhibited by James Donelan, M.B. (President).

The points are inserted well back on the dorsum of the tongue and the fenestrated depressor keeps a large and troublesome tongue out of the way. The tongue is at the same time drawn forward so as to secure a clear air-way and field of operation.
Large Submucous Lipoma of Palate and Pharynx.¹

By HERBERT TILLEY, F.R.C.S.

Miss N., aged 15, consulted me on April 7, 1919, for a "swelling in the throat" which had increased "during the past month." The voice was "throaty" and she snored at night. There was also a varying degree of deafness in the left ear.

Examination: The left side of the palate was very swollen and presented an appearance similar to a large quinsy, except that the mucous membrane was normal in colour and no symptoms indicative of an abscess were present. The swelling was tense and semi-solid in consistence, its surface was not ulcerated, and there were no enlarged glands behind the angle of the jaw. The left tonsil was enlarged and appeared to be normal but was pushed downwards and inwards by the palatal swelling. The right tonsil was much hypertrophied.

April 12: At 2.45, \(\frac{1}{100}\) gr. atropine sulph. was given hypodermically and at 3.30 Dr. Rood administered "open ether," but in a few moments the difficulty of oral respiration necessitated laryngotomy; Abel's cannula was inserted, and the lower pharynx was plugged with a long strip of sterilized gauze. The operation carried out was identical with that employed for enucleation of the tonsil by dissection. The palatal portion of the tumour was easily separated from its attachments and so was its lower pole which extended to the lateral aspect of the pharynx opposite the pyriform fossa, but there was an area about the size of a two-shilling piece corresponding to the situation of the lower pole of the tonsil where the tumour was more intimately attached to the lateral

¹ At a meeting of the Section, held June 6, 1919.
Tilley: *Submucous Lipoma of Palate and Larynx*

wall of the pharynx. Bleeding was slight and only one ligature was applied to the tonsillar branch of the posterior palatine artery. The pillars of the fauces were united by three catgut sutures. The operation lasted some fifteen minutes, and as the haemorrhage had been so slight, and the general condition of the patient was so good, the right tonsil was also enucleated. The laryngotomy cannula was removed and the small skin incision sutured.

Convalescence was rapid and free from any disturbing symptoms. The patient left the nursing home ten days after operation. On May 7,

The fauces presented the appearances following an ordinary enucleation of tonsils.

The tumour has been examined by Professor Shatlock and by Dr. Teale, and they report that it is a lipoma and of rare occurrence in the submucous tissues of the throat.

[Mr. Shatlock's report was read at the meeting, and the tumour—and a microscopic section of it—was shown.]
Section of Laryngology

Histological Report by Professor S. G. Shattock, F.R.S.

The tumour consists of simple adipose tissue, the groups of fat cells being supported by scanty trabeculae of connective tissue furnished with lamellar corpuscles. In a few of the fat cells the nucleus presents a single vacuole, as it may in normal fat. As shown by the red coloration produced by the use of Sudan III, such intranuclear vacuoles, like that in the cytoplasm, are filled with fat. Certain of the trabeculae are, in varying degrees, infiltrated with plasma cells and lymphocytes, but polymorphs are everywhere absent.

In regard to the source of the fat, this, of course, is the submucous connective tissue. And it may be remarked that in every position in which submucous lipomata have been met with, fat cells may normally be found in the well-nourished individual; although theoretically this is not a necessary condition for the growth of a lipoma. So far as laryngeal lipomata go, an examination made of the mucosa of the normal larynx from the various positions in which such tumours have been encountered has demonstrated the presence of fat cells.

As regards the present case, in this connexion, microscopic preparations were made from the lateral wall of the pharynx of a well-developed female, the sections being cut longitudinally so as to include a strip of the whole stretch from the tonsil to the lower limit of the pyriform sinus. It may be recalled that beneath the mucous membrane the pharyngeal wall consists, not of unstriped muscle, but of a tough aponeurosis formed of intersecting bundles of fibrous tissue, on the exterior of which there lies the sheet of striated muscle furnished by the pharyngeal constrictors. In these longitudinal sections (ignoring the striated muscle) no fat cells were encountered in the higher part of the pharyngeal wall, but in that forming the outer boundary of the pyriform sinus, there were many rows and groups of such cells lying within the fibrous wall close beneath the mucosa.

Lastly, the tonsil of the side corresponding with the lipoma presents nothing of interest as bearing on the growth of the tumour. It was cleanly removed at the same operation, and though slightly enlarged is in no way connected with the lipoma. Its capillaries exhibit, in places, endothelial proliferation; and there is some intrusion of the investing epithelium into the tissue beneath; the epithelium is here and there,

moreover, thinned and abraded, the several changes being the results of an ordinary inflammatory process.

The pathogenesis of this, like that of any other circumscribed lipoma, although the simplest of all benign neoplasms, is as yet quite lacking a scientific explanation.

DISCUSSION.

Dr. Jobson Horne: I remember a very similar case being shown before either the Laryngological Society of London or the Otological Society of the United Kingdom. It was a larger tumour, and extended the entire depth of the pharynx, from the nasopharynx downwards. The history of the case and the operation done were almost identical with what we have been told about this case. Fortunately, they are rare, and, also unfortunately, when they do occur they are not difficult to remove.

Mr. Norman Patterson: I have seen a case of retropharyngeal lipoma, which was diagnosed as retropharyngeal abscess, but when operated upon it was found to be a huge lipomatous mass in the retropharynx.

Dr. Irwin Moore: Professor Shattock¹ read a paper on the subject of submucous lipomata before the Pathological Section in 1909. In that contribution he refers to all the specimens of laryngeal and pharyngeal lipomata known in literature. It appears that Mr. Tilley's case is almost unique. I believe that no similar case has been recorded in that part of the pharynx except the one by Sir William Milligan² to which Dr. Jobson Horne has just referred, and another by Dr. Bond in 1899.³ This case is included in Professor Shattock's list, along with the description of ten other lipomata which originated either from the aryepiglottic fold, from the arytenoids, or the back wall of the larynx, so they were practically pharyngeal or partly pharyngeal in origin. Amongst them is one remarkable case in which a lipoma originated from the epiglottis and base of the tongue, and an excellent drawing is shown of it. The patient went into a restaurant to have a meal, and in the course of it he suddenly put up his hands, made a slight noise, and died. It was suggested that the lipoma got grasped by a spasm of the pharyngeal constrictors—so causing occlusion of the larynx and death. In other words, that the patient involuntarily tried to swallow his tumour.

Mr. Tilley (in reply): When seen a fortnight ago this patient was well. I thought the tumour was so exceptional, that I had a sketch made of it. Mr. Shattock made a careful dissection of it, and he has made a very complete report on the subject (p. 191). I suggest—as we have a lot of business to get through—that this should be taken as read, but published in the Proceedings, where

members can study it at their leisure. If it is the wish of the Section that the drawing be also published in the *Proceedings*, I shall be pleased to bear the necessary expense.

(This was agreed to by vote.)

**Child in whom a Suppurating Dermoid Cyst has been removed from the Lower Central Region of Forehead.**

**By Herbert Tilley, F.R.C.S.**

The patient was admitted into hospital under my colleague, Mr. Percy Flemming, for acute inflammation of the right upper eyelid and adjoining lower frontal region. He gave exit to pus by an incision over the region of the frontal sinus and freely opened the latter cavity. Symptoms subsided except that, at intervals, a slight discharge of pus would make its way out of the scar of the wound. When I examined the patient under general anaesthesia, a probe passed through the suppurating fistula towards the middle line and seemed to end in a rounded bony cavity about 1 in. above the “nasion.” The exposure of this region revealed a thin-walled, membranous sac about the size of a pea and filled with thick pus and granulation tissue. Its posterior aspect was in close contact with the dura, and there was a free escape of cerebro-spinal fluid when this portion of the cyst wall was being removed. The cavity was smeared with B.I.P., a small drain inserted, and dressings applied.

The patient made an uninterrupted recovery.

**DISCUSSION.**

Mr. W. Stuart-Low: The case is an interesting one. The result seems better than it proves on investigation. I concluded that it was a cure, but the mother tells me that when the child cries the material exudes as before, from two holes, one at the top, and the other at the bottom, also when she presses it. Such is the trying feature about these cases—their tendency to recur. I have often found that the discharge does not come from any depth, and, if one has a fine wooden probe, puts a little wool on it moistened with trichloracetic acid, and moves it up and down a little in the fistula, it will clear up the discharge. I do not like B.I.P. for these cases. The method that I have found so successful in dealing with these intractable dermoid cysts consists in dissecting away the cyst wall as thoroughly as possible, and then rubbing into the exposed parts a solution containing chloride of zinc—20 gr. to the ounce. This starts a rapid healing action, and union is usually obtained by first intention after neat, careful stitching with horsehair sutures.
Mr. Tilley (in reply): I have not seen the patient for six weeks until today. The operation was a very interesting one, because the cyst went deeply down to the dura, and during the operation we were flooded with cerebro-spinal fluid. I used a B.I.P. smear because I wanted to leave something in the wound which would help to preserve asepsis after the operation. I agree that the zinc preparation might have done as well. I admit there is still some discharge, but I think some of it is coming from the outer end of the frontal sinus incision, which was made before the child came under my care. If anything of further interest supervenes I will bring the child again.

Carcinoma of Right Antrum and Orbit; Treatment by Excision, Radium, and X-rays. (Photographs shown.)

By Norman Patterson, F.R.C.S.

Patient, a male, aged 50.
April 22, 1918: Radium inserted for twelve hours. Tumour continued to increase in size.
June 17, 1918: Right external carotid ligatured. Growth removed from antrum and partially from orbit. Radium inserted for forty-eight hours.

Since the operation, X-rays have been applied once or twice a week for half an hour at each sitting. There was a good deal of swelling in the orbit for some time after the operation, but it has now quite gone.
At the end of April X-rays were applied for an hour by "a new hand"; this has caused rather a severe burn. Ulceration followed, and the ulcer is only beginning to heal.

Cyst of Larynx.

By Norman Patterson, F.R.C.S.

Man, aged 47. Patient complains of irritation in the throat of four to five months' duration. A cyst is seen lying above the level of the rima glottidis; it appears to be attached to the epiglottis and left aryepiglottidean fold.
Cyst of Larynx.

By W. Jobson Horne, M.D.

The specimen I exhibit of a larynx with a cyst of the epiglottis intact, illustrated an article on "Cystoma of the Larynx" I contributed to the Journal of Laryngology in 1907.1 The specimen is almost unique. It was met with accidentally, the patient having died from another cause in 1901. At that time there was only one other in the pathological museums in London. Opportunities for getting such now are still more remote. In Mr. Norman Patterson's case, the cyst is in an unusual position: the cyst appears to be intrinsic and not extrinsic in origin.

DISCUSSION.

Mr. W. Stuart-Low: I have had to deal with a number of these cysts, and I am referring now to what I am certain this is—viz., a simple serous cyst—not a dermoid, from which it should be definitely discriminated. Simply applying the galvano-cautery puncture deeply into the cyst and moving the point about somewhat in the interior causes a striking disappearance of the swelling, with no recurrence following. In this region this is a most advantageous method of treatment, as it evacuates the fluid contents of the cyst aseptically and causes the patient no more inconvenience than the application of the galvano-cautery puncture to the lingual tonsil.

Mr. W. M. Mollison: In one case a cyst of the arytaeno-epiglottidean fold extended from the upper part of the epiglottis to the arytaenoid. But this case is very much more internal, and does not seem to come from the fold at all. I dissected mine out with the aid of suspension laryngoscopy. The pathologist said it seemed to come from thyroid remains, and that several of these growths or cysts in the region of the tongue or pharynx had a similar origin.

Mr. Tilley: I would like to remind members of a case I brought forward four years ago, in which a cyst of this kind obstructed the patient's breathing, and when he seemed to be in danger of suffocating one night, he pushed his finger down his throat, and this act burst the cyst. On other occasions I punched out pieces of the cyst wall, but it always refilled, and produced much stridor. There was a curious swelling between the top of the thyroid and the chin. Eventually I asked Mr. Trotter to see the patient, and he thought it was a bursa, and proceeded to dissect it out. Eventually he got into the

centre line and into the larynx. When I saw this case to-day, I wondered whether it was of the same type, and since Mr. Mollison mentioned one that originated in the thyro-lingual duct, I think we may find something of that kind here. I suggest that Mr. Patterson looks for it.

Mr. Cyril Horsford: I have had a case of the kind, with a similar history: occasional rupture, followed by temporary relief, but in my case the cyst refilled on several occasions, and I got Mr. Trotter to dissect it out. He did it by the external route, through the neck. He completely dissected out the whole cyst without perforating the interior of the larynx. Although the man had an attack of bronchitis afterwards, he did very well. It was a beautiful operation, especially as the wall was very thin and would easily have been ruptured.

The President: I showed a case of thyro-lingual cyst years ago, which went not only along the upper border of the cricoid, but there was a large finger-shaped extension to the left superior cornua of the hyoid bone. It was a difficult operation, but we were fortunate enough to get the cyst out without rupture. If one uses a blunt dissector and proceeds carefully it should be generally possible to remove them without tearing.

Mr. Norman Patterson (in reply): This cyst I think is attached to the posterior aspect of the epiglottis and the aryepiglottic fold. Since hearing Mr. Tilley's and Mr. Mollison's remarks, I think I shall try to dissect it out, making use of suspension laryngoscopy, and thus get it away intact.

Dr. Jobson Horne (in reply): The two cysts under discussion may have a different pathogenesis. The one I show is of the less rare and more simple variety, which develops from a gland in the tip of the epiglottis. The other may be of the congenital kind, a very rare form of cyst, which develops in the ventricle of the larynx. The congenital cysts cannot be dealt with in the same way as the simple variety. In the case of the latter, one should use not a cold snare, but one through which the electric current can be passed when the snare has been brought home. Thereby the cyst is completely removed, leaving only a lineal scar: the result is good and neat. If the cyst wall is left there is the prospect of it refilling.

Case of Delayed Breaking of the Voice.

By W. Jobson Horne, M.D.

The patient, a lad, aged 18½, seeks relief from what he calls a weak voice, and what others might term a falsetto voice. At 15 years of age it was thought that the voice was breaking, but the voice did not become deep as in boys when growing up. The larynx presented the cords on phonation in the falsetto position. The treatment has consisted of iron and nux vomica.
The case is brought forward with a view of eliciting the experience of others in the frequency, the treatment and the prognosis of the condition. The text-books on laryngology are remarkably reticent on the matter. It may be that in the past the condition was regarded as one that would right itself in time, and was not deemed of such importance as to be brought under the notice of a laryngologist. In the present day, however, when a lad is a man before he is a boy, at least from the wage-earning standpoint, a youth resents any physiological delay which may handicap him in the labour market.

DISCUSSION.

Mr. G. W. Dawson: I have had a similar case in a youth aged 18½.

Mr. Tilley: When I was on the staff of Golden Square Hospital, a man who was a shop-walker consulted me there because he was threatened with loss of employment as his treble voice caused so much amusement among the female members of the establishment. The larynx seemed normal. I sent him to a teacher of voice production (whose name I now forget) and he cured him effectively in a very short time. I suggest that the same be done in this case.

Mr. Hope: Is this boy fully developed in other parts?

The President: I suggested to Dr. Horne that he should make that inquiry.

Mr. E. D. D. Davis: A student at Charing Cross Hospital had a similar voice at the age of 22. I saw him occasionally. Ultimately, he developed tuberculosis and went to a sanatorium. There he was chaffed a good deal, and one morning to excite sympathy he pretended to have a sore throat and to be hoarse, and from that time he suddenly developed a bass voice. I regard the present case as a functional one, and that if he has lessons in voice production he will obtain a normal voice in a short time. I have seen the larynx in some cases of breaking of the voice, and the appearance is very similar to that of cases of functional aphonia, with slight pinkness of the cords and the adductor chink, which this boy has. The process of breaking of the voice is so gradual and imperceptible in most cases that we are rarely consulted.

Mr. Cyril Horsford: These cases are very common, and the success in treatment depends not so much on what has been termed "exercise," as on the discovery of the one chest production note which would be the basis for the development of the rest. By teaching him the "knack" and by imitation, getting him to produce one chest note, and when he is conscious of it, building on it, there will not be much difficulty.

Mr. Lawson Whale: The time occupied in the breaking of any boy's voice is purely relative: it may be accomplished in a week, or it may take
months, if not years. I think Dr. Jobson Horne has drawn attention to the case because, as he says, these are days when boys want to "grow up" too quickly: otherwise such a case would not have come before a laryngologist at all. In this patient I saw a chink such as is evident in a hysterical woman, and I have seen it two or three times in people whose voice is "breaking." The cords in this larynx are not at all in what I should call the falsetto position: it is, rather, the position of the functional aphony of the text-books.

The President: In many of these cases the disadvantage may be overcome, by training, as Mr. Horsford and others have suggested, especially if one hits on the proper note. I ask Dr. Horne a question which may already have occurred to him, that is, not only as to this boy's general development, but whether, in this case, the functional aphony may be a sign of tuberculosis. There is a chink, it is not the falsetto position, but seems more like that of functional aphony.

Dr. Jobson Horne (in reply): I am glad the condition has given rise to discussion, because I think we ought to be able to answer usefully the questions raised by the patient and the friends. The boy has been seen by me only once so I am not at present able to give all the clinical details. One question was whether the condition might be functional; and another was whether the boy was in other respects fully developed. Subsequent examination showed that the answers to the questions were in the negative.

Case of Tongue for Diagnosis.

By W. Douglas Harmer, M.Ch.

History: For some months patient has had an increased amount of saliva and phlegm in the mouth. Recently for the last three months he has had increasing difficulty in articulation, so that at present his speech is very indistinct. He has had no operation on his tongue, and no treatment. There is no pain. There is no history of syphilis.

Local condition: The pillars of the fauces are drawn forward by the shrunken tongue, the left more than the right (being at the level of the first molar tooth). The tip of the tongue is absent; the tongue is represented by part of the posterior portion and remains on the floor of the mouth which runs straight into the ramus of the jaw in front. The surface of the tongue is covered with small protuberances especially in the mid-line. The tongue cannot be protruded. There is no glandular enlargement.

The Wassermann reaction is negative.
Section shows atrophy of the muscular elements in the tongue, with increased proliferation of the epithelium. The superficial layers of the epithelium are oedematous. The deeper cells show typical epithelioma.

DISCUSSION.

Dr. Irwin Moore: Mr. Harmer has unexpectedly been called out of town, and is prevented from being present. He would like to have the opinion of members on this case, since the diagnosis is obscure.

Mr. Stuart-Low: I have no doubt this is epithelioma. There are glands in both submaxillary spaces, and with one finger in the mouth and another outside, one can feel a stony hardness in and under the tongue. Moreover, there is a profuse flow of saliva. Secondly, he cannot move his tongue freely—it is moored to the deep structures. I do not know of any other disease which would affect the tongue in such a way. It is inoperable, but I would advise frequent multi-diathermy puncture and the local application of elixoid mucin as a soothing and emollient agent.

The President: I have a case with much the same appearances, which I had hoped to be able to show to-day: there is no doubt about it being cancer. In my case there is a much more marked involvement of the glands than I can make out in the case now shown.

Mr. G. W. Dawson: The tongue appears to have shrunken and got smaller. The possibility of lupus must be considered.

Epidiascopic Demonstration of the Normal Histology of the Vocal Cord and Ventricle of the Larynx, considered in connexion with the Development of Adenomata.

By Irwin Moore, M.B.

(WITH REMARKS BY PROFESSOR S. G. SHATTOCK, F.R.S.).

A recent case of removal of a growth from the vocal cord by thyrofissure and reported at a meeting of this Section by Dr. Macleod1 as an adenoma, has drawn attention to the great rarity of these growths in the larynx, and the question has again arisen as to the possibility of such a growth originating from the vocal cord itself.

Whilst many authorities—e.g., Lennox Browne, Kyle, Schwartz, Luschka, Gottstein, and others have even doubted the possibility of

their occurrence in the larynx, Morell Mackenzie\textsuperscript{1} has expressed the opinion that they do occur, but very seldom. It is said that they may spring from the laryngeal surface of the epiglottis, which is their favourite site, or the mucous membrane of the arytenoid cartilage, or from other parts outside the larynx. Their rarity may be gathered from the statistics of benign growths of the larynx compiled by various writers, e.g., amongst the 500 cases collected by Massei\textsuperscript{2} only two cases of adenomata were recorded and these were said to be doubtful. Amongst Fauvel's\textsuperscript{3} 300 cases there were none; whilst Morell Mackenzie\textsuperscript{4} recorded only two cases, one of which was said to be an adenocarcinoma. A search of the literature reveals only eight cases recorded of adenomata of the larynx, and in a number of them the diagnosis was said to be questionable; only two cases apparently originated from the vocal cord itself. In the first case reported by Paul Bruns\textsuperscript{5} in 1868, the growth was on the left vocal cord, and in the second case recorded by Morell Mackenzie it was situated below the anterior commissure of the vocal cords, and looked like an ordinary cauliflower excrescence. Dr. Macleod's case is apparently the only other that has been recorded in the world's literature.

We know that the most characteristic feature of the ventricular band is the presence of numerous acino-tubular glands whose ducts open upon its median as well as on its ventricular or saccular aspect, but the possibility of an adenoma originating from the vocal cord itself depends entirely on what structure the vocal cord is considered to include.

While some authorities hold that the vocal cord consists of only the apical portion of the triangular mass of elastic tissue, which constitutes the projecting portion of the cord, covered by thin adherent mucous membrane and possessing no submucous layer or glands; others are of the opinion that glands do occur in and open on the surface of the vocal cords, thus supporting Coyne\textsuperscript{6} and Desvernine's\textsuperscript{7} definition

\begin{itemize}
  \item 1 “Growth in the Larynx,” 1871, p. 52.
  \item 3 “Traité pratique des maladies du larynx,” Paris, 1876.
  \item 5 “Polypen des Kehlkopfs,” Tübingen, 1868, p. 30.
\end{itemize}
of the vocal cords as consisting of the whole ligamentous system constituted by the thyro-arytaenoid-cricoïd fasiculus and by the ascending crico-thypo-arytaenoid fibres. The mucosa of the cord therefore extends inferiorly with the band to the upper border of the cricoid in the whole ascent of the cricoidal ascending fibres. All the glands embedded in this region would therefore be considered constituent elements of the glandular apparatus of the vocal cords. The question of the position of these glands is of importance since they may be the starting point of neoplastic changes both innocent and malignant.

In view of this difference of opinion as to the true definition of the vocal cord and the question at issue in connexion with adenomatous growths, the assistance of Professor Shattock has been sought. He has very kindly made investigations which I now bring to your notice. He has prepared some beautiful coronal sections through the larynx, showing the true limitations of the vocal cords and the neighbouring glands. These have been specially drawn for me under his supervision, together with sections of the adenoma exhibited by Dr. Macleod (by kind permission of the latter).

Remarks by Professor S. G. Shattock, F.R.S.

The occurrence of adenoma of the ventricular band, and of the wall of the ventricle is well established, and seeing that these structures include a striking collection of mucous glands the position of such growths presents no difficulty. The subject of adenoma of the vocal cord, however, is by no means so straightforward, and whether such tumours may arise in connexion with this, depends upon what anatomical definition is adopted of the structure in question. Over this writers are not in agreement.

For the elucidation of this matter a series of microscopic sections were made in the coronal or vertical transverse plane, from the larynx of a middle-aged woman who died of puerperal eclampsia, the tissue being hardened in Kaiserling’s formol solution, followed by increasing strengths of alcohol. The sections were made through the middle of the cord and ventricular band, including the entire wall of the ventricle, the parts being dissected away (after they were hardened) close up to the inner surface of the thyroid ala. As is shown in the accompanying figure (fig. 3), the free edge of the vocal cord is covered with a closely adherent mucosa furnished with papillae over its summit, and invested with squamous epithelium. Beneath this the structure consists of a
compact collection of fibrils of elastic tissue (viewed in cross section); and immediately on the outer side of the latter lies the muscular mass of the thyro-arytenoid, the bundles of which are likewise viewed in transverse section. An inspection of the figure will show that in the subglottic area there is an abundance of gland tissue, one of the ducts from which (lined with columnar epithelium) passes upwards to open on the surface a short distance below the summit of the ridge forming the cord. Passing next, to the upper aspect of the cord it will be observed that although there are no glands in the thicker, apical portion of the elastic tissue, there are a few acini scattered in the immediate vicinity of the last-named tissue when this is spread out towards the floor of the ventricle. The question at issue thus resolves itself, as
before said, into what definition is adopted of the vocal cord. There are two criteria which suggest themselves in this connexion: (1) The character of the epithelium; (2) the limitation of the elastic tissue. The presence of a squamous epithelium on the vocal cord is obviously related to the function of the latter. Whether the substitution of

![Fig. 2.](image)

Portion of the lowest part of the preceding section as seen under \( \frac{1}{3} \) obj. Some of the gland cells are finely vacuolated. The gland spaces are filled with mucin. The acini are supported by adipose tissue. A, vacuolated gland cells; B, mucin; C, fat cells.

(Specially drawn for Dr. Irwin Moore by kind permission of Dr. Macleod.)

squamous epithelium for the columnar-celled which covers the mucosa of the ventricle and of the trachea has been acquired and afterwards inherited, or whether its presence is due to an antecedent germinal
Coronal section through middle of normal human (female) larynx, showing the distribution of the gland tissue and the lymphoid tissue of the ventricle (logwood and cosin stain; 2 in. obj.). A, Elastic tissue of cord; B, gland acini lying in periphery of the cord; C, gland duct, near surface of summit of cord; D, gland acini lying in elastic tissue and in an area covered with squamous epithelium; E, lymphoid tissue in wall of ventricle.

(Preparation by Professor S. G. Shattock, F.R.S., specially drawn for Dr. Irwin Moore.)
change (or "mutation"), co-ordinated with the evolution of the glottis is a problem outside the present subject. That the squamous character of the epithelium, however, is not acquired from the use of the glottis in each individual after birth is readily proved. For, I find, by means of coronal sections, that the differentiation is well pronounced in the human foetus at the sixth month: whilst the ventricle and subglottic area are covered with typically columnar epithelium, the covering over the vocal cord is a thin layer of the squamous-celled kind. The extent, then, of the squamous epithelium is one criterion of the area which may be assigned to the cord. Secondly, there is the extent of the elastic tissue,

![Diagram showing the true cord from another section of the same larynx stained with Unna-orexin. The elastic tissue is stained a deep blackish-brown. A, elastic tissue; B, gland acini lying in elastic tissue and in an area covered with squamous epithelium; C, gland ducts opening near summit of the cord; D, gland acini lying in elastic tissue and in an area covered with squamous epithelium.](image)

(Preparation by Professor S. G. Shattock, F.R.S., specially drawn for Dr. Irwin Moore.)

into the outer aspect of which the deepest fibres of the thyro-arytaenoid muscle (aryvocalis) are inserted at increasing lengths from behind forwards. The precise limits of the elastic tissue as demonstrated by the use of Unna's acid orcein (which stains the fibres of a deep blackish
brown) are depicted in fig. 4. The tissue, of which the volume as viewed in coronal section, is comparatively small (much more so than is frequently imagined), is disposed somewhat in the form of an inverted V, its angle being the thickest part: the thyro-arytenoid muscle lies immediately on its outer side. What, therefore, is the distribution of gland tissue in relation (1) to the squamous-celled area, and (2) to the elastic tissue?

In the logwood-eosin section, gland acini lie in the connective tissue where the latter is (as studied microscopically) invested with squamous epithelium. And as seen in the orcein-stained section, the gland tissue at the same spot lies fairly included in the peripheral limit of the elastic tissue, without the intervention even of muscle fibre. It is clear, therefore, than an adenoma may arise in the peripheral portion of the upper moiety of the vocal cord. And that such a tumour, as it increased, would extend inwards, and might eventually come to involve the more prominent edge of the cord needs no proof. It is equally obvious that an adenoma might arise, also, in the adjoining floor of the ventricle, whence it might secondarily involve the cord. There is no gland tissue in the thicker, main portion of the cord, although as shown in the orcein-stained section, gland ducts penetrate the connective tissue intervening between the elastic and the squamous epithelium, close to the summit of the ridge. In the subglottic area, the gland tissue reappears, its higher portion being included in elastic tissue; and the epithelium of the suprajacent mucosa being squamous-celled. It is hardly possible here to define a limit to the elastic tissue, for it passes inferiorly into the thin expansion of the lateral portion of the cricothyroid membrane which is fixed below to the inner edge of the upper border of the cricoid cartilage. The squamous epithelium, moreover, is continued farther downwards over the subglottic area than is that which lies in the plane of the floor of the ventricle. In coronal sections prepared from an adult male, the junction of the subglottic squamous epithelium with the true columnar-celled epithelium of the trachea took place at a distance of 7 mm. below the summit of the vocal ridge.

So far as any definition of the lower limit of the vocal cord can be given, it is nevertheless difficult to offer a better one than that it corresponds with the transition of the two kinds of epithelia referred to. It may be noticed in passing, that the epithelium over the inner—i.e., the exposed, surface of the ventricular band—is largely of the squamous kind, and consists superficially of a few layers of flattened cells over a zone of polymorphous epithelial elements, and a deeper
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pallisade series. In a series of similar coronal sections made from a male larynx, the arrangement of the several structures was similar, with the exception that the acini in the outer extremity of the horizontal portion of the elastic tissue lay beneath a columnar-cell clad portion of the mucosa. In such a case the cord must be reckoned as extending to the limit of the elastic tissue. Some may be disposed to make the vocal cord include the aryvocalis muscle, and even the adjoining part of the thyro-arytênoid which occupies the fold. If so, it may be suggested that the term “pars elastica” might be advantageously applied to the elastic element, as facilitating reference to the different constituents of the cord.

In conclusion, attention may be directed to the conspicuous amount of lymphatic tissue distributed in the submucosa of the ventricle. And it might repay pathological research to ascertain how far this becomes involved in such diseases as lymphadenoma, lymphatic leukaemia, typhoid, or tuberculosis.

Dr. Jobson Horne: Referring to the last remark of Dr. Irwin Moore that Mr. Shatock had come to the conclusion that an adenoma could spring from the vocal cord itself, the whole question turns on what we mean by the vocal cord. It is now some years ago—I think it must be twenty years ago—that I demonstrated that the vocal cord proper needed to be defined histologically, as that part, and only that part, of the cord which is covered with squamous epithelium. I do not see how an adenoma could arise from that portion, and therefore it could not arise from the vocal cord proper. But, as we have see in the diagrams exhibited to-day, an adenoma can arise immediately outside the vocal cord boundary, which I have defined. An exact knowledge of the histology of the mucous lining of the larynx is most important because one can practically base the whole of the pathology upon it. Once you have defined where the columnar epithelium and glandular structure begin and end, and where the squamous epithelium begins and ends, one knows the sites of origin of different diseases. The reason, for instance, that tuberculosis does not start in the vocal cord proper is, because there are no glands there. And the reason you see that disease starting and spreading in other parts is because in those other parts there are glands, and they are covered with columnar epithelium. Squamous epithelium is more defensive against tuberculosis than is columnar, and it thus protects the larynx. On the other hand, in most cases of intrinsic malignant disease of the larynx, the disease fortunately starts in the vocal cord proper, that is in the squamous portion. There is, as we know, another variety of intrinsic carcinoma which starts in other parts of the larynx and is more malignant. This is a governing factor in the results obtained from operations for intrinsic carcinoma. Very good results are obtained when the growth originates in and
is confined to the cord itself as I have defined it. In fact those cases are hardly malignant. But if malignant disease originates in or spreads to a part with glands and columnar epithelium then the outlook is more serious, and those are the cases in which recurrence is more probable. Then again in cystic disease of the larynx, which we have been discussing this evening, I have shown that the site of origin is the governing factor in treatment and prognosis.

Given an exact knowledge of the histology, then the whole pathology of the larynx is understandable.

Nasopharyngeal Growth.

By Irwin Moore, M.B.

Patient, a female, aged 63, was sent to me by Dr. Gibb, of Maidstone, on May 9, complaining of difficulty in breathing through the nose, first noticed four years ago, gradually progressing and accompanied by deafness. The nasopharynx is occupied by a large hard swelling which apparently originates, subperiosteally, from the sphenoid. It is evidently (?) malignant.

A skiagram, by Dr. Finzi, shows both sphenoidal sinuses to be very opaque. The posterior surface of the posterior clinoid process is considerably eaten away and is no doubt involved in the growth. The left antrum is somewhat opaque.

Opinions are invited as to diagnosis and treatment.

DISCUSSION.

Mr. E. D. D. Davis: Three or four years ago I showed a more advanced case of this type, a woman who was in hospital with complete double ophthalmoplegia, involving the third, fourth, and sixth nerves, and was blind. She also had a growth in the nasopharynx. The post-mortem examination showed a carcinoma of the pituitary fossa and sphenoid, and it involved the nerves of the muscles of the eye in the same way as in this patient. This patient has a squint, and her pupils react sluggishly, therefore I think it is malignant growth invading the sphenoid and pituitary fossa. In my case there were no metastases: it was localized to the skull, where there was considerable destruction of bone. The patient was comatose and died of cerebral compression. In the fatal case I mentioned the sections were shown to be carcinoma, and the infiltration was characteristic of the same disease.

Mr. Lawson Whale: It would be instructive to have the perimetric charts of this patient's vision.
Dr. Jobson Horne: Are these cases of nasopharyngeal growths strictly speaking malignant in the sense that they destroy the patient by metastases? Do they not tend to destroy by direct pressure and extension, and therefore are not malignant in the commonly accepted meaning of the term?

Dr. Irwin Moore: The skiagram shows destruction of the right posterior clinoid process. I have recently examined the patient under an anaesthetic and found the whole post-nasal space blocked by this growth, which seems to originate under the periosteum. It is hard, smooth and not ulcerated. Dr. Gibb is anxious to hear any suggestions as to further treatment. He has used colloidal copper injections through the nose into the growth, and apparently it has had a marked shrinking effect, for the growth has previously been seen by him bulging below the soft palate, but that was not the case when I first saw the patient on May 9.

Dr. J. A. Gibb (Maidstone): I saw the patient in the hospital in September, 1918, and after post-nasal examination, not under anaesthetic, I concluded it was at any rate partly malignant, and coming from the sphenoid. The growth extended below the soft palate, and it seemed to be inoperable, and so I thought it would be well to try colloidal copper, supplied by Parke Davis. I started with it, and the prolongation of the growth disappeared. After I had gone on with it for a fortnight, however, she thought her hearing and breathing were worse. Then I made injections through the fossa into the growth, and these gave her some pain at the moment. She said she could not do without these injections. I kept it up fortnightly, and then sent her to Dr. Irwin Moore in order to ascertain what further action should be taken.

The President: There has been a great interest of late years in the use of various forms of copper for inoperable cancers, and I should like to hear the experiences of members as to its use.

Mr. Tilley: The copper preparations have been tried on large obviously malignant excavating growths which I have seen lately, but they did no good at all. One of the patients considered that it made him worse. I suggest that members should try these remedies for disease in regions which are easily seen by the naked eye. In the case of a growth in a dark cavity, where you cannot be certain as to the extent of the disease, you are apt to think diminution or retardation of growth is due to the use of the remedy. It reminds me of the claims made for fibrolysin. Cures of structures in invisible regions were reported, but I never saw a Dupuytren’s contraction much benefited.

Mr. W. Stuart-Low: I have seen two cases in which the copper preparation was assiduously used in malignant disease of the ear for over six months. One lady had obvious malignant disease which was operated upon twice. She went to several surgeons and specialists, and copper was elaborately tried, the case being observed very carefully but with no beneficial result.
Malignant Growth of the Deep Pharynx

By Irwin Moore, M.B.

Referred to me for further opinion by Dr. Jeffery, of West End, Hants.

Female, aged 47, with a large fungating growth in the deep pharynx, overlapping the larynx and causing much stridor; relieved by a recent tracheotomy. Patient has been able to swallow only liquid food since Christmas.

Opinions are invited as to the question of further treatment by diathermy or radium.

Epithelioma of the Tonsil and Fauces in a Woman, aged 57.

By William Hill, M.D.

Opinions are invited as to the best form of treatment.

It was suggested that this case should be treated by combined surgical operation and diathermy.
Case of (?) Sarcoma of the Tonsil.

By W. M. Mollison, M.Ch.

A. W., aged 22, presented herself at Guy's Hospital, on May 6, on account of pain in the right side of the face, and a swelling in the neck. For eight or nine months she states that she has had neuralgia, referred vaguely to the upper jaw. A month ago a molar was removed. There is a mass of enlarged glands about the angle of the jaw: the glands are movable. The right tonsil region is much swollen, and there is redness and oedema of the right side of the palate. The swelling is tense but not tender; puncture with a needle did not produce any pus. The nasopharynx is completely blocked. The Wassermann reaction is strongly positive: the patient has been given a dose of novarsenobenzol, but no obvious improvement followed except that swallowing became rather easier.

DISCUSSION.

Mr. W. Stuart-Low: The malignant change has advanced considerably in this case in the tonsillar region, as ulceration and induration are evident, and there is a hard gland in the neck. It is obviously inoperable in the usual way with the knife, but the carious stumps should be extracted, as they keep the ulcerated surface septic, and thus increase the pain. It is a case for removal as freely as possible by means of the sharp diathermy point, or with the knife, and then by freely searing the wound with the flat diathermy point. Elixir of mucin as a mouth-wash and gargle is most soothing in such cases.

Mr. G. W. Dawson: I showed a similar case here some time ago, though the glands in my case were not so large as in the patient now shown. I did a laryngotomy and removed the tumour, and there was surprisingly little bleeding. I also removed the glands. I then sent my patient to the Radium Institute, where he had two applications of radium at an interval of a month. I showed him here again after eighteen months, and he is now quite well.

The President: Would it not be well for Mr. Mollison to try the old syphilis remedy, mercurial inunctions, in view of the positive Wassermann reaction?

Mr. Mollison (in reply): I am not convinced as to the value of the Wassermann reaction in this case. She has not improved as a result of galyl injections and mercury and iodide given by the mouth. I consider the case malignant.
Case of Extensive Lupus of the Alveolus, Nose, and Larynx.

By E. D. D. Davis, F.R.C.S.

A. H., male, aged 34, a clerk, was first seen in 1912 at Mount Vernon Hospital for extensive tuberculous infiltration of the arytenoids, ventricular bands and epiglottis. The stridor and laryngeal obstruction were so marked at one time that tracheotomy was advised but was refused. The infiltration was treated by cautery puncture, and in December, 1913, the top of the epiglottis was amputated. The patient steadily improved until December, 1917, when he developed lupus of the nose, and, later, lupus of the alveolus and palate. The larynx has healed but there is fixation of both vocal cords. The Wassermann reaction is weakly positive.

DISCUSSION.

The President: Dr. Shaw-Mackenzie claims that the copper preparation has a selective action in tuberculosis, and it might be worth trying it in the present case. I have tried it intravenously in a case of tuberculosis of the larynx, and there seems to be some improvement in two cases, though whether this can be attributed solely to the alanine remains to be seen. Has Mr. Davis tried biniodide of mercury?

Mr. E. D. D. Davis (in reply): I am willing to try anything. The patient has been visiting the department since 1912, and he is very persevering. His condition oscillates considerably; he will be comparatively well for five or six months, and then a fresh outburst of lupus occurs in some part of his nose and throat. The Wassermann reaction is weakly positive. After that report he had potassium iodide and mercury, also neo-salvarsan: I have not tried mercury inunction. The lupus has been curetted and cauterized, and he has had yellow oxide of mercury ointment, and other treatment. I will apply pure carbolic, which I know has been used for lupus of the face in the Finsen Light Department.

Case of Chronic Osteomyelitis of the Maxilla.

By G. W. Dawson, F.R.C.S.I.

A housemaid, aged 30, was sent to the hospital in May, 1912, with an alveolar abscess behind the right central incisor which had burst into the nose and produced a sinus in the floor of the nose. In addition there was a sinus passing through the right nasal process of the maxilla
to the right middle meatus following operations at an eye hospital. The maxillary antrum had also been opened and part of the inferior turbinal removed. The Wassermann reaction was negative and sections of granulation tissue from the nose showed chronic inflammatory changes, but there was no evidence of tubercle.

As the alveolar abscess did not heal and suppuration of the antrum was profuse, a Caldwell-Luc operation was performed on the right antrum: the alveolar abscess was curetted and a small sequestrum removed. A chronic abscess in connexion with the right canine was also curetted. Later a large sequestrum of the right alveolus separated and was removed. There is now a large opening into the right antrum in the canine fossa, and the condition of the interior of the nose resembles lupus, particularly the granulation tissue on the anterior part of the septum.

The patient has not at any time had a temperature and has not appeared to be ill.

If one attacks these cases through the nose by scraping and applying carbolic acid, ten days are required for healing, and at intervals of a fortnight the soft parts can be attacked. In this way I have got several such cases well.

Bony Nasal Growth.

By G. W. Dawson, F.R.C.S.I.

Girl, aged 17. Complains of nasal obstruction for the past twelve months, and for the last few weeks of watering of the eyes, particularly the right. The right superior maxillary bone is prominent and hard. The right nasal cavity is filled with a hard, bony tumour, producing complete obstruction to the airway. That portion of the left nasal cavity below the inferior turbinate body is occupied by a similar formation. There is no pain.

Discussion.

Mr. Tilley: Mr. Dawson asked me to see his case, and I referred him to an excellent paper by Mr. Westmacott, and published in the Proceedings of the 1913 International Medical Congress in London, with some excellent illustrations. Since that date I have seen two cases. One patient had pain and nasal obstruction, with some purulent nasal discharge. I tried to do the ordinary canine fossa operation for him, but it was very difficult because the whole upper jaw was a
mass of very cancellous bone, the antrum was reduced to a chink, and the whole anterior part of the nasal fossa was filled with this hyperostosis, probably some chronic inflammatory condition. We removed a good deal of bone by means of chisels, &c. Eventually he recovered, and was very pleased with freer nasal respiration. I mention this in order to warn Mr. Dawson that if he attempts to operate in this case he will find his task difficult, and granulations are liable to spring up afterwards. Unless his patient is suffering a good deal I should be inclined to let her go until such time as she insists on something being done, rather than letting the proposition come from the surgeon's side.

Mr. Lawson Whale: Mr. Dawson does not say anything about the skiagram. I suppose the jaws are solid. If the bony growth were a little more symmetrically grouped and a little higher on the left side, it would be like the condition described by Mikulicz as a symmetrical hyperostosis of both upper jaws. This is supposed to be a trophic condition in connexion with the fifth nerve. I saw one before the war, but I have now lost sight of it.

Mr. G. W. Dawson (in reply): It would be a difficult case to tackle, and I agree with Mr. Tilley. I am doubtful what deformity the operation would leave if one were to chisel away most of the jaw. The region affected extends up to the eye, and she is complaining of some watering from it.

Case of Tuberculous Laryngitis.

By T. B. Layton, F.R.C.S.

Regimental Sergeant-Major N. is in the First London General Hospital (T.F.) with tuberculous disease of both lungs; the bacilli have been found in the sputum; the condition of the larynx is an improvement on what it was a month ago. There is some pain on swallowing. The exhibitor would like advice as to whether this is a suitable case for cautery or other local treatment. The patient has abstained from speaking for about one month, and members are asked to refrain from making him speak more than is absolutely necessary.

Discussion.

Dr. Jobson Horne: The best medicine this man can have is complete silence in suitable surroundings. I would treat the man's general condition, and leave the larynx absolutely alone. The gratifying results following the use of the cautery are not uncommonly obtained in cases in which the general condition has been treated and the larynx would have improved without outside assistance.
Mr. Layton (in reply): I brought the case in order to ask advice. He is living on the balcony of the hospital, and steps are being taken to get him into a sanatorium. We tried to keep him absolutely silent, but after three weeks we had to give it up and allow him to talk a little, as it was too great a mental strain upon him.

**Tumour of Right Submaxillary Region and Floor of Mouth; ? Actinomycosis.**

By W. H. Jewell, M.D.

G. D., male, aged 59. There is a history of symptoms extending over eight weeks. His occupation is connected with horses. There is a very hard, painless, almost immovable swelling in the right submaxillary region, connected with the lower jaw and continuous with an equally indurated swelling in the floor of the mouth, which bleeds readily when probed. There are no enlarged glands nor a sinus. The skin is not involved. Wassermann reaction negative. X-ray plates (negative) shown.

**DISCUSSION.**

Mr. Lawson Whale: Upon inspection I thought the growth was carcinoma.

Dr. Jewell (in reply): I regard this as actinomycosis. If it were carcinomatous I should have thought there would have been more pain and involvement of glands. Should this diagnosis be correct one would expect a sinus to form in the course of two or three months, and then to find the ray fungus, which would be conclusive evidence.

Mr. Sydney Scott, F.R.C.S., exhibited Three Instruments to simplify the control of Hæmorrhage in difficult cases of Enucleation of the Tonsils.

**DISCUSSION.**

The President: I find that the ordinary Spencer Wells forceps, applied to the base of the pillar towards the conclusion of the operation, generally prevents bleeding when the tonsil is finally severed. The more we see of these instruments in connexion with enucleation, the more formidable the operation is made to appear.

Mr. E. D. D. Davis: One of the many advantages of enucleation of the tonsil is that all hæmorrhage is arrested before the patient leaves the operating table.
Section of Laryngology.
Presidential—Dr. James Donelan.

SUMMER CONGRESS, May 2-3, 1919.
(May 2.)

Dr. James Donelan, President, and subsequently Brigadier-General H. S. Birkett, C.B., C.A.M.C., Honorary President, in the Chair.

Some Suggested Alternatives to Operation for "Adenoids" and Enlarged Tonsils in Young Children.¹

By James Donelan, Ch.M., M.B.

Object of paper: To elicit the views of the Section on certain recently suggested methods of treatment of nasopharyngeal vegetations and enlarged tonsils as alternatives to their immediate removal, especially in the case of very young children showing symptoms of nasal obstruction.

The desire of medical men as well as the relatives of young children to try anything rather than an operation has called forth periodical suggestions of this kind. Some objections to operation, especially for enlarged tonsils, when there is a growing conviction that enucleation is the only efficient operation. See Discussion on Adenoids in Proceedings, (Section for the Study of Disease in Children), pp. 26-58.

Some of the suggested alternatives have no rational basis, and are only mentioned because, in view of the wide publicity given to them by "stunt" journalists, it seems desirable in the public interest that they should receive an authoritative condemnation by this Section. The recent advocacy of "snuffs" for the treatment of "adenoids" is the

¹ The papers read at the Congress are published in the Proceedings in abstract and will be printed in extenso in the Journal of Laryngology, Rhinology and Otology.
most prominent and typical example. A brief glance at the history of treatment by errhines in itself gives sufficient ground for condemning them. An invitation to the Section to condemn them and to find some method, consistent with medical ethics, by which such an authoritative condemnation may reach the general public. A reference to "breathing exercises" showing that their usefulness is more likely to be post-operative.

Other suggestions have the support of eminent scientific opinion, and are therefore worthy of our careful consideration. Medicinal treatment—e.g., by thyroid extract, based on the views of many well-known writers that "adenoids" and enlarged tonsils are in many cases a symptom of thyroid inadequacy or chronic minor congenital hypothyroidism. Consideration of the evidence for and against this view. A brief analysis of twenty cases of children of from 1 to 4 years in which thyroid extract was tried by the writer; effect of varying dosage and method of administration; results and general conclusions.

DISCUSSION.

Dr. P. Watson-Williams: The subject of Dr. Donelan's contribution still lends itself to a good deal of further investigation. I do not know that we have such a clear idea on the pathogenesis of adenoids as seems desirable. When we find the suggestion made that adenoids may be due to thyroid insufficiency, I think it shows there is room for a good deal of clarifying of ideas. Adenoids, as we know them, are a response of lymphoid tissue to infection. Enlarged tonsils, too, are essentially a septic hypertrophy of lymphoid tissue. I have never seen anything to lead me to the view that there is any connexion between typical adenoids and thyroid insufficiency. If they are due to infection, there are two stages: the preliminary stage with rhinitis only, before hypertrophy occurs; later there is hypertrophy. In the first of these, certain measures, short of operation, may be useful. But, if we accept this pathogenesis, when hypertrophy has occurred, it is futile to suppose that anything short of operation will get rid of the septic focus. If they are due to septic infection, one might hope something even from vaccines. Some years ago I published, in the Proceedings, accounts of a research by Professor Walker Hall, of Bristol, and myself, on the pathogenesis of adenoids and enlarged tonsils. Dr. Hall used all the needful precautions, and made cultures from the interiors of tonsils and adenoids. It occurred to me that in cases in which no operation could be justified, a multiple vaccine could be used. Though I am not a great believer in vaccines, I have tried them in some cases, with benefit resulting. I do not suggest resorting to such less adequate measures, sneezing, and so on, when we have ready to hand a method of operation which effects a ready removal. I cannot understand how anyone with a practical experience of operations for adenoids, can suppose that it would be useful to employ any
alternative to this practical and simple procedure for any but exceptional cases. I suggest that in many cases—in more than we are inclined to think—a constant rhinitis may be due to sinus infection, either latent or manifest. We are apt to think that children are not subjects of sinus infection, hence we do not look for it in them. More often than we suppose, such infection leads to the hypertrophy which constitutes adenoids.

Mr. Layton: Six months before the war started, I read a paper before the Medical Society of London, in which I put forward a plea for fewer operations for adenoids. This paper by Dr. Donelan fits in with what I then urged; not that when there is a large mass of fibro-adenoids in the nasopharynx there is any other way of getting rid of them than by operation, but that there are an enormous number of adenoid operations done in this country on conditions which are not enlarged adenoids, but, rather, temporary enlargements of lymphoid tissue in the nasopharynx, which can be treated in other ways and will get well under such treatment. There is no essential difference between the stand I took then and that taken by Dr. Donelan now. The very fact that other methods of treatment are put forward, by people who are not laryngologists, is a sign that the public are not quite satisfied on the matter. It is interesting to note that the difference of opinion which prevails comes from Australia. I have found that Australians are extremely quick in picking up criticism of any old-standing treatment, and very ready to try new methods of treatment, and during the war I found them distinctly ahead of the ordinary English people in trying such new methods.

Dr. Perry Goldsmith: In answer to the last speaker, I take it it is not the size and consistency of the adenoid that points to operation, but the symptoms produced by the growth. If the mass is so large that it produces symptoms, whether in the ears or in breathing, the only remedy to consider is operation. Postponing operation, meanwhile using paints, pigments, and enjoining breathing exercises, means allowing the adenoids to do irreparable damage in connexion with the ear. I should not like it to go out from here that England is peculiar in the too frequent operations for adenoids, for the same is true of Australia and my own Continent of America. We have to remember that lymphoid tissue very easily becomes inflamed and slightly enlarged, and then such a patient will exhibit all the symptoms of adenoids: he has got acute adenitis, and it is only when there is a recurrence of that adenitis, associated with rhinitis, or a moderate ear attack, that operation should be considered. With regard to the giving of thyroid extract in these cases, I hope it will not be gathered from us by general practitioners that there is any effective treatment for adenoids short of operation. I do not say that every case of enlargement should be operated upon, but cases requiring operation sustain a great deal of damage by the adoption of temporary measures, such as sniffling snuff, and that sort of thing.

Sir William Milligan: This subject should be considered under two aspects: first in relation to children under 4 years of age, secondly in relation to those over that age. In the first period true fibrosis is comparatively
infrequent, but there is vascular engorgement, which causes trouble and requires treatment. After 4 years of age, I think fibrosis is very frequent, and I cannot understand the position of the laryngologist or rhinologist who refuses to operate for what is organized hypertrophied tissue. Whether it is septic or not is a debatable point. If it is left in situ it is likely to do the patient material harm. Surgery offers the best course of treatment for such cases. Some little time ago, I was interested in the question of thyroid inadequacy, and I tried thyroid extract in adenoid cases, both in young children, and in those over 4 years, and I never saw the slightest result in either class of case. I admit it might have some effect upon children of under 4 years in whom the condition is one of vascular engorgement. But the snuffling which is being practised so largely, is highly dangerous, and this Section should authoritatively condemn it. It is the height of folly to expect it will do good, and I am convinced I have seen considerable damage done to the ears by the persistent irritation of the nasal mucous membrane, with the result that the tympanic membrane has become flaccid, and the tympanum more or less inert. Where there is—and one cannot deny there is at times—a legitimate reason for not interfering in a child under 4 years of age, it is fair to try some other form of treatment, warning parents that it may not succeed. In those cases I have seen material improvement follow the use of alkaline detergent lotions if carefully used. I do not think it does harm to the aural appendages, but relieves the passive congestion at the back of the throat and nose. But I have no hesitation in surgically removing adenoids at any stage or at any age provided there is sufficient obstruction to demand it. With regard to breathing exercises, we do not hear so much of these as we used to. Still, we cannot deny their value as a post-operative measure. Many adenoid operations are done in this country in patients having no adenoids, and I think we, as a Section, are somewhat to blame for speaking of the adenoid operation as a simple operation. It is constantly referred to as a trifling operation, yet I know of no operation in surgery which can be made more of a mess than this. I have seen part of the uvula, and part of the Eustachian tube torn off by an inexperienced operator in dealing with adenoids. As I say, there may be no adenoid tissue at all, though there may be marked congestion. The nasal obstruction is frequently due to some other cause which has been overlooked—such as septal or turbinal trouble. Operations in these cases should be left in the hands of those who are competent to operate.

Colonel Sharp: The operation for tonsils and adenoids is too frequently performed. The practice at present is something as follows: Given a debilitated child, with enlarged tonsils and adenoids, operation is recommended. In the majority of cases no doubt the treatment is sound, but in many cases I believe operation is not only not sound, but a misfortune to the individual. More information is required regarding the physiology of tonsil tissue, and until this has been definitely determined I think fewer enucleations should be carried out. With regard to nasal hygiene and the present popular movement towards sneezing agents, &c., some authoritative statement should be made by this
Section. That a sound nasal hygiene is desirable and that benefits follow nasal douching cannot be gainsaid. Just as the mouth and teeth prepare the entrants to the stomach, in like manner do the nose and nasopharynx function for the lungs; yet in the ordinary daily toilet the nose is left to itself, while the mouth and teeth are specially cared for.

Mr. Tilley: I agree entirely with those who take the standpoint that once adenoids are well developed and are causing definite symptoms which are becoming chronic, there is nothing for it but surgical removal. But in the class of case under discussion, in which one is doubtful as to whether operation is necessary or not, that is, where the symptoms are sometimes latent, and at other times exaggerated, operation might be saved in many instances if care were paid to the teeth. Four or five years ago, Mr. Layton emphasized this same point and I agreed with his contention. Probably in most of these cases of mild lymphoid enlargement, the true cause of the trouble is a mild form of infection from the surrounding regions. Dental sepsis, even in milk teeth, is often overlooked.

The President (Dr. Donelan) (in reply): I am sure we are agreed that the lymphoid enlargement may be variable and often temporary whereas what chiefly calls for operation is the increase of connective tissue. Sir William Milligan has rightly called attention to the advantages of alkaline detergents for removing the thick germ-laden secretion. Many years ago Morell Mackenzie substituted sulphate of sodium for sodium chloride in his well-known three alkaline formula, as the chloride was found to dissolve the cement between the epithelial cells of the normal nasal passages. Colonel Sharp also referred to the desirability of some regular system of nasal toilette. As I said in my paper, the nasal mucus is the natural detergent and blowing the nose without a handkerchief the best way of getting rid of secretion that has fulfilled its office. I am glad that so strong an opinion has been expressed on the suggested snuff treatment. It is satisfactory too to have one's experience of the limited influence of thyroid extract confirmed by that of Sir William Milligan and that it is only in cases where the obstruction is chiefly the result of functional engorgement or where there is little or no fibrosis that any lessening of it may be looked for.

Latent Sinusitis in Relation to Systemic Infections.

By P. Watson-Williams, M.D.

Case I.—Rheumatoid Arthritis with Chronic Catarrhal Deafness.

W. B., male, aged 54, referred to me by Captain Duckett, R.A.M.C., at Bath, on account of deafness. The deafness was due to chronic adhesive otitis, both tympanic drums were retracted, and the malleus
in both ears almost fixed; Eustachian tubes patent, catheterization improved the hearing. Nothing abnormal was observed by anterior rhinoscopy, and by endo-rhinoscopy only a few strings of sticky secretion from the posterior ends of the inferior turbinals. But I was struck with his depressed condition and inability to concentrate his mind, which he attributed to his deafness. Further, I found he had a typical well marked rheumatoid swelling of the left wrist and finger joints, and the same in less degree was true of the right wrist and hand, the skin giving the soft silky sensation that I think is not unusual in rheumatoid arthritis. He had first noticed that the left wrist was becoming stiff and swollen three years previously, and later the right wrist, right ankle, and both knee-joints were involved. Two years ago he had been to Buxton for a course of treatment, and one year later had a course of baths and massage at Bath with benefit.


Report of cultures, &c., by Professor Walker Hall, from the sinus fluids. Maxillary antra: Right—film: small amount of mucus, occasional polynuclear cell, no cocci; culture sterile. Left (the copious thick pus)—film: heavy deposit of degenerated pus; no mucus, no organisms; culture, no growth. Sphenoidal sinuses: Right—film: considerable amount of mucus, a few polynuclears, no cocci; culture, heavy growth of Staphylococcus albus. Left—film: few flat cells; no polynuclears, no cocci; culture, sterile.

I would invite particular attention to the fact (1) that the left antral discharge of apparently almost pure pus was sterile to culture, and that the discharge was essentially degenerated pus; (2) that notwithstanding the seemingly innocent turbidity of the right sphenoidal sinus washing, with only a few polynuclears, the culture yielded a heavy growth of Staphylococcus albus. Undoubtedly those who take pus as the criterion of infection would have opened and drained the left antrum while leaving the right sphenoidal sinus untouched. This case shows the value of cultures being made when there is little pus to be obtained from a suspected sinus.

A fortnight later he returned for Eustachian catheterization, but was pleased to find that all his joints were better and the left wrist obviously less swollen and less stiff.
In this case the left antrum, the left frontal sinus, and both sphenoidal sinuses were subsequently freely opened and drained. He improved in general health and lost his depression, while his power of concentration had greatly improved. But the joint swellings and stiffness had much abated, and within a month all the old pains had gone. He found he could carry weights in his hand, that for many months would have caused too much pain.

**Case II.—Rheumatoid Arthritis with Appendicitis, &c.**

Miss B. D., aged 46. Case of rheumatoid arthritis, referred to me by Dr. Lemarchand, of Barnstaple, for post-nasal catarrh with clear or slightly muco-purulent discharge.

Nasal history: Between 1884 and 1886 she was under Dr. Woakes, and she says he cauterized the nasal mucosa with some "caustic," as it was said to be velvety. Subsequently she consulted three very distinguished rhinologists still with us, and there was apparently little evidence of sinusitis then, since no operation was advised beyond the removal of tonsils.

Rheumatic history: About the age of 14 she began to suffer from rheumatism in her left knee, and about a year later the right knee was involved as well as the left. Since then she has always been more or less stiff and rheumatic. About the age of 31 the left wrist and finger-joints became swollen and rheumatic, and have never recovered. Shortly afterwards she had neuritis in the region of the left shoulder. During the ten years previous to my seeing her she had also developed rheumatic pains and stiffness in both ankles and wrists, and she had undergone five courses of baths at Bath in different years, and for three years had courses of baths at Vittel. In 1911 she saw the late Dr. Lewis Jones for her sciatica, &c., and he suggested there must be some source of poisoning causing the rheumatism.

Appendicitis: In 1906 she underwent operation of appendectomy, which was performed by the late Mr. Lockwood. She was making excellent recovery till, on the ninth day, she developed pleurisy followed by pneumonia.

Nasal examination: Anterior rhinoscopy revealed nothing of importance beyond slight diffuse turgescence of the mucosa; no discharge seen. Endo-rhinoscopy showed a few strings of clear discharge and streaks of muco-pus in the right and left olfactory fissures. A good deal of tenacious opalescent mucous discharge was discharged post-nasally, and formed a film on the granular posterior pharyngeal wall.
Section of Laryngology

Exploration of sinuses: The absence of any definitely purulent discharge led me to withhold a diagnosis of any sinus infection until the maxillary antra and sphenoidal sinuses had been explored under cocaine anaesthesia. With my suction syringes, throwing in distilled water and sucking back the contents of the sinuses, I obtained clear returns from the right and left antrum and apparently almost clear returns from the right and left sphenoidal sinus. A sterile swab was also taken from the nasopharynx. Some collosol argentum was finally thrown into each of the sinuses. Culture and film preparations from the washings made by Professor Walker Hall:

From the Maxillary Antra. Film showed no cells, no cocci, and on culture both were sterile.


She felt better the next day or two, and thirteen days later—on October 24, 1916—declared she felt quite well and that her joints were certainly less stiff and that she had lost her eye headaches. For the next day or two the sphenoidal sinuses were mopped with disinfectant. She continued to feel so very well that she refused to have any further or more radical treatment, though I feared the symptoms would return after a month or two.

January, 1917: The pains and stiffness at the back of the neck, in the knees and left shoulder, had returned. Endoscopy showed a distinct streak of muco-pus coming from the left sphenoidal sinus.

January 19, 1917: Operation; both sphenoidal sinuses freely opened under general anaesthesia.

February 7: After daily lavage of the sphenoidal sinuses, all rheumatic pains gone, but the most striking objective feature was the marked diminution of the chronic rheumatoid arthritic swelling of the left wrist and the absence of pain.

Interpretation of history: In early childhood she probably developed a mild nasal sinus infection which remained "latent" and non-purulent. From systemic infection she began to develop "rheumatism," and later developed chronic rheumatoid arthritis. The nasal mucosa became thickened and velvety from the sinus infection in 1884, when she was aged 14. Later the tonsils hypertrophied in response to the infection,
became septic, and called for removal. After repeated attacks of gastric catarrh from swallowing the organisms, the deficiency in hydrochloric acid allowed constant intestinal infection, till the appendix became infected. At the age of 36 the appendix was removed, and all went well till a bronchial infection caused a septic pneumonia, nine days after the operation. Subsequently the removal of a definite and proved sphenoidal sinus infection was followed by marked improvement in general health, and by a remarkable improvement in the rheumatoid arthritis.

**General Remarks.**

Two cases prove nothing, but afford examples which, taken in conjunction with others, have led me to the following conclusions:

1. That a latent sinus infection may persist for a great many years. When one remembers that an infected sinus is an ideal physiological culture tube, provided polymorphonuclears are inactive, the varying virulence of the infecting organisms correspond with the variations in the symptoms due to systemic infection.

2. That chronic rheumatoid arthritis and other infective rheumatic symptoms may be due to a sinus infection.

3. That appendicitis may be due to infection through the gastrointestinal tract from a nasal sinusitis. A large percentage of chronic sinus cases have suffered from appendicitis, but though I believe a nasal sinusitis is not rarely the source of the appendicular infection I do not suggest that this is the usual source.

4. That a very copious or thick purulent discharge may be sterile, whereas a thin, opalescent or almost colourless discharge may yield a free growth of pyogenic organisms on culture.

5. The previous history of nasal sinusitis cases is worthy of careful notes.

I suggest that some cases of systemic septic infection attributed to tonsillar infection may be due to a nasal sinusitis, which has caused the tonsillar infection, with hypertrophy of the tonsillar lymph tissue structures.

**DISCUSSION.**

Mr. Layton: This is a very important paper, because, if the two cases narrated by the author prove to be common, we have in them an explanation of much that has been puzzling us for some time. The paper confirms the feeling we had recently when discussing matters with the Section of Ophthalmology that we do not quite know where we are treading in relation to these sinus infections. Without commenting on Dr. Watson-Williams' rhinological
technique, I would raise one or two points seriously with regard to bacteriological technique. In both these cases it was the sphenoidal sinus in which the organism was found: the maxillary sinus contained no organism. To draw fluid out of the sphenoidal sinus without infecting the cannula must be more difficult than it is in the case of the maxillary sinus. Another point is, that in four out of the five findings the organism was the *Staphylococcus aureus*, and in one only it was *streptococcus*. When we extract blood from a vein for cultural purposes, how often does the bacteriologist say there is only *Staphylococcus aureus*, and that that is probably a skin infection? that is frequently his statement even when he himself takes the blood. Will Dr. Watson-Williams tell us exactly what his bacteriological technique is, in order to ensure that in these sinus cases there has not been bacterial contamination from the skin or nasal mucous membrane?

Dr. William Hill: In reference to Mr. Layton's remarks, if Dr. Watson-Williams is correct—and I am not sure that he is—does it matter whether the infection is in the sphenoidal sinuses or in the nasal cavities proper so far as his main thesis is concerned—viz., the far-reaching effects of latent infective processes in the nose?

Dr. Dan McKenzie: There is one point on which, probably, the whole discussion depends. It is no doubt within your recollection that Sir StClair Thomson and Dr. Hewlett stated, as the result of investigations undertaken by them, that the normal nasal cavity is sterile. But recently there has appeared, from the pen of an author whose name I have forgotten, the account of a similar investigation conducted in America, and the nasal cavity was found by him to harbour micro-organisms which could be cultivated. Until this matter is cleared up it is impossible to estimate the value of the discovery of organisms in either the nasal cavity or the sinuses. The matter is therefore one which needs considerable investigation.

Mr. J. F. O'Malley: The association of sinus disease with rheumatoid arthritis, as brought out by Dr. Watson-Williams, is of special interest to me at the present moment, because eighteen months ago I had a case which typified the condition perfectly. The patient was a man, aged about 50, and he came to see me on account of orbital cellulitis. He gave a history of very bad rheumatoid arthritis for twenty-five years. When dealing with the orbital cellulitis I opened the ethmoid region and evacuated a large amount of foul-smelling pus. I drained it thoroughly and it healed well. He came to see me recently and said, “The extraordinary thing is that I have no aches and pains in my joints now, whereas previously they had ached for years and years.” I must have tapped the source of his infection.

Colonel Sharp: Some years ago I had a case which is worth relating in view of the present paper. The patient was a woman, aged 35, married, and had a child 18 months old. For twelve months she had suffered from fleeting rheumatic pains, and during the two or three months immediately preceding her admission to a nursing home had developed melancholia: she
had ceased to take an interest in husband or home and neglected her child. She was under the care of a physician for two months; many forms of treatment were tried. In the end, I was asked to see her, to examine her tonsils, in the hope that something there might be found to account for the rheumatism. Nothing abnormal was found in the tonsils, and the nose was healthy. The left sphenoidal sinus was easily probed, was found to be large, and a tiny quantity of slightly milky fluid escaped. The sphenoidal sinus was freely opened, and with the happiest results. Within three days she asked for her child, and was restored to her usual cheerful disposition. In ten days she left the nursing home well, and quite free from her rheumatic pain. I have seen her husband two or three times, and he says she has remained well and been normal since the operation.

Dr. Batty Shaw: As a general physician I have learned many lessons from specialists. I am convinced that arthritis—whether we call it rheumatoid arthritis or by any other name—is really an infection, and in such cases one hunts for every possible source of infection. Mr. Tilley has been busy lately in watching some of my cases for me to ascertain whether there was any centre of infection in them: nor do I spare the dental department of my hospitals. And one keeps an eye on dysentery and colibacilluria: so that in attending this meeting I find myself in a congenial atmosphere. As a physician treating rheumatoid arthritis as part of his hospital work, I have had to learn this: that, over and over again, the source of the infection is hidden, and that even when we think we have found the point of entrance we cannot find the causa causans—it has entered the seat of infection and has disappeared into the system. It may be that some of us are able to harbour organisms without serious symptoms. It is my belief, and it is based on the teaching of the late Dr. Charlton Bastian, that when we are apparently healthy we are not necessarily free from bacterial infection of the blood stream—in other words, we may be quite healthy disease-carriers. The view, if necessary, that carriers are far more common in medicine than we think, needs enlarging. It is not only an affair of typhoid fever, or other specific diseases; the carrier has a wider range. Therefore I am not surprised that Dr. Watson-Williams' cases showed comparatively harmless non-arthritic organisms such as the staphylococcus. I expect the causative organism or organisms have vanished locally, but entered into the system. We must invoke the principle of carriers in medicine, and this paper clearly shows a seat of infection not likely to be detected by the general physician.

Dr. Watson-Williams (in reply): Mr. Layton asked me to describe the technique of the bacteriological work. That is rather difficult to do in detail within the limits of a short reply. But I have published the technique in a paper published some years ago in connexion with meningococcal infections of the sphenoidal sinus. As a general rule contamination does not take place in

exploration. I go through the anterior wall, not through the ostium, therefore
the cannula gets wiped in forcing its way through the anterior nasal wall.
Unless there was a real infection a culture would only give a poor bacterial
result. The criticism does not bear on cases in which there is a well-marked
growth, particularly when polynuclears are present: this phagocytosis is more
an evidence of an inflammatory change with infection. The staphylococcus is
not innocent: it is one of the worst enemies we have, it grinds slowly, but
very surely, and it is more difficult to get rid of than is the streptococcus. The
staphylococcus is more widespread as a cause of local infection than any other
organism, though the results may be less persistent. I am not suggesting that
the nose is the source of infection in all the cases of rheumatoid arthritis, but
my cases seem to prove that it is sometimes a hidden causative agent. With
regard to carriers, mentioned by Dr. Batty Shaw, one patient, aged 46, had
had infection going on since the age of 14. Probably we are all carriers, more
or less, the only difference is that in some there comes a time when the
organisms carry us—they get the upper hand. It is true my paper is founded
on only two cases, but I preferred that you should be presented with two fully
described cases than with a wealth of varying material from many. As I am
only a rhinologist, cases of rheumatoid arthritis do not come to me in the
ordinary way. In doubtful cases it is useless to rely on inspection: bacterio-
logical investigation must be carried out in detail.

A Method of treating Atrophic Rhinitis with Ozæna based
on an Alteration in Composition and Reaction of the
Substrate on which the Bacterial Ferments are acting.

PART I.—BACTERIOLOGICAL.

By T. H. C. Benians, F.R.C.S.

(ABSTRACT.)

(This paper is published in extenso in the Journal of Laryngology, September, 1919, p. 20.)

The treatment, which consists in a copious application of glycerine,
or glucose, or both together to the infected area of the nasal cavities, is
directed primarily against the fæctor of the disease. This fæctor, it is
assumed, is due to the destruction of protein bodies by bacterial trypic
ferments, resulting in the formation of various stinking end-products of
protein digestion. This proteolytic action we attribute to the bacillus
of Perez, which can be isolated from most severe cases of ozæna, and
which gives the characteristic fæctor in its cultures.

In addition to the bacillus of Perez various other bacteria are to be
found in the infected region, and among these is usually some type of the Staphylococcus albus. This latter organism is normally present on the skin, and is usually to be found in the anterior part of the nose. The aim of this treatment is to exploit the activities of such normal organisms to the detriment of the obnoxious bacillus of Perez. The activities of the bacillus of Perez are largely directed towards the destruction of protein bodies; whilst the staphylococci have a well developed capacity for the fermentation of sugars. This is one factor on which the method of treatment is based. The other factor lies in the necessity for tryptic ferments to act on a protein substrate and in a slightly alkaline medium.

Experiments show that when the bacillus of Perez is grown in broth together with the staphylococcus it readily maintains its position in the culture; but that if glucose is present in the fluid medium the carbohydrate is fermented by the cocci with the production of acidity, and the bacillus is very rapidly killed out. By altering the substrate in composition and reaction we thus get an inhibition of those processes of fermentation which produce the factor; and a replacement of the obnoxious proteolytic Perez's bacillus by the glycophilic bacteria, the products of whose carbohydrate fermentation are at least not obnoxious. Glycerine is also readily fermented by most types of staphylococci. Its use in the treatment of another not very dissimilar condition, i.e., bromidrosis, has already been published by one of us.¹

Although the treatment of ozaena by this method, which has yielded successful results, was founded on the theoretical and experimental concept outlined above, which is readily demonstrable in vitro, we have nevertheless been unable to demonstrate an alteration in reaction of the nasal secretions in our cases under treatment. In one or two instances in which there was a nearly pure culture of Perez's bacillus in the nose we have implanted the Staphylococcus albus in conjunction with the carbohydrate medium.

**PART II.—CLINICAL.**

**By C. H. Hayton, F.R.C.S.Ed.**

The object in reviving the discussion of the treatment of atrophic rhinitis with ozaena is to present the outline of a simple and effective method based upon a study of the character of the bacterial flora

inhabiting the nose in this disease. As a result, the most distressing features of the complaint, the foetor and the headache, quickly disappear, the disagreeable crusts are rapidly diminished, and improvement is noted in the conditions of the mucous membranes and the general health of the patient.

We do not claim that this treatment will restore old standing structural changes in the tissues which have taken place during the course of the disease.

**General Conditions of Methods of Treatment.**

Heretofore the majority of the investigators of this disease, Perez, Ael, Hofer, Kofler, McKenzie and others, have endeavoured to isolate a specific organism or a group of organisms, and to prepare a vaccine therefrom. Another group of investigators, believing the cause to be due to pus-producing organisms or a group of such, have worked along lines of surgical cleanliness and drainage, trying to compound a satisfactory disinfectant to be applied locally in the form of a nasal douche, powders or paints. In the method initiated by my colleague, who has already published an article on this subject,¹ the aim has been to substitute a carbohydrate medium in order that the infecting proteolytic bacteria may be replaced by the harmless glycolytic bacteria found in the nose, the theory being that these latter bacteria by the fermentation of the carbohydrate bodies create an environment in which the activities of the Perez bacilli are inhibited and the bacteria themselves finally destroyed.

**The Method Employed.**

The substances used thus far in this method of treatment are glycerine and liquid glucose. These substances are applied copiously to the mucous membrane of the nose in a mixture consisting of pure glycerine with the addition of 25 per cent. liquid glucose. Several applications a day are made with a cotton-wool applicator, the whole of the nasal cavity being gone over and the crusts rubbed off as far as possible. This is done in the first instance by the surgeon, and subsequently by the patient at home. At each future sitting the nose is carefully examined and further applications made to the neglected parts. From time to time the flora of the nose is examined to note the cultural properties of the bacteria present, and to determine

whether or not they are the strain to ferment the media supplied. Glycerine, though less satisfactory from a theoretical point of view since it is less easily broken up by bacteria, has some advantages over glucose on account of its physical properties. In some cases where a spray or irrigation is required, a mixture of the two substances has been used in a 10 per cent. aqueous solution.

**The Success of the Treatment.**

The whole secret of the success in this method of treatment, and I wish especially to emphasize this point, lies in the fact that the solution must be applied thoroughly to every part of the mucous membrane infected at least four or five times daily to begin with. As improvement is noted it will not be necessary to paint so often. The application is a simple matter in early cases, but sometimes most difficult in severe cases, in which the crust formation goes on in the nasopharyngeal regions, especially the roof and the Eustachian cushions, or in the middle meatus around the bulla ethmoidalis, and the uncinate process, or in the olfactory sulcus, and in the posterior parts of the inferior meatus. To get over this difficulty it is well gently to irrigate the nose at first, to flood these parts with solution, and later, besides the wool application, to use a coarse spray. In a case of thirty years' standing this was found to be most effective.

**Glycerine and Sugar.**

Glycerine and sugar have been used in the treatment of ozena in times past—glycerine, with one exception, as the excipient of a disinfectant, and sugar in the one instance mentioned in a simple syrup and powder.

In the *British Journal of Laryngology,*¹ the author recommends the application of glycerine borax by a nasal spray and recounts successful treatment in a number of cases. Glycerine is the base of the well known Mendl's paint still used in ozena. The glycerides of tannin and ac. carb. in weak solutions have also been used. In 1913 Abbott² recorded the application of 5 per cent. solution of reniform in glycerine as successful. Logan Turner has used pure glycerine previous to douching the nose. Harry,³ in 1915, used simple syrup and powdered

³ *Prescriber,* 1914-15.
sugar in the nose, and reports successful treatment. It will be noted, however, in the cases of glycerine that the disinfectants used were of a mild order. In the opinion of the writers the success obtained in these cases was not due to the disinfectants but rather to the glycerine and sugar and also to the absence of repeated douching.

**Douching the Nasal Cavities in Ozëna.**

The constant and continual douching of the nose with aqueous solutions in ozëna cases is a practice not to be encouraged. While it may temporarily relieve the nose of crusts it exposes the whole upper respiratory tract to numerous complications: *First,* it tends to spread the infection to other parts of the nose and throat. One patient declares positively that the crusts were confined to the left nostril until she began to douche and then they soon appeared in the right and in the throat. *Secondly,* it washes away the harmless flora and the normal protective secretions, and exposes the bare mucous membrane to other infections and to catarrhal conditions. In this method of treatment little douching is required. The harmless organisms in the nose are encouraged and the protective secretions are strengthened with the result that the abnormal organisms are finally extirpated. The investigations as to the ultimate value of this form of treatment are as yet in their early stages, and we hope in due course to make a further report, but enough has been done to create the belief that this method of treatment is more simple and effective than the disinfectant douching method.

**The Results of Glycophilic Treatment in Ten Cases.**

The histories of the ten cases, extending over a period of two years, show that none were syphilitic, none were tubercular, but four gave a history of tuberculosis in the family. In two cases two sisters were similarly affected. After careful examination with one exception all showed the sinuses and antra free from infection. Four had complete loss of smell, and all but one were affected with frontal headaches. The factor of all the cases disappeared and the greenish crusts were replaced by mucoid secretions. At a recent examination of the cases no typical crusts were seen, the mucous membrane appeared healthy, but the treatment has not as yet affected the anosmia or the atrophic conditions. All the patients are being advised to continue the applications once a day for the time being. The patients were all females, and the ages ranged from 15 to 68.
DISCUSSION.

Mr. R. A. Worthington: The authors have alluded to the important fact that there is a normal bacterial flora of the nose. For many years I have accepted the statement that investigations showed the nasal cavity to be normally sterile, but this statement now needs re-consideration. May I suggest that the Section might with advantage appoint a committee to report authoritatively as to whether there are or are not micro-organisms in the normal healthy nose, and if there are, what they are and where they are, whether they are in the fossae alone, or whether they may be in the sinuses without being regarded as indicating an abnormal or unhealthy condition. Until this point is decided the further investigation and progress of nasal surgery is liable to be delayed, for we do not know where we stand in this matter.

Dr. W. Hill: Mr. Hayton said that the sinuses in atrophic rhinitis are free from infection. Does he mean free from the ordinary infections of the sinuses, or from those specific infections which are presumed to be associated with atrophic rhinitis of the ozaenic form? Are the sinuses free in the majority of cases from the same lesion we find in the nose? Robertson many years ago claimed to have cured a very large number of cases of atrophic rhinitis by opening the antra, and in view of the article, which appeared in the Journal of Laryngology, I opened a few antra in cases of foetid atrophic rhinitis until I was tired of the procedure. In no case in which there was well-marked atrophic rhinitis did I find crusts in the antra, although Robertson said that on opening the antrum, where he found the disease, it was evidenced by crusts. I have not been encouraged to embark upon another set of experiments to find whether that experience of mine was highly exceptional, but I presume that my experience was such as most here will endorse. Yet Robertson reported a series of cures, and his results were never disputed at the time or subsequently, not even by myself, although, of course, I ought to have recorded my experiences. Why the mucosa of the accessory sinuses escapes—that is, if it does escape—in this form of ozaena is to me a matter of very great surprise, and I shall not be surprised to find that we have been half wrong, and that the nasal labyrinth is infected but not in crust form; but, if infected, how is it that keeping the nose clean so relieves matters considering that this form of treatment, or any form of treatment, in ozaena never gets at the accessory cavities? My experience is that the nasal sinuses do appear to escape in this particular disease of the nasal cavities proper, but why it should be so I do not know.

Mr. Herbert Tilley: Dr. Hill’s experience is not exceptional. Dr. Grünwald, of Munich, was one of the first to suggest that in the majority of the cases of ozaena the cause was the suppuration of the accessory sinuses. For some two or three years I gave this matter very careful attention by examining, from this point of view, all my cases of atrophic rhinitis. The first conclusion I came to was that atrophic rhinitis was not due to sinus
suppuration. I suppose the disease is really explained, in the light of the paper which Dr. Benians read to us this morning, by the fact that the infection is a very superficial one in the mucous membrane—that it does not penetrate deeply, and does not spread like some other types of infection. A few years ago someone—I think it was Gottstein—suggested that glycerine would be one of the best methods of treating atrophic rhinitis, and the method I followed was to take a long slip of gauze, soak it in glycerine, and pack it into the nose. This was done two or three times a week, the intervals being lengthened as time went on and the condition improved. I can only say that in the worst cases I do not think any treatment up to the present moment is more satisfactory than packing the nose over night in this way, and taking out the packing next morning.

Sir W. MILLIGAN: This is a most valuable contribution to the aetiology and treatment of atrophic rhinitis, and it shows the value of scientific investigation of the disease. All members of the Section will be particularly grateful for any hint upon the treatment of this very troublesome condition. I have used glycerine for years in the treatment of atrophic rhinitis, but I never had the slightest idea why it was more effective than some other methods of treatment. Now daylight is dawning, and this paper is certainly an eye-opener! We have all used glycerine more or less in combination with other drugs, and I have used glycerine—just the ordinary commercial glycerine—merely with the idea that it was a good emollient. The scientific explanation as to why it should be used is very interesting. I remember Dr. Robertson's work very well, and at that time I operated upon a very considerable number of maxillary antra. On opening the antrum I never found one that was diseased. The only advantage was that by opening the antrum one got a good opportunity of flushing the nasal cavities. I am not sure that the same can be said of the ethmoidal labyrinth. In some of these cases of ozäna I have felt that there was disease in the ethmoidal labyrinth. The ethmoidal labyrinth is, in many cases of young anaemic girls, extremely attenuated, and it is quite conceivable that what commences as a superficial implantation on the mucous membrane of the nose may ultimately involve the bony labyrinth. Therefore I would not altogether condemn the idea that there may be some focal infection in atrophic rhinitis. Will the authors of this paper give exact details of the glucose solution, and have they ever tried spraying thin honey into the nose in the treatment of this disease?

Dr. W. S. SYME: I am sorry to find myself in opposition to the last speakers, but as I think it quite possible that my paper will not be reached, perhaps you will allow me to read an extract from it now bearing on this subject. [Dr. Syme here read from his paper, which was read in extenso the following day. See p. 225] I think, as a matter of fact, that the whole question rests on the stage of the disease of the atrophic rhinitis. As far as my experience goes, I am always very ready to operate on an antrum in atrophic rhinitis in early life, and I find that the result on the atrophic condition is good. What happens
later on may be a different matter, but certainly in atrophic rhinitis in early life antrum disease is a common condition, and the changes are those that obtain in ordinary antrum disease, hypertrophic to begin with, and, I should say, atrophic later on.

Dr. P. Watson-Williams: We are not all agreed as to what is a case of ozena. To my mind it is not a term interchangeable with atrophic rhinitis. I, too, have been interested in this question of sinus infections as a causal factor, and, like others, have been disappointed in the anticipations I was led to make. Dr. Symes's are cases of maxillary disease, I think, and therefore if he came across cases in which there was atrophic rhinitis it is not a matter for surprise; cases of pure ozena are a different question.

Mr. Benians (in reply): With regard to the "normal" bacteria of the nose I used the term "so-called normal," meaning to indicate those bacteria, notably staphylococci and diphtheroid types of bacilli, which are fairly constantly found both on the skin and mucous surfaces of the body. I think it would be, as Mr. Worthington says, a good thing if the question of the normal bacteria of these mucous surfaces could be settled definitely. Another thing we want to know more about is the action of the nasal secretions on the growth of various bacteria. As regards the use of honey in treatment we have no experience but, as a mixture of fermentable carbohydrates, it would presumably be quite effective. We have been guided in our treatment by two factors, how far the substance is readily split up by the bacteria, in the first place, and, secondly, how readily the medium will mix with the mucoid secretions of the nose. Glycerine is an extremely good emollient. Glucose is a substance readily broken down by almost all bacteria. In experimental work in the test tube I have found that, in the presence of a fermentable carbohydrate, the glycophilic bacteria gain the ascendency, and lead very quickly to the extermination of such bacteria of proteophilic type as are present.

Mr. C. H. Hayton (in reply): I examined these antra and sinuses as we ordinarily do in cases of infection, and they gave no signs of infection; in one case I punctured these cavities, and they were not infected in the common sense of the term. With regard to infection in the ethmoidal region, infection does seem to travel to the ethmoidal region, and the difficulty in those cases is to get the media in contact with the growth in that region by mechanical application. We have had to spray the nose carefully in order to get the media in contact with all the parts of the mucous membrane affected. We have simply relied upon the application of these different media, which we find very effective.
Spasm at the Entrance to the Oesophagus.

By A. Brown Kelly, M.D.

A variety of dysphagia is not infrequently met with in middle-aged women which may have been present for years, is referred to the level of the larynx, and is so pronounced that even a small fragment of food becomes impacted and causes complete occlusion. As a rule, this affection is diagnosed as spasm of the upper end of the gullet and, without endoscopic examination, is treated and benefited by the passage of a bougie; the subjects are considered to be neurotic.

It is doubtful whether our conception of this disease is correct. In view of the steady persistence of the dysphagia for years, the immediate and striking benefit following the use of a bougie, and the fact that a membranous diaphragm has been met with at the entrance to the oesophagus in a few cases, are we justified without an inspection of the parts in excluding the possible presence of an organic stricture—e.g., resulting from the union of adjacent surfaces? Further, are the patients neurotic or have they become nervous solely in connexion with deglutition? Hysterical dysphagia differs from the affection under consideration and is not taken into account.

In order to determine the local condition at fault an examination was made under chloroform of the entrance to the oesophagus in a number of patients suffering from the above-mentioned symptoms in pronounced form. The obstruction was found, in accordance with the generally accepted view, to be caused by strong spasmodic closure at the entrance to the gullet. The majority of the patients, on the other hand, were not at all neurotic.

The causes of the affection will be discussed.

A Clinical Type of Dysphagia.

By D. R. Paterson, M.D.

Dysphagia in women frequently associated with a condition of the mouth cavity due to atrophic changes in the mucosa and aggravating the difficulty in swallowing. Changes also seen in the lower pharynx and of importance in direct examination. Condition accompanied by marked anaemia and loss of flesh.
DISCUSSION.

Dr. F. de Havillard Hall: There is a cause of spasm of the oesophagus which I have not seen mentioned, but which I noted some twenty years ago in the case of two doctors who consulted me. They were both habituated to morphia to a great extent, and on giving up the drug they were quite unable to swallow solid food. In both cases a large-sized bougie passed quite easily into the stomach, but with no alleviation. One of the patients passed out of my knowledge without my being able to learn anything of his subsequent history, but the other gradually improved and was able to take a fair amount of food. He lived on milk for over a year, the milk being gradually thickened. Unfortunately, he returned to his morphia, and on relinquishing it he again found inability to swallow. Eventually he made a perfect recovery, but as I have been unable to find his name in the Medical Directory for the past two years, I assume he is now dead. The cessation of the habit by the withdrawal of the accustomed stimulant much reduced the nervous system, and rendered the men liable to spasm.

Sir W. Milligan: The moral of these two interesting communications is that in all cases of middle-aged people who suffer from dysphagia a direct examination of the oesophagus should be made. The number of women who suffer from dysphagia, when the disease is just at the commencement of the oesophagus, is remarkable, and I think that my statistics correspond to those of Dr. Kelly: 70 to 75 per cent. of the women suffering from disease of the food passage have it in that particular situation. The blind bougie method should be put aside; the examination should be direct. It has always seemed to me that possibly trauma is a very considerable aetiological factor. From the number of times in which it is possible to get some little erosion of the oesophagus owing to swallowing something very hard, such as a spicule of bone, it is not to be wondered at that these disturbances are not uncommon. I look upon it in much the same way as I do fissure of the rectum or anus. Trauma is an aetiological factor which we should not dismiss too lightly. Then again, how many of those cases of spasmodic dysphagia, if followed out over years, have been found to develop malignancy? I can only recall one case in which malignancy developed. One loses sight of these cases, of course, but I do remember one case, a woman, aged about 45, who had this particular type of spasmodic dysphagia, and the only thing to be seen was just an annular tumefaction at the commencement of the oesophagus. It was not infiltrated, nor even hard, but that woman, two years afterwards, did develop a definite malignant growth in the lower end of the hypopharynx. For that reason only one ought to make a direct examination. The dysphagia may be a passing phase or it may be the commencement of organic disease, and it is well to know what it is at the earliest possible moment.

Dr. W. Hill: Though the titles of these two thoughtful papers are far from identical, yet the authors traverse very much the same anatomical area. They both include obstruction at or near the upper end of the gullet.
Dr. Kelly definitely calls his paper "Spasm at the Entrance to the Oesophagus," but it was evident to everyone that he was dealing very little with spasm of the oesophagus itself, but almost wholly with obstruction of an indefinite pathology at the lower end of the pharynx. Dr. Paterson was fairly definite in localizing the obstruction producing dysphagia to the neighbourhood of the inferior constrictor. I have an idea; often expressed here, that members of this Section, especially those who are not very conversant with oesophageal disease, are obsessed with the idea of primary spasm as an explanation of dysphagia in certain cases. On the other hand, I have the reputation of being "spasm-blind." I am extremely familiar with the spasm in the lower pharynx especially, and also in the gullet, as a secondary symptom, but primary spasm I have looked for in vain, except on one occasion. I am not unacquainted with the question of dysphagia behind the cricoid, say in the lower pharynx, but I do not know that I have ever seen a case in which I was able to satisfy myself that it was purely a primary spasm. I am very familiar with the class of cases which to Dr. Paterson has alluded—e.g., the case of a woman with stomatitis—and Dr. Kelly has alluded to something very similar. In my own cases, parallel to those alluded to by the readers of the papers, the first thing I do with a woman who cannot swallow, and in which there appears to be obstruction in this area, is to ask her to swallow before me, giving her a biscuit or something of the kind. Instead of showing the typical phenomena of spasm, she almost invariably makes no effort to swallow. The last act of pharyngeal swallowing and the first act of oesophageal swallowing consists in pulling the larynx upwards and forwards by the muscles connecting it with the terminal hyoid bone and tongue. Any man in this room can, if he likes, determine whether the patient does move the larynx forward. If there is any spasm the effort will be evident, the muscles will be seen to contract, and if the larynx does not come forward then it is due either to spasmodic or organic stricture. In the class of cases discussed here in which there is no stricture to speak of, I have no doubt that if members will only study this question they will see that this effort to pull the larynx forwards and upwards away from the vertebral column does not take place. We have inertia—i.e., paresis. We are all agreed that there are a certain number of cases which may be properly called functional or neurotic, but they show paresis rather than spasm. Bougieing is a very useful treatment in these cases whether we do not find stomatitis or whether we do. It stimulates; it is a method of massage. In a certain number of cases, however, we do get actual tumefaction, and evident narrowing. I think we should separate those cases which show definite narrowing owing to slight inflammatory and other organic causes from the smaller group of those which are unequivocally functional and of the nature of paretic rather than spasmodic neuroses.

Dr. Dan McKenzie: Dr. Hill has been accustomed to give us definite facts and opinions on this point, but now they have become rather less definite, until now I believe, in his heart of hearts, he thinks that spasmodic stricture does, after all, exist. I suggest that what he means by primary spasm is a
spasm induced by the higher nerve centres, while a secondary spasm is one that is induced by the local centres.—[Dr. Hill: No.]—To come to practical matters, in the cases I have reported there was no obvious local lesion, no tumefaction, and yet there was definite stricture visible on direct inspection. One saw that the calibre of the gullet was narrowed down to an opening 3 to 5 mm., having an appearance which could be easily overlooked. The two patients had both been suffering from the effects of this spasm for very many years. One of them had never been able to finish a meal in her life. She was ashamed to eat out of her own house, as some of the food stuck at every meal and had to be regurgitated. I saw this spasm with the oesophagoscope; it was also seen to occur by X-ray examination, yet the moment I passed the bougie it disappeared, and the patient has been all right ever since. If that was not a functional spasm I do not know what it was. I had two cases within a short time, under almost precisely the same conditions, and both showed the same good effect of treatment.

Mr. R. A. Worthington: The statement that all cases of dysphagia should have an endoscope passed is perhaps going rather too far, but every case of dysphagia in which it is thought necessary to pass a bougie should have an endoscope passed first. I would make an absolute rule against blind bougieing until an endoscope has been passed. I would not have any particular regard for the age of the patient. I remember a few years ago being consulted by a young lady aged 27, suffering from symptoms of dysphagia, and I regarded it as probably a functional condition, but I said that I would advise examination if under treatment she did not improve in two or three weeks. She did not improve, and as is often the way in those cases she went up to London, which was quite unnecessary! and there, I learned afterwards, she had been oesophagoscoped and that an epithelioma had been found. That in a young woman aged 27! So I thought after that that I would not be influenced by considerations of age. One of the symptoms which I have always regarded as of extreme value as making it practically certain that there is a real obstruction in the oesophagus, and that it is not a purely nervous dysphagia, is the presence of froth in the throat. Where there is any froth—that is, unswallowed saliva—I always advise an examination. Many of these cases are what I still venture to call spasmodic, and in spite of much instruction on the subject from my friend, Dr. Hill, I had one such case, not very long ago, of a woman who had had dysphagia for about five years, and she was of the kind who perhaps would be regarded as typical, a rather thin, nervous woman, aged 36 or so. The presence of froth made me diagnose a definite and more or less constant block. I oesophagoscoped her, and found that the oesophagus contained curds of milk. The estimation of dilatation of the oesophagus is often exceedingly difficult unless it is a very marked condition. I passed the bougie in this case through the endoscope, and at the cardia I had some resistance, which suddenly gave, and then the bougie—a full-sized one—passed easily. I could see nothing abnormal around the wall. That I regard as a ease of spasm. If the whole of the oesophagus had been in the condition that
Dr. Hill describes as paresis, then I should not have had the feeling of resistance to the bougie at the lower end. The woman has completely recovered her powers of swallowing after that single examination.

Dr. Paterson (in reply): I suggest that we should examine a series of cases where the difficulty is a paretic one. The only one I have had an opportunity of examining was a case of myasthenia gravis, and there the tube went in easily.

An Operation for the Complete Removal of the Soft Palate (Staphylectomy).

By Dan McKenzie, M.D.

It is claimed for the procedure about to be described that by a combination of the use of diathermy and the cold snare the entire soft palate can be removed with little or no loss of blood.

Indication: Malignant disease (epithelioma or sarcoma) of the soft palate, especially when it occurs on the posterior aspect where the new growth cannot easily be excised as an island of tissue.

Contra-indications: If the disease has spread so as to involve the bone of the palate the removal of the soft palate alone would be an incomplete operation.

By using diathermy in making the section it is possible to cut through the tissue of the growth without any risk of disseminating living cancer cells as the tissues in contact with the diathermy terminal are destroyed.

Operation: The anaesthetic may be given by the mouth as haemorrhage is minimal.

Technique: (1) By means of a diathermy knife terminal a transverse incision is made ½ in. behind the posterior edge of the bony palate and is carried right through the soft palate to its posterior surface. The incision extends from the level of the alveolar process on one side to the same level on the other side.

(2) Using the diathermy knife again two incisions are carried, one on either side of the base of the uvula, from the highest point of the arch of the velum palati to join the first incision, and the middle of the soft palate, including the uvula, thus isolated is removed.

(3) The lateral portions of the soft palate left are removed by means of Lernoyez’ tonsil snare applied close to the lateral wall of the palate (the cold snare is here employed, being gradually tightened) in preference to diathermy, in order to minimize the risk of secondary
haemorrhage from the arteries entering the palate at this place which might follow the separation of diathermy sloughs.

(4) Step No. 3 opens up the upper part of the tonsil fossa, and these glands can then be quickly and easily removed, thus completing the operation by the removal of the glands which drain the soft palate and which otherwise are liable to become the seat of subsequent recurrences (Sir StClair Thomson—verbal communication).

The gap in the pharyngeal roof formed by the operation can be easily closed with an obturator and nasal speech prevented. (See figures.)

Mr. W. G. Howarth: For what type of growth is this set operation designed? It certainly would not be useful for a growth in the middle line, and the disadvantage of any set operation is that it is not applicable to all growths. I have not found the after-bleeding which Dr. McKenzie thinks likely to occur when one uses the diathermy knife on the lateral wall of the pharynx, and one need not of necessity be very anxious about secondary haemorrhage three or four days later. My experience is only a small one, however, of five or six cases, and definite conclusions can scarcely be drawn from these.
Section of Laryngology.

President—Dr. James Donelan.

SUMMER CONGRESS, May 2-3, 1919.

(May 3.)

Brigadier-General H. S. Birkett, C.B., C.A.M.C., Honorary President, in the Chair.

Gunshot Wounds of the Nasal Accessory Sinuses.

By John F. O'Malley, F.R.C.S.

Number of cases, 59. Sinuses involved: Antra, ethmoids, frontals, sphenoids. Missile: Bullet, shrapnel. Site of lesion: (1) Passed through sinus, side of entry and exit; (2) retained. In sinus, soft tissues near sinus or elsewhere.

Extent of injury to: (1) Soft tissues; (2) sinus; (3) nose; (4) mouth; (5) orbit; (6) eyeball. Eye: loss or injury.

Sepsis.—I: Arising from passage of missile through tissues or sinus. II: Associated with retention of missile.

Examination: (A) X-ray—(1) bone injury; (2) appearance of sinus (cloudy, opaque, clear). (B) Transillumination. (C) Puncture of antrum.

Treatment: (1) Nil; (2) simple dressing of wound; (3) operation for (a) removal of foreign body (sinus tissues); (b) sepsis (wound sinus); (c) nasal adhesions; (d) sinus into mouth; (e) lachrymal sac suppuration.

Results: Condition of sinus found on removing foreign body (healthy, septic, polypi).

Conclusions.
DISCUSSION.

Mr. W. G. Howarth: I had a considerable number of these cases during the war, and, although one is only able to see them quite late in their development, I found that much the best way to treat the frontal sinuses was by external operation and the establishment of free drainage as possible. The experience I have had with a good many of these through-and-through wounds in which the antrum or frontal sinus is involved, is that sometimes, after the case has apparently cleared up, a recrudescence of symptoms occurs and another sinus is formed. It is not uncommon to have to operate on cases two or three times for residual or recurrent symptoms, but, on the whole, the cases do extremely well with free drainage, and preferably some form of external operation.

Brigadier-General H. S. Birkett, C.B. (Hon. President): So far as our experience in France goes, working at a base hospital, most of the cases were passed on immediately out of our sphere of operations. Therefore I cannot speak definitely on this point at all.

Dr. D. R. Paterson: We had a case of gunshot wound of the upper jaw, which came into one of the sections of our hospital, and the patient started violent bleeding from the nose and throat; thereupon the surgeon tied the common carotid on that side, and the hemorrhage stopped. Ten days afterwards it recurred, and the carotid on the other side was tied, it ceased for a few days and recommenced, and the man came under my care. Finding that the bleeding was from the antrum, I tried at first to stop it by making a large opening through the inner wall of the cavity and packing it. Then I ascertained that it was not possible to control it properly, so I opened the antrum from the cheek, and found the bleeding point to be the terminal branch of the artery on the internal maxillary posterior wall. I plugged it for ten days, but every time the plug was changed bleeding restarted, so eventually I cut down on the external carotid, found some pulsation in it, and the whole trouble came to an end.

Captain E. F. Risdon, C.A.M.C.: From the point of view of controlling the hemorrhage, we have found the wide opening up of the wound and leaving it wide open a useful procedure. Latterly we opened the wound in these cases widely, without putting a ligature on, and there was no bleeding afterwards. We had many interesting cases, but we frequently found that the condition of the antrum did not clear up. After operating on a case we did not irrigate, and the discontinuance of irrigation was effective. In one interesting case in which the discharge continued for some time, the X-ray photograph showed both antra to be very similar, but when opening them up I removed a piece of wood, $\frac{1}{2}$ in. long, $\frac{1}{2}$ in. wide, and $\frac{1}{4}$ in. thick, which had been there for some months.

Dr. Dundas Grant: My experience with a comparatively small number of military cases is that the healing in the antrum is extremely good as compared
with the cases one sees in civil life. Sometimes a very simple drainage through the nose is enough to bring about recovery. The frontal sinuses are much less easy to deal with, and I have had to chip away the whole of the anterior wall of the frontal sinus in order to produce collapse and obliteration of the cavity.

Mr. J. F. O'Malley (in reply): Mr. Howarth mentioned the fact of recrudescence in frontal sinus cases. I have had so very few of these that I cannot speak with authority, but I think the frontal sinus the most difficult to deal with, because of the difficulty of establishing satisfactory drainage; and the antrum cases are the least difficult. Dr. Paterson mentioned a rather interesting case of bleeding. I may rather emphasize the importance of having a special surgeon to deal with cases in situations with which he is more familiar than the ordinary medical man can possibly be. I recall a case which came in with a history of bleeding for several weeks. It was that of a Belgian soldier; the bullet passed through the vertex and traversed the sphenoid. The bleeding was coming from the nose, and the patient's wound was packed in a more or less blind fashion, and the bleeding used to stop temporarily. When I examined him I could see a bleeding point at the posterior nares, where the bullet had escaped near the base of the skull; and by the introduction of a post-nasal plug for twenty-four hours the trouble was overcome. The man became perfectly well, except that the sight of one eye had been lost, and within a few weeks he left the hospital. I am very much opposed to irrigation of the antra. In gunshot wounds I never irrigate the antra. The procedure destroys the epithelial protective cells, and in cases of an open granulating wound the newly formed granulation tissues absorb the liquid quickly and the cells burst and are discharged as pus; if left alone to carry on their function, healing takes place very much more rapidly.

The Treatment of Enlarged or Diseased Tonsils in Cases where Surgical Procedures are Contra-indicated.

By Irwin Moore, M.B.

The following are frequently considered as contra-indications to operative procedures:

Local.

(1) Acute infective condition of the tonsil—e.g., streptococcal infection, scarlet fever, measles, Vincent's angina, &c.

(2) Physiological enlargement of a temporary character accompanying the four periods of molar eruption.
GENERAL.

(1) Cardio-vascular changes: (a) Cardiac disease, e.g., endocarditis, pericarditis; (b) constitutional diseases where anæmia is very marked, e.g., chlorosis, pernicious anæmia, Hodgkin’s disease, &c.; (c) low coagulating powers of the blood, e.g., in true haemophilia, purpuric diseases, &c.; (d) blood-pressure over 225 systolic; (e) arteriosclerosis.

(2) Advanced tuberculosis.

(3) Syphilis.

(4) Kidney diseases, e.g., Bright’s disease.

(5) Diabetes mellitus.

(6) Grave nerve or mental disease.

(7) The “status lymphaticus.”

(8) Professional voice-users.

THE CONSIDERATION OF NON-SURGICAL METHODS OF REDUCTION OR DESTRUCTION OF THE ENLARGED OR DISEASED TONSIL.

(A) Chemical:

(1) Absorbents—e.g., iodine—may be of use in the earlier periods of hypertrophy, but are useless in the ordinary run of cases.

(2) Astringents—e.g., tannic acid, zinc sulphate, &c. Their action is limited simply to the reduction of the inflammatory process and have no influence on the hypertrophy.

(3) Caustics or escharotics: (a) Mineral astringents—e.g., silver nitrate, zinc chloride, are mildly caustic, their action is too superficial and too slow; (b) mineral acids—e.g., chromic, trichloracetic acids, &c., cause marked inflammatory reaction, and act only superficially and slowly; (c) alkaline caustics—e.g. (i) caustic potash causes inflammatory reaction and there is difficulty in localizing its action: (ii), caustic soda combined with slaked lime, first introduced by Morell Mackenzie in 1864; it possesses many advantages over the potash salt. It causes no inflammatory reaction. It penetrates rather than spreads circumferentially, therefore its application is more localized. Its action is less severe and continues for a longer time. There is no pain if cocaine is applied to the tonsil before using the escharotic.

The results obtained by the author with the caustic soda and slaked lime, in cases ranging from childhood to advanced age, have been of the most striking character. The largest tonsils have been
reduced to normal size, whilst in the case of diseased tonsils there has been no blocking or sealing up of septic crypts, as has occasionally been observed after the galvano-cautery. The author recommends this treatment, long overlooked and discarded, as a useful substitute, and as the treatment of choice, in certain cases, in which radical operation is contra-indicated.

(B) Electrical.

(1) Electrolysis has been employed in the past, but the results were not worth the trouble of the performance.

(2) Galvano-cautery or puncture: This was a method in great favour twenty years ago in those cases in which the removal of the tonsils was contra-indicated, e.g., in adults, or where haemorrhage was likely to occur. If properly used it may be employed with great success in many cases.

(3) Diathermic puncture: The tonsils may be destroyed by this method, and it has been employed with satisfactory results in a number of cases.

DISCUSSION.

Dr. J. DONELAN: Dr. Moore’s review of the situation with regard to operations on enlarged tonsils is important. Yesterday Sir StClair Thomson made a remark in which he seemed to suggest that I was rather against the operation in my paper. I was merely emphasizing the desirability, in cases in which there is a good deal of lymphoid enlargement and not very much fibrous tissue, of getting over this physiological swelling by milder means than operation, and not applying operative methods in every case which occurs. Dr. Moore made certain remarks with regard to Morell Mackenzie’s caustic paste. As I had four years’ close experience of his (Mackenzie’s) methods, I can say that he never used caustic paste during that time, having given it up in favour of the electro-cautery. The electro-cautery, in my experience, is very satisfactory. I frequently use it for suppuring follicles in tonsils, especially cases of singers. In such patients it is peculiarly suitable because it disturbs the pharyngeal anatomy very little and it is not necessary for such singers to undergo their training afresh, as often happens after tonsillectomy. Many singers have become accustomed to faulty throats with large tonsils, and these ought not to be interfered with in a radical manner.

Dr. A. J. HUTCHISON: Will Dr. Moore give us an exact description as to how he applies the paste? Does he have to hold the paste on for a certain length of time?

Dr. DUNDAS GRANT: How frequently should the application of London paste be made? It is peculiarly interesting that the subject should be brought up just now, especially in connexion with Dr. Donelan’s relation of his experi-
ences with Sir Morell Mackenzie, who gave such a wonderful impetus to laryngology in this country, and whose great talent and industry is worthy of some memorial. There is one form of operation on the tonsils which never seems to have had its proper chance in this country. With some trouble I have acquired the art of completely enucleating tonsils in their capsule by means of the guillotine, but I consider the ideal operation is to remove all the interior of the tonsil and leave the capsule, and it would be well if those who are progressive would keep that in mind. One of our American friends calls it the intracapsular operation, and it is done by means of punch forceps. Our energy and our skill in extracting the whole capsule, and the pride with which we turn it out and display the whole capsule, are doubtless laudable, but nevertheless indicate excess of zeal. With the employment of the intracapsular operation, the objections to the removal of diseased tonsils would very largely disappear. Reference has been made to the use of iodine. This will not reduce tonsils, but it is an excellent antiseptic. It acts better if acetic ether is added to the iodine solution. I should like to hear something about the lymphatic gland extract.

Mr. J. F. O'Malley: Will Dr. Irwin Moore state the extent to which the cautery is actually pushed? As I regard the tonsils, they come under two main types: (1) Tonsils which are seen to be very superficial in their attachments on depressing the tongue. The chief inconvenience of these tonsils is that they are an obstruction to articulation. I had some difficulty in coming to the conclusion that I could deal with a tonsil of that type by galvanopuncture unless I was able to reduce the tonsil, to get rid of all its crypts, and leave nothing but the actual attachment to the capsule. (2) In the other type of tonsil, which is much more serious, the chief part of the lymphatic tissue lies below the free edges of the pillars; here the ordinary apparatus for draining the tonsil squeezes over the orifices of the crypt, and prevents it from being drained. This latter type of tonsil is the one from which often systemic infection arises. This is also the type in which one gets a tendency to recurring abscesses. I scarcely see how I could get to the bottom of that tonsil with a cautery. Of course I realize that Dr. Moore is only dealing with cases in which an ordinary surgical operation would be contra-indicated.

Dr. W. Hill: Sir Morell Mackenzie, who was always optimistic, but very unreliable in his statistics, published a glowing account of this paste method; many may have thought that he used it on occasion to the last, and yet if it had not been for Dr. Donelan we should never have known that he not only abandoned it but came to think very poorly of it. With regard to Dr. Moore's experiences, in how many cases has he used the paste method and in how many cases was the result satisfactory? If his own tonsils were enlarged, would he prefer paste to surgical methods? A nine-tenths or four-fifths removal—a partial tonsillectomy—is probably quite sufficient in these cases as a start, where bleeding is feared. In singers there
are many who hold we should be very chary of complete enucleation of the tonsils. The contralto voice and the contralto production seem very largely to depend upon the configuration and muscular action of the fauces, which often include large tonsils. Are we going to tell such a singer to have the paste applied? Can we say with increased experience, as Dr. Moore seems to say to-day, that the mouths of the crypts are not likely to be harmfully sealed up after paste application? I am not prepared either to assert or deny it, because I confess I have never used the paste. I have used the galvano-cautery and have been very much disappointed. At the same time, there are patients upon whom I should be very glad to avoid operating on the tonsils if I could treat them efficiently by other means.

Mr. Rose: I have had a limited experience in the treatment of enlarged tonsils with the electro-cautery. I found the treatment painful to the patient, and the results not particularly satisfactory. I therefore welcome Dr. Irwin Moore's suggestion that this paste may be preferable to the electro-cautery. But in applying any of these non-operative methods the question always arises as to what is going to happen to the deeper part. It seems inevitable that if one only attacks the upper part of the tube, when healing occurs the lower part must be turned into a closed cavity. It may be possible to avoid that, and if so, we shall all be glad. In many cases there is a large piece of tonsillar tissue lying within the palate. Is Dr. Moore able to get at that and deal with it, without injuring the mucous membrane or the muscle of the palate?

Dr. Kelson: Dr. Moore has done well in bringing forward this method. When enucleation came into fashion I was somewhat carried away by it, but have come to the conclusion that the subject wants revision, for every large tonsil does not require enucleation. No operator is very proud of hæmorrhage after enucleation, but it is both common and serious. Will Dr. Moore state his experience as to whether hæmorrhage after the removal of the tonsil is not more troublesome now than formerly? Any method short of enucleation is worthy of consideration.

Dr. Irwin Moore (in reply): I wish to make it plain that I did not mean in my paper to imply that any colleague would be so foolish as to operate actually during the period of the menstrual flow: many cases I have seen have been those in which too little care had been taken to ascertain the usual menstrual time. The patient may be five days off the period, but, owing perhaps to the nervous strain of the complete operation, menstruation comes on before it is due. Fuller details of this method will be published in my full paper in the Journal of Laryngology, and I hope to illustrate it by a drawing. The cautery is not satisfactory in my opinion in the open cryptic forms of enlarged tonsils, because it is practically impossible to ensure that the cautery point is passed into the tissues between the crypts. One cannot be certain of passing it down to the bottom of a crypt, and destroying it, hence if only the superficial portion of the crypt is destroyed it may become blocked and an abscess result.
in the deeper portion. I have only used the galvano-cautery for fibrous tonsils, where the crypts are few; I do not advise it in the cryptic type. The escharotic paste does not block up the openings of the crypts, whatever size they are, and it is astonishing what the escharotic paste will do in getting at the deeper parts and causing shrinkage of the tonsil. With regard to statistics, I have not only put forward Morell Mackenzie's statistics, but also those of Fournié and Ruppenner. The latter had a great reputation in New York in the sixties, and had a large clientele in connexion with the reduction of tonsils. My own experience belongs mostly to the days before I was a specialist, and before I knew how to use a guillotine, or to enucleate. But I have done certainly fifty cases, and the results were satisfactory in every case. The cases were very mixed, both in regard to age and type of tonsil. Speaking generally, one may say that this paste method can be used in every type of tonsil, but the cases in which in my opinion it should be employed are those in which it is not considered advisable to perform enucleation. My paper only deals with those cases which are unsuitable for tonsillectomy. The galvano-cautery causes a good deal of pain, whereas there is practically no pain with this method, especially if the patients are carefully prepared with cocaine before applying the paste. In answer to Mr. Rose, my experience is, that where it is necessary to destroy the tonsillar tissue lying under cover of the palate, it can be reached quite well with my instrument. My experience of complete enucleation of the tonsil by dissection or guillotine compared with incomplete tonsillectomy is that if bleeding occurs, it is much easier to control: if any vessels are spurting, you can easily pick them up and tie them.

Dr. Irwin Moore showed his instruments and demonstrated the method of application. He said: All you require to do is press the patient's tongue down with a spatula for from five to ten seconds while applying the paste. The important thing is the mixing of the powder with alcohol to get the right consistency. You can watch the changes taking place on the tonsil; at first it shows a blood red patch, which rapidly becomes dark. Before the patient leaves your consulting room the paste has done the first stage of its work.

**Pharyngeal Diverticula with Notes of Two Cases—in one the Pouch was removed under Local, in the other under General, Anaesthesia**

By W. H. Kelson, M.D.

The following headings summarize the main points of the paper:
(1) Early work of Rokitansky, Zenker, Wheeler, Rosenthal, Slack, Kocher, Bergmann. (2) Butlin's first case and his report of subsequent cases, his methods and conclusions. Stettin's case and tables.
(3) Advances in our knowledge of the parts affected and in our methods of diagnosis. (4) Various methods of treatment other than excision.
(5) Waggetts', Mosher's, Hill's and Grant's cases and tables from the Mayo Clinic. (6) Views as to the best operative treatment for these cases.

Case I.—G. W., male, aged 70, first seen in November, 1913, when he came to hospital with the diagnosis of malignant stricture, and with the history of difficulty in swallowing, and regurgitation of food for over two years. He was stated to have lost much weight, scaling at this time 7 st. 12 lb. On examination a variable swelling was detected low down in the neck on the left side. The oesophagoscope very readily entered a large pouch, but the oesophageal opening was very difficult to find, but, once found, the absence of stricture was demonstrated. After preliminary cleansing of the mouth and pouch, the pouch was removed on December 3, 1914, the operation being performed under local anaesthesia.1 A feeding tube was passed into the stomach during the operation and retained, the neck of the sack was sewn up (two layers of sutures), the wound in the neck drained. Patient suffered much from cough, and a slight leakage of food took place through the wound on the fifth day. The fistula closed and the patient slowly but steadily gained strength. Dr. Jordan's X-ray report made just before patient left hospital on January 30, states: "A thick bismuth emulsion passed through the oesophagus with perfect freedom, and there was no obstruction or deviation at the seat of the former pharyngeal pouch." In 1917 he came to hospital quite well, and had gained 3 st. in weight; he swallowed without difficulty, and there was no evidence of recurrence on examination with the oesophagoscope. Only a slight puckering was visible at the site of the excision.

Case II.—P. W., male, aged 70. Seen in consultation with Mr. Woakes, who assisted me at the operation. Patient was feeble and emaciated, tired out by the constant regurgitation of food and drink, sleepless, irritable, and depressed. The case was diagnosed as one of pharyngeal pouch, this was confirmed by an X-ray photograph and oesophagoscopy: no stricture could be found. April 30, 1914: Pouch was removed under general anaesthesia, and its neck sewn up. A feeding tube was passed through the mouth into the stomach. Patient was very restless and tolerated the tube badly, and it had to be finally removed on the sixth day. A fistula formed, a little milk coming

1 Vide Journ. Laryng., 1914, p. 198.
through the wound, which was well drained. The leakage, however, soon ceased, and on May 27 patient left hospital, able to swallow well, and rapidly gaining weight and strength. Inquiry in March of the present year elicited the reply that he was able to go out and about, and could swallow all right.

DISCUSSION.

Captain E. F. Risdon, C.A.M.C.: Did the taking out of the tube on the sixth day make any difference? It seems that the feeding tube was kept in much longer than necessary, and I have been surprised to find how well the patient could swallow food of a semi-solid nature.

Dr. Kelson (in reply): It did not appear to make any difference.

Dr. Irwin Moore: With regard to the successful results obtained in these two cases, the ease in which removal of the pouch was carried out under local anaesthesia constitutes a record. I have extensively searched the literature of pharyngeal diverticula and have not come across a case done under local anaesthesia. I show skiagrams of similar conditions in two elderly ladies, with pouches of early type. Both these cases have been treated on hygienic and dietetic principles: I advised no operation, but recommended that they should take plenty of time over their meals. The results have been most satisfactory. I also show drawings of well known specimens from the Royal College of Surgeons. In one of these cases a bougie was accidently passed through the pouch, followed in forty-eight hours by the death of the patient from acute mediastinitis. It is surprising to find how thin the sac is, especially its lower portion: it is like tissue paper and consists of mucous membrane only. The fatal issue in this case should be a warning not to use the bougie, and it also indicates that great care is necessary in using the oesophagoscope in those cases in which a diverticulum is suspected.

Sphenoidal Sinus Empyema in Cerebro-spinal Meningitis.

By D. Embleton, M.B., and E. A. Peters, M.D.

Westenhoeffer originally regarded the sphenoidal sinus as the origin of the infection of the meninges. Our observations were made at the Cerebro-spinal Fever Camp at Netley. Sphenoidal empyema was found (1) in recent acute cases, (2) in relapsing phases of the disease.

(1) In three acute cases abscess was located and the sinus drained. Slight exacerbation of symptoms followed and all three died: post mortem, extensive involvement of the whole pia arachnoid was found.
Early treatment by means of sera was found to be the only reliable method of treatment, and the mortality was much diminished by the use of improved polyvalent sera. We are unable to appreciate the value of draining the sphenoidal abscess in these acute cases, though in very early phases of the disease it might be recommended. Elsewhere we have pointed out that these meningococcal empyemata are closed: the closure is probably due to a catarrhal process involving narrow sphenoidal ostia apart from any question of individual resistance and pathogenic power of the meningococcus. Further observations on recovered cases showed that the sinuses could be readily explored by a probe. Bacteriological examination of the sphenoidal sinus in recovered positive cases and positive contacts did not in our hands lead to the discovery of the meningococcus in a single case. Bryant showed that the zone of meningococcal distribution in positives was the palate level. It may be remarked that as the ciliated epithelium ceases at this line the cilia are responsible for the concentration of cocci on the stratified epithelium. It is probable that the stratified epithelium overlying adenoid tissue may also accumulate cocci in children.

We would urge the importance of nasopharyngeal hygiene by the use of a suitable nasal paint which is swept into the nasopharynx by active cilia, and the paramount necessity of fresh air.

(2) In relapsing cases of cerebro-spinal meningitis drainage of the sphenoidal sinuses gives definite and gratifying results. In a sequence of seven cases coming under this category two died of the disease and the sphenoidal sinuses could only be explored post mortem: in both closed empyemata were found. The other five were treated by exploration and drainage: in all five pus varying from a thick yellow cream to glairy mucus was found. Two cases, which gave a negative post-nasal swab, became positive after the operation. All five cases exhibited exacerbation of the disease: three made good recoveries, one has, and had previous to drainage, signs of internal hydrocephalus, and the last case died somewhat suddenly ten days after the operation, and the meninges post mortem showed internal hydrocephalus and meningitis serosa benigna.

DISCUSSION.

Dr. P. Watson-Williams: The import of the observations and conclusions of the authors' paper is far-reaching. Valuable as they are in connexion with cerebro-spinal meningitis, they are of even more value when applied to various other acute infective diseases involving the upper air tract. As far back as 1900 I accidentally came across an example of a carrier of meningococcal infection
of the sphenoidal sinuses and this case is referred to in my "Rhinology," published in 1901. That case led me to believe that it was to the sphenoidal and other sinuses that we must look for the essential source of the infection which spreads upwards to the meninges, and hence I was greatly impressed with the clinching evidence of this afforded by Dr. Peters' and Dr. Embleton's work. The nasal sinuses form such perfect physiological test tubes, maintained at blood heat and with nutrient media constantly available, that once an infection is established, the unfortunate host is exposed to constant re-infection. Hence it seems very possible that acute influenzal or diphtheritic infection may in the same way take a far graver and often fatal course from involvement of the nasal accessory sinuses. I would urge the importance of a routine exploration of the sphenoidal and other sinuses with cultural and film investigations in all cases of cerebro-spinal meningitis, and that this should be done in the early stages of the disease, not only in the case of the moribund. If the resulting evidence shows that these sinuses are a usual site of infection, their early lavage and disinfection is likely to control the course of the disease to a useful degree. Such routine bacteriological investigation of the sphenoidal and antral sinuses can be easily and quickly done under local anaesthesia in all but highly nervous patients and children over 8, and even in the latter light general anaesthesia lasting a few minutes should allow of such investigations being satisfactorily performed. In young children the sphenoidal sinus is so undeveloped that approach to that sinus cannot be commenced without grave consideration and under circumstances of extreme urgency.

Mr. Howarth: The authors' paper, based upon large experience at Netley Hospital, is authoritative. Though one may get good results, in acute cases, it is the chronic type of case which is I think the more interesting, as carriers of cerebro-spinal meningitis are a danger to the community. I cannot agree with Dr. Watson-Williams that every case of cerebro-spinal meningitis should have the sphenoidal sinuses explored, but I certainly think that every "carrier" should undergo this exploration.

Sir StClair Thomson: This paper needs studying in extenso. The authors say it is easy, in relapsing cases, to probe the sinus. Why is it so? Was there any atrophy? I did not find it so easy. I have had my sphenoidal sinus explored. When I felt the probe entering the sinus it was not at all pleasant. I am glad to say the result was negative.

Dr. Peters (in reply): The sinuses are readily examined even in nervous patients under cocaine. The nose is sprayed and then packed far back with 10 per cent. A cannula (or preferably Watson-Williams' style and cannula) is introduced in line with the posterior and lower edge of the middle turbinal. A ridge can be felt on the posterior wall of the pharynx and a point \( \frac{1}{4} \) in. in front of this and \( \frac{1}{4} \) in. from the middle line is located. Pressure with the probe results in egg-shell crackling as the probe enters the cavity. There appear to be two conditions of rhinopharyngeal sepsis: (1) superficial; and (2) deep. The superficial consists of débris from the air and detritus from the
deeper crypts. The deeper sepsis is due to infection of the follicles: according to Wright, entry of micro-organisms into the blood depends on surface tension. It is deep sepsis which results in infection of the sphenoidal sinuses and sensitization for the meningococcus, that is, cerebro-spinal meningitis.

**Sellar Decompression for Pituitary Tumours.**

BY WALTER HOWARTH, F.R.C.S.

At the present time there does not appear to be any general agreement as to the best method of approaching tumours in the pituitary fossa. Opinion seems to be divided between the fronto-orbital method of Frazier and some form of trans-sphenoidal operation. A large number of operations have been done by the trans-sphenoidal route and there seem to be many reasons why it should not lightly be given up. The sella turcica can be approached trans-sphenoidally by three satisfactory methods:—


Although the first of these methods has been employed successfully by Cushing in a large number of cases, the rhinologist may object that it has no advantage over the purely endonasal route and in addition opens into the septic cavity of the mouth. The paranasal method supplies an admirable and less limited view of the sphenoidal region and can withstand the criticism that it is a septic avenue of approach.

Operations in the pituitary fossa are usually undertaken for symptoms which are due to increased intracranial pressure—e.g., headache, mental changes, &c., and increasing blindness due to pressure on the optic chiasma and tract, with marked diminution of the temporal portion of the visual fields. The trans-sphenoidal route enables decompression to be effected by a quick and fairly simple operation. It attacks the tumour directly in its fossa and enables the fossa to be emptied. It attacks it from below and if undertaken early may prevent or hinder the upward extension which presses on the optic fibres. If the tumour is cystic, drainage is adequately provided for and there is a good chance for removal of the cyst wall. Radium may readily be inserted on any subsequent occasion.

Of five personal cases that are referred to, one died the day following operation from hæmorrhage into the growth. Three of the remaining
cases benefited considerably for varying periods, whilst two of these were subsequently treated by the insertion of radium into the fossa. Better results would probably be obtained if the neurologist and ophthalmologist sent the cases earlier for operation.

DISCUSSION.

Dr. E. A. Peters: How does Mr. Howarth avoid the danger of haemorrhage on incision of the dura mater?

Dr. William Hill: Dr. Peters has put his finger upon the point that had occurred to me. I operated upon one of these cases by the Hirsch method through the septum, and it was a comparatively easy operation. I excised the dura, and began removing some tissue, and then I encountered some previous haemorrhage. I at once applied gauze soaked with adrenalin, and allowed it to remain in fifteen minutes. When I removed the plug bleeding re-commenced. I re-plugged. Later I was called to the hospital because the patient was dying from cerebral pressure. I took the plug out and bleeding re-commenced. I got the house surgeon to plug again, but the patient died. Post-mortem: It was found that the sphenoidal sinus had not been wounded. Nine-tenths, probably nineteen-twentieths, of these tumours are in the diaphragm of the sella, and this route enables radium to be applied: and if a cystic case this route enables it to be drained. In serious cases, at all events, the more obvious method was decompression through the skull direct rather than by the nasal route. I published some statistics about this operation in the Transactions of the Medical Society of London. The septal route, in my case, did not present any difficulty owing to the acromegaly.

Mr. J. F. O'Malley: Dr. Hill's case was the last communication we had on this subject. Occasionally I had the opportunity of watching Hirsch do an intranasal operation of this type. When Mr. Howarth had entered the sellar cavity how much of the tumour did he think he would be able to remove? Had he any means of satisfying himself that he was removing the whole tumour, and as to whether the benefit was really due to having removed the mass of tumour or to relieving the pressure?

Mr. W. G. Howarth (in reply): With regard to bleeding at the time of the operation, I have only operated on a few cases and I am fortunate in having had practically no bleeding. In my one fatal case the haemorrhage occurred about ten hours after operation, and took place into a cystic portion of the growth above the diaphragma sella. If bleeding should occur during the operation I do not see what we could do except adopt the ordinary surgical method of plugging. As regards the type of operation that I personally prefer, it is the endonasal. This is a comparatively simple procedure, and gives a very good exposure of the posterior sphenoidal wall. The operation was not designed for the removal of large growths and should be regarded mainly as a decompression operation. It should, I think, be performed earlier than is usually the case.
A Series of Cases of Maxillary Antral Disease: Some Points of Interest.

By W. S. Syme, M.D.

(Read by Dr. Irwin Moore.)

The communication is based on 878 cases of antral disease which have been observed since January, 1914. Of these, both antral cavities were affected in 599—the right alone in 143, and the left alone in 136. Operations were performed on 293 double, 56 right, and 47 left, and, except for three cases in which the intranasal procedure was adopted, the radical antral operation was performed. Local anaesthesia was used in 306, and general anaesthesia in 90 cases. In 26 cases choanal polypus was present, and in two of these there was a choanal polypus on both sides. Ordinary nasal polypi were present in 89 cases, and atrophic rhinitis in 27. Thirty-one patients complained of asthma. Other points of interest are discussed.

Microscopical Demonstration in the Museum Room.

By Wyatt Wingrave, M.D.

(1) "Bleeding wart" of the nasal septum.
(2) Plasmoma (granuloma simplex) of the nose. (Pyronin green stain.)
(3) Villous papilloma of nasal septum (osseous region).
(4) Arteritis obliterans—middle turbinals—tertiary syphilis. (Acid orcein stain.)
(5) Fauclal tonsil removed by enucleation to show elastic fibres. (Orcein stain.)
(6) Acid-fast bacilli occurring in atrophic rhinitis. Pseudo-tubercle bacilli stained by usual method (Z. Neelsen, \( \frac{1}{2} \) in.). Their close relationship to tubercle bacilli.¹
(7) Spirocheta faetida in Vincent's disease. "Negative" staining by silver colloid method (collargol). Superior to Indian ink method and easier than positive staining \( \frac{1}{2} \) in.

(8) False "Vincent" spirillosis. Spirilla and leptothrix (silver colloid stain) $\frac{1}{2}$ in.
(9) Perithelioma and endothelioma.
These growths are the most frequent of the malignant mesoblastic group (sarcomata). They possess but a local malignancy, as they do not attack adjacent glands.

**Notes on Buccal or Sucking Pads.**

Coronal sections (frozen) through heads of infants, from birth to 6 months old. Spherical circumscribed mass of true adipose tissue in each cheek. Surrounded by well defined fibrous capsules from which it can be easily "shelled" out.

In one emaciated infant (2 weeks old) the pad could not be found.
Although imperceptible in some cases of primary emaciation, the "pads" do not appreciably share in the general disappearance of body fat.
Their absence is responsible for the "sucking" difficulty and for "false adenoidism."
Existence not referred to in standard works on anatomy.

**References.**

Demonstration on "Cases, Casts, and Models illustrating Rhinoplasty."


Recent Improvements in the Technique of the Application of Radium in Malignant Diseases of the Oesophagus.

Epidiascopic Demonstration by William Hill, M.D., in the Barnes Hall, elucidated by forty-five screen projections of skiagrams, illustrations and diagrams.

Replies were subsequently given to a number of questions put by a member of the audience.

[This communication could not be usefully reported in abstract without reproductions of the whole of the illustrations, which the question of space precludes at present.]

APPENDIX: MUSEUM.

INSTRUMENTS AND APPARATUS, AND SPECIMENS.

Electrodes for the Treatment of Ozæna of the Nose by Ionic Medication.¹

Exhibited by Mr. A. J. Hutchison.

Specimens representing diseases of the following regions were exhibited:—

1. Head and Face.
2. Nares and Accessory Sinuses.
3. Pituitary Fossa.

A full description of the more interesting specimens will be published later in the Proceedings.

**SCIENTIFIC RELICS OF MORELL MACKENZIE.¹**

*Exhibited in the Barnes Hall.*

(1) Manuscripts and documents formerly belonging to the late Sir Morell Mackenzie, and presented to the Royal Society of Medicine by Mr. Mayer.

(2) Laryngeal and nasal instruments formerly in the possession of or designed by Sir Morell Mackenzie.

A number of these were presented by Mr. Mayer, whilst the remainder, which came into the possession of the late Mr. Cresswell Baber after Sir Morell Mackenzie's death, were presented by Mrs. Cresswell Baber through Dr. Dan McKenzie.

**HER HIGHNESS PRINCESS MARIE LOUISE,** visited the Summer Congress of the Section of Laryngology on Saturday afternoon, May 3. She was received by Brigadier-General Birkett, C.B., C.A.M.C. (Hon. President of the Congress), Dr. James Donelan (President of the Section), Sir Humphry Rolleston, K.C.B. (President of the Royal Society of Medicine), Dr. William Pasteur (Hon. Treasurer), and Mr. J. Y. W. MacAlister (Secretary), and also Dr. Irwin Moore, one of the Hon. Secretaries of the Congress.

Her Highness was greatly interested in the exhibition of Plastic Facial War Injuries from Queen's Hospital, Sidcup, shown by Major H. D. Gillies, R.A.M.C., and also in the collection of manuscripts and instruments belonging to the late Sir Morell Mackenzie.

¹ These relics have been catalogued, with illustrative notes, by Dr. Irwin Moore, and published in the *Journ. Laryng., Rhin, and Otol.*, 1919, xxxiv, pp. 278-285.
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Section of Medicine.

President—Sir A. E. Garrod, K.C.M.G.

Spirochaetosis Icterohæmorrhagica.

By Sir Bertrand Dawson, G.C.V.O., C.B., M.D.

(Abstract.)

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'Spirochæta icterohæmorrhagica', discovered by Inada and Ido in 1914, and first cultivated by Ito and Matsuzaki, is one of few spirochætes known to be pathogenic. It varies in length between 4 and 25 μ—average 8 to 9 μ; the ends are sharp and often hooked; it is irregularly undulated as a rule, but other shapes occur. It can be grown with comparative ease in a variety of media, at temperature between 22° and 32° C. Its vitality is considerable, though capricious. These spirochætes are agglutinated by appropriate antiserum.

Clinical Course.

The disease is most often sudden in onset, beginning with shivers, head and body pains, great prostration, vomiting and diarrhœa, and a quick rise of temperature to 102° F. or higher. Within three or four days the conjunctivæ become suffused, and labial herpes (often hæmorrhagic) is common. Bleeding from nose, lungs, stomach, or bowel, or in the form of purpura, occurs in severe cases, early hæmoptysis being a valuable diagnostic sign.

Jaundice appears on the fourth or fifth day, sometimes as early as the second or as late as the seventh, and reaches its height about the tenth or twelfth; it may be intense. Constipation is marked, with light-brown or clay-like stools; the tongue is furred and brown; the

† At a meeting of the Section, held March 28, 1918.
liver is often definitely enlarged and the upper abdomen tender. The spleen is not palpable. The axillary and inguinal glands may be enlarged and shotty.

In severe cases there is acute bronchitis, with respiration-rate up to 30. The pulse is slow in proportion to the pyrexia, but the blood-pressure is higher than in enteric fever. Early weakness and prostration are characteristic. Death is preceded by coma, and sometimes by twitchings, convulsions and uræmic breathing. The urine contains much bile: albumin and casts are commonly present.

In acute cases irregular pyrexia lasts ten to fourteen days and ends by lysis, but a secondary rise of fever may occur early in the third week. Convalescence is slow, but recovery, when it takes place, is complete. Many cases are mild and ill-defined.

**Morbid Appearances in Fatal Cases.**

The duodenal mucous membrane is very œdematous and congested and of a dark-blue plum colour, with a red raised area round the bile-duct orifice. The stomach and first 3 ft. of jejunum are also inflamed, but less noticeably; the rest of the intestines normal and likewise the common bile-duct except at its termination. Many enlarged lymph glands are found at the edge of the lesser omentum and about the bile-ducts. The ampulla is swollen, congested and blue, and in one case was filled with a plug of cell nuclei and mucin. In some instances the spirochetal infection is evidently localized in the duodenum, but in others the duodenum shows no notable change.

The morbid changes in the liver vary—they may be insignificant, slight, or advanced. Sometimes the naked-eye changes are ill-marked when microscopical section shows cell damage somewhat resembling that seen in acute yellow atrophy. In a rapid acute case the liver showed advanced changes—both naked-eye and microscopic: it was small, with wrinkled capsule, and contained many necrotic areas in which the cells were quite destroyed and only the interstitial framework was left; elsewhere the cells showed some signs of reaction, and the endothelial lining of the bile capillaries appeared to be healthy.

The kidney changes also vary, though not so much. They range between cloudy swelling and disorganization approaching to necrosis. The spleen and pancreas are unaffected, but peritoneal, subpleural and subpericardial hæmorrhages are common, and hæmorrhage may occur within the lung substance.
JAUNDICE.

Thus, clinically as well as pathogenically, the brunt of the infection does not always fall on the same organs. But the duodenum and liver are often affected, and hence jaundice is common.

(1) Where the duodenum and papilla are inflamed and swollen, and the liver itself is unchanged save for bile stasis, the jaundice is due to obstruction of the outlet of the common bile-duct.

(2) Where the lobules are disorganized and the cells and intra-hepatic ducts are damaged, the jaundice is due to interference with the drainage of bile within the liver.

(3) Where there is no definite liver change and no duodenal swelling, jaundice is absent.

Pathologically, there is a striking resemblance between spirochaetosis icterohæmorrhagica and acute and subacute yellow atrophy. Clinically, acute yellow atrophy, though running a rapid course as a rule, may last fourteen or more days. Again, the clinical picture may be that of acute yellow atrophy, yet no characteristic liver changes are found after death, and such cases are called “icterus gravis.” Further, Rolleston has described a case of acute yellow atrophy without jaundice.

I suggest that what we style in the post-mortem room acute yellow atrophy is the fullest anatomical expression of a destructive process of which there are several grades, and which may be caused by several agencies, including Spirochæta icterohæmorrhagica.

The disease as it occurs in the guinea-pig has bearings upon the human problem. Mice, rats, and rabbits are also prone to it, but guinea-pigs are the most susceptible. In the guinea-pig the incubation period is six to thirteen days; the manifestations are fever, conjunctival congestion, hæmorrhages, jaundice and albuminuria. Jaundice appears when the pyrexia is at its height; twenty-four hours later the temperature falls below normal, collapse sets in, and death follows. Spirochætes appear in the blood and urine. Jaundice is occasionally absent. The pathological changes are characteristic. The skin and internal surfaces are yellow, hæmorrhages are widespread, the lymph glands and the bowel are congested and often hæmorrhagic. The liver to the naked eye is natural, but microscopically changes are seen varying from cloudy swelling to acute parenchymatous degeneration with necrotic areas. The kidneys are congested and hæmorrhagic and show parenchymatous nephritis. Hæmorrhages are usual under the
pleura, in the lung, and in the suprarenals. Abundant spirochaetes are found with ease in the blood, urine and faeces, and in the liver, kidneys and suprarenals.

The source of infection in man is probably to be found in water and food fouled by the urine of infected rats.

**Diagnosis.**

In the early stage the symptoms are those common to enteric, influenza and trench fever. Hæmorrhages should arouse suspicion; also conjunctival suffusion and hæmorrhagic herpes. Diagnostic inoculation of a guinea-pig is of small use after the eighth day; but the test, when positive, is conclusive. Spirochaetes appear in the urine about the ninth day, increasing in number towards the fifteenth day, and disappearing at the end of the fifth week; the technique of identification needs practice. The specific spirochaete is excreted by the kidneys, and contaminating urethral spirochaetes of the meatal type must be eliminated. Recent investigations, however, have shown that spirochaetes other than *Spirochaeta icterohæmorrhagica*, but resembling it, are excreted by the kidneys in some cases of obscure fever (P.U.O.).

A positive diagnosis must rest on the presence of the typical spirochaete, taken in conjunction with the clinical evidence.

**DISCUSSION.**

Major-General Sir David Bruce: No spirochaetes have been found in cases of jaundice in South Africa. There is no proof that spirochaetes have been found in trench fever. None are present if precautions are taken in collecting the urine. There does not yet appear to be sufficient proof to warrant the assumption that the infection with *Spirochaeta icterohæmorrhagica* takes place by the mouth. The same thing was said of malaria before it was proved otherwise. Infection may occur through the skin.

Dr. H. D. Rolleston: Probably many cases of "Weil's disease" are spirochetal jaundice, whilst the splenic enlargement in others may be evidence of enteric jaundice. Is there any evidence of acidosis in the terminal stages (with coma and air-hunger)? Has Sir Bertrand Dawson tried the serum of patients who have recovered? I believe the Japanese have used horse serum.

Sir Bertrand Dawson (in reply): I have not used the Japanese serum, as the cases have become so mild. We have used serum prepared in England, but the number of cases have been too few to enable any judgement as to the result being formed. I tested for acidosis in one case, but obtained a negative result.
Section of Medicine.

President—Sir A. E. Garrod, K.C.M.G., F.R.S.

Transfusion in Diseases of the Blood.

By O. Leyton, M.D.

Although I have transferred more than 60 litres of blood from donors to recipients, I am not in a position to make any positive statements as to the efficacy of transfusions. The main object of this paper is to describe the method adopted and the result obtained.

About seven years ago a man suffering from pernicious anaemia was admitted to a ward in the London Hospital and placed in a bed adjacent to one occupied by a patient whose blood contained nine million red cells per cubic millimetre and 150 per cent. haemoglobin. This coincidence suggested the transfusion of the anaemic with blood of the erythraemic, and the transference of a small amount of the anaemic blood to the erythraemic. The risks associated with the transfusion were explained to the patients, both of whom asserted that their lives were not worth living unless some alleviation were given them, and that therefore they would submit most willingly to any treatment that I thought advisable, however great the risk.

A 20 c.c. syringe was fitted with a three-way tap, suitable rubber tubes attached, and appropriate needles. The whole of the apparatus was washed out with liquid paraffin and drained. Blood was drawn into the syringe from a vein of the erythraemic patient and injected into a vein of the patient suffering from pernicious anaemia. After 40 c.c. had been injected the man complained of extremely severe cramp.

\footnote{At a meeting of the Section, held February 28, 1919.}
in his limbs. The transfusion was stopped. No anaemic blood was injected into the erythremic. A quarter of an hour later the symptoms subsided. It was found that the blood had improved from 1½ million to 2½ million red cells per cubic millimetre.

At that time I was not acquainted with Moss's work on iso-agglutinins, and inclined to the view that the symptoms which simulated caisson disease must have been caused by minute quantities of liquid paraffin obstructing the capillaries of the central nervous system.

The anaemic patient felt so much better that he requested me to give him another transfusion in spite of the discomfort the first had entailed. On the second occasion care was taken to expel all excess of liquid paraffin; a glass window was put in the tube close to the needle to be certain that no air was injected; 80 c.c. were injected when the same symptoms appeared along with nausea, vomiting, and severe palpitation of the heart. The cause, in all probability, was temporary obstruction of the finer vessels of the central nervous system by clumps of red cells. The donor belonged to Group 2 whilst the patient was either Group 3 or 4. No lasting improvement followed the second transfusion, and the patient left the hospital several months later in a condition similar to that in which he was admitted.

In June, 1916, I was anxious to carry out a series of transfusions in a case of recurrent pernicious anaemia, but did not obtain consent of all concerned until January, 1917, when the patient was in extremis. The red cells were about half a million per cubic millimetre, there was considerable oedema, and his mind was wandering. The patient's blood was of Group 4. Students of the London Hospital volunteered as donors. I decided to use the multiple syringe method because it was obvious that many transfusions would have to be performed, and therefore some method which did not entail any cutting or obliteration of veins should be adopted. I found that there was no difficulty in transferring 200 to 300 c.c. by this method, but then the blood in the needle inserted into the donor's arm clotted, and a fresh needle had to be inserted into a fresh vein. In order to obtain 600 c.c. the donor had to be pricked at least three times, and sometimes when fright hastened the clotting of the blood the number might rise to four or five.

In my hands this difficulty could not be overcome by having especially well-polished needles, nor by lining them with grease unless the lumen was very great, too great to introduce with ease into the vein of an average man: therefore I had needles made with cannula. If the blood in the cannula clotted, that cannula was withdrawn and
a fresh one inserted. By this method an unlimited quantity of blood might be obtained by pricking the donor on one occasion only. Needles and cannulae of various gauges were made. Perhaps the most important detail is the sharpness of the needles. It is essential for the needle to be sharpened every time it is used, and it is wise for the operator to sharpen it himself, since the correct angle and curve make it easy to insert into a vein. Instrument makers rarely combine the correct angle, curve and point. The cannula should project about 0.5 mm. beyond the point of the needle, and the end should be rounded to prevent it damaging the wall of the vein.

The syringes used were of 20 c.c. capacity and had about 10 cm. of rubber tubing attached. The capacity of the tubing was about 1 c.c. At the end of the tubing a nozzle was fixed which fitted the mount of the needle. The nozzle made an air-tight joint when inserted into the mount of the needle. The syringes were coated with vaseline and washed out with liquid paraffin in order that the tubing and nozzle should be lined with grease. The rubber tubes were filled with a solution of sodium citrate 5 per cent., and sodium chloride 0.45 per cent. The object of this solution was twofold: it prevented the waste of blood, and coagulation. Very often it was unnecessary to make the solution mix with the blood in the syringe, it floated upon the blood and when the piston of the syringe was pressed down, it filled the rubber tubing and ensured all the blood drawn from the donor reaching the recipient. It might be argued: Why have any tubing attached to the syringes, why not fit the syringe direct to the needle? Any operator who attempts to inject thirty syringefuls of blood will appreciate the advantage of having a flexible union, and of being able to move the syringe a little without damaging the vein. The tubing must be about 10 cm. long, in order that the nozzle at the end may be held between the finger and thumb whilst the syringe rests in the palm of the hand. This allows the operator to hold the shank of the needle with the left hand whilst he fits the nozzle into it with syringe and nozzle in the right hand. If the blood showed any tendency to clot, the citrate solution was made to mix with the blood by shaking the syringe slightly.

At the present day it is almost unnecessary to insist on the importance of the solution being made with distilled water which has been sterilized immediately after distillation, before there has been any growth of microbes in it. Vessels containing sterile normal saline should be at the side of recipient and donor, in case the rate of drawing
and injecting the blood does not correspond: the interval may be filled by passing saline through the cannula and thereby preventing any clot forming in the vein. The introduction of the needle into the vein is facilitated by distending the vein. The maximum pressure of blood in the vein may be attained by applying a pneumatic armlet and compressing the air in it to the diastolic blood-pressure of the donor or patient. The arm should be kept warm until all has been prepared, and everything ready for the introduction of the needle into the vein. The pressure of the armlet should not be released until the cannula has been passed through the needle. When the transfusions are carried out with short intervals the needle may be introduced on several occasions into the same vein, through the same puncture in the skin.

The selection of the donor depends upon a variety of circumstances. Of course it is advisable for the donor to belong to the same group as the recipient. Some surgeons assert that this is not true when transfusing for a simple loss of blood: on that point I am not in a position to express an opinion, but that it is essential in transfusion for diseases of the blood is extremely probable. The experience related earlier in this paper proves it, and the following confirms the view: A girl, aged 20, suffering from aplastic anaemia belonged to Group 4 and had had several transfusions from donors of the same group. On one occasion after I had injected two syringefuls of blood she complained of feeling faint and sick, then of her skin itching and her hands swelling. The transfusion was stopped. The patient had a variety of symptoms—palpitation of the heart, rigors, urticaria, slight purpura, vomiting, albuminuria and haemoglobinuria. The donor had been placed in Group 4 several months previously, and had given blood to a patient of that group with quite happy results. Upon investigating his blood again, he was found to belong to Group 3. This appears to be a case of a man changing from one group to another, but it is within the limits of possibility that the earlier grouping was an error, but then one would have expected his blood to have disagreed with the patient of Group 4. Not only did the patient’s blood agglutinate that of the donor on this occasion but also haemolysed it, so that harm only could have resulted from a more voluminous transfusion. I have heard of a case of death following transfusion with blood of a donor taken at random. I believe the surgical practice is to take blood from donors of Group 4 only, because their red cells will not be agglutinated by the serum of the recipient. It is within the limits of possibility that this practice would not be detrimental in diseases of the blood: there must, however, be the
risk that the serum of the blood injected may haemolyse and damage
the red cells of the patient if he be of some other group. Malaria,
syphilis and tuberculosis might possibly be transferred, and therefore
donors must be free from these diseases. When questioning a donor it
is my custom to point out that these diseases are communicable, and if
he has any of them to tell me. By putting the question in this way,
the donor does not acknowledge syphilis by stating that he is unsuitable.

Although the blood of a close relative, when of the same group,
may possess certain advantages, nevertheless I prefer a donor who is
unacquainted with the patient, because frequent transfusions may be
necessary, and the donor may assert falsely that he is feeling quite well
in order to supply further blood to his relative or friend. If the trans-
fusion is for a surgical operation and only a single transfusion is
anticipated, I see no reason for avoiding relatives as donors. The
preparation of the donor consists in explaining to him that he will not
feel any ill-effects of the loss of blood: that only 600 to 700 c.c. of
blood will be taken, less than half the amount which he could lose
without any discomfort. Usually the donor is given a cup of coffee
shortly before the transfusion (I found that this was better than a
whisky and soda) and is encouraged to smoke during the operation.
His attention is diverted by suitable conversation. On two or three
occasions subjective symptoms have arisen, but nothing of any
importance. It has been noticed that when the donor is frightened
his blood usually clots rapidly: sometimes when he feels faint the blood
cesses flowing from the vein. A short interval during which saline is
injected is required, and the transfusion proceeds.

There are no after-effects. Often a donor has given blood in the
evening and been up at six o'clock the next morning and done physical
work before breakfast: one of the London Hospital porters gave blood
at least six times in three months, and did not have one extra hour
off duty. I am in favour of using the same donor frequently, because it
is within the limits of possibility that the repeated removal of blood
leads to the production of some hormone, which will stimulate the
blood-forming cells. This hormone may be useful to the patient
suffering from aplastic anaemia. I am not acquainted with any
argument which tends to show that it is unwise to use the same
donor on many occasions. None of the donors who have given blood
on many occasions have suffered any ill-effects.

The preparation of the patient is simple, and consists of depriving
him of all but the lightest of food for six hours before the transfusion.
After some transfusions the patients have had rigors and short pyrexia. It is my impression that these are rendered less frequent and less intense by the hypodermic injection of a small dose of morphine and hyoscine half an hour before the transfusion begins. The origin of its use was the work of Drs. A. S. and Helen Leyton, who found that anaphylactic effects were diminished or abolished by those drugs.

The 100 transfusions upon which this paper is based were distributed most unevenly amongst ten patients, one having more than fifty transfusions, whilst two had only one transfusion each. I should like to record the observations which seem common to the majority, before giving details of each case. The immediate effect of the transfusion is masked by that of morphine and hyoscine. When these drugs have passed, the patient feels much less ill. Some have described it as like the effect of a bottle of champagne without the subsequent depression. When there has been haemorrhage from the bowel or uterus that haemorrhage has ceased. The vomiting and nausea—sometimes accompanied by diarrhœa common in extreme anœmia—are alleviated. The blood picture improves, and often the increase in the number of red cells on the day following the transfusion is greater than that which could be accounted for by the simple addition of red cells to the plasma of the patient, together with the red cells that were there before the transfusion. Perhaps the explanation of this may lie in polyuria, which is very noticeable during the first few hours after the transfusion, but one must remember that this follows the thirst which is created by the hyoscine and morphine. This increase in the number of red cells may be maintained or may disappear during the next four days. My experience is insufficient to decide whether one may conclude that the rapid disappearance of the red cells points to ordinary pernicious anœmia, in which red cells are manufactured and destroyed at an excessive rate; whilst the maintenance of the red cells occurs in aplastic anœmia, a condition characterized by marrow which normally forms red cells altering into a fatty variety, the main cause of the disease being the diminution in the manufacture of red cells, rather than any abnormal destruction. In one case the red cells were estimated and found to be 500,000 red cells per cubic millimetre. Taking the weight of the patient as 9 st., it might be assumed that her blood volume was 4 litres, therefore the total number of red cells in her blood was $2 \times 10^{11}$. After five transfusions of 600 c.c. each,—i.e., 3 litres—her red cells were 5,000,000 per cubic millimetre, that is to say, we added $15 \times 10^{12}$ of the red cells, and after that it was found she
possessed $20 \times 10^{12}$ red cells, the difference being $5 \times 10^{12}$, whilst the number of red cells she originally had was $2 \times 10^{11}$. Therefore, during that time she must have made $48 \times 10^{11}$ red cells, in addition to those which she had destroyed, or her blood must have become concentrated to a considerable extent.

At first sight this seems a marvellous success for treatment by transfusion, but it is only apparent because relapse occurred within a month. The red cells were below a million after that period. A further series of transfusions raised the number of red cells once more to within measurable distance of 5 million, but the rate of manufacture was slower than the rate of destruction, and therefore from time to time further transfusions had to be given.

In the first case of pernicious anaemia which received a series of transfusions the improvement after each injection was very transient, and after eight injections of about 400 c.c. each no appreciable alteration was observed. These injections had been given at an interval of several days. I decided that it would be wise to diminish the interval, and a further series of twelve were given on alternate days. This led to the red cells rising to $3\frac{1}{2}$ million, and since the spleen was enlarged it was decided to remove it. The operation was carried out with extreme care, and although the spleen was adherent over quite a large area, the loss of blood was small. The patient made a good recovery. Three weeks later a transfusion was given, and the patient then went into the country; but during the two years that followed, relapses occurred from time to time, and altogether more than 50 pints were transfused. The patient was able to walk about, and just before his death—which was traumatic and in no way connected with the treatment—he expressed the intention of undertaking useful work, for he felt quite equal to it. Usually five transfusions sufficed to raise his blood from slightly below 2 million to $3\frac{1}{2}$ million red cells per cubic millimetre, and he followed the advice of having transfusions whenever his blood fell and remained for more than three weeks below 2 million. In this case transfusion may have been considered a success, although one must realize that no cure had occurred, but the patient was being kept alive indefinitely by transfusions. One must also realize that pernicious anaemia is an extremely variable disease, but I cannot help thinking that the patient would have died two years earlier from anaemia if transfusions had not been adopted. This case was exceptional in another way—namely, that it was possible to carry out this very large number of transfusions. Frequently the
vein into which the injection of blood is made becomes thrombosed. It is true that when left alone for some weeks the thrombus is absorbed, and the vein can be used again: but this is not invariably the case, and in more than one patient transfusions have had to cease, because the superficial veins in the arms became occluded, and no other suitable veins presented themselves. In some of these cases, veins have been dissected out and cannulae tied in, but this method, too, is limited.

One of the most interesting cases in which transfusion appeared to do good was a woman, aged 44, who had been admitted to the London Hospital with a diagnosis of ovarian cyst with a twisted pedicle. Laparotomy was performed, and a large spleen with a twisted pedicle was removed. Examination of her blood showed that she was suffering from myeloid leukaemia. She was given arsenic in various forms, and improved greatly. After several months she returned to the London Hospital suffering from anaemia, red cells less than 1 million, haemoglobin 20 per cent. Arsenic did not lead to any improvement. Her blood happened to belong to Group 3, and just at that time an erythroæmic patient of the same blood group was admitted to the hospital to be bled. His blood was injected into the patient suffering from leukaemia: 600 c.c. were transferred. Following this transfusion her red cells gradually mounted to 5 million without any treatment with drugs, and she left the hospital feeling comparatively well. One must be guarded in drawing any conclusions, because myelogenous leukaemia is a disease with a very variable course.

Two cases of aplastic anaemia underwent several series of transfusions, and it was found that the first series led to a greater improvement than the second series, but after three or four series, haemorrhage occurred from the uterus and bowel, and although this haemorrhage ceased for a short time after another transfusion, it returned again within a few days, and it became a practical impossibility to replace the blood lost and destroyed. Both these cases developed vomiting and diarrhoea. One patient is still under observation, and all that can be ascribed to the transfusions is an amelioration of symptoms, which may make the end less trying.

In one case of so-called chronic pernicious anaemia in which the patient was complaining of loss of strength, and was under the impression that he was becoming more ill than ever before, a series of five transfusions raised the number of red cells from 2 to 3½ million per cubic millimetre, and the general condition improved greatly.
Although the number of red cells was not maintained, the patient has been able to carry on arduous work for more than a year, and has not needed any more transfusions. In one case of acute pernicious anaemia associated with cirrhosis of the liver, I was induced to give two transfusions. Although the donor was of the same blood group as the patient, each transfusion was followed by vomiting and discomfort, whilst the improvement in the blood picture was almost negligible. I did not think that I was justified in continuing the treatment, especially as the patient was fairly advanced in years.

In one case I gave two transfusions from the son of the patient. I was unable to take more than 300 c.c. Other donors were not procurable. These two transfusions, it is true, prolonged the life of the patient, but only by a few weeks.

Conclusions.

(1) In the majority of cases symptoms are alleviated by the transfusions.

(2) In some cases a series of transfusions at short intervals led to the blood becoming normal for a time.

(3) Perhaps in a small percentage of cases life can be maintained indefinitely by supplying the blood at the rate it is destroyed.

(4) In many cases disease is progressive, and that in spite of giving transfusions. The blood picture becomes worse and worse and death is only postponed for a short time.

I must express my best thanks to Dr. P. N. Panton, who has assisted me in every way in carrying out these transfusions.

The expenses have been defrayed in part by a grant from the research fund of the London Hospital Medical College, and in part by a donation from Captain Percy Sabel to the London Hospital for the express purpose of this investigation.
Section of Medicine.

President—Sir Archibald Garrod, K.C.M.G., M.D., F.R.S.

Apyrexial Symptoms in Malaria.¹

By Gordon Ward, M.D.

In the course of conversations with those who have seen many cases of malaria, I have been unable to find any general recognition of those symptoms which are unaccompanied by pyrexia. Nor have I been able to gather any opinions as to what symptoms, excepting those described as malarial cachexia, which might be held to justify a diagnosis of malaria when the parasite is not present in the blood—e.g., when a patient presents himself the day after an alleged rigor who has in the meantime taken quinine.

In the last thousand hospital cases which have been under my care particular attention has been paid to these symptoms in the belief that post-bellum practice will be very much concerned with them. Nor will this concern necessarily be short-lived, for cases which have endured at least five to ten years since return to a malaria-free climate have not been very infrequent, and in one case the Plasmodium vivax was found fifteen years after the patient’s return from India. One aspect of the problems involved may be put as follows. When an employer of labour asks his medical adviser whether such and such a workman shows any signs of having had a malarial attack on the previous day, for what signs confirmatory of the workman’s statement should the doctor look? When an ex-soldier applies for increase of pension or other consideration on the ground that he is suffering from chronic malaria, what are the symptoms which he is most likely to show?

¹ At a meeting of the Section, held January 28, 1919.
This paper is an attempt to answer these questions and is based on cases of malaria seen in special wards in England and France, but in which the disease has been contracted on an average twenty months previously in India, Salonica, Egypt and many other places.

I have often been asked by fellow practitioners whether these cases were in fact suffering from malaria and from nothing else when they exhibited the symptoms here described. On being pressed as to what evidence they require as proof of the disease being malaria, it is usually apparent that nothing short of the demonstration of the parasite in the blood will satisfy them. Alternatively they will sometimes accept the occurrence of rigors at regular intervals, and, of course, accompanied by pyrexia. Now, the symptoms with which this paper deals are apyrexial, and, the patient being under the influence of quinine, are seldom accompanied by the presence of the parasite in the blood. All the reply that one can make to such criticism is therefore as follows: The symptoms occurred in men who were sent to hospital as suffering from malaria. In many cases they were accompanied by a statement that the parasite had been found. In others the parasite was found after admission. In many the evidence of an actual rigor was accepted as sufficient when taken with the patient’s statement that he had had malaria and was, to the best of his knowledge, now suffering from an attack. In the great majority of cases there was no apparent reason to doubt the patient’s word, which was borne out by his symptoms. In a few cases (not included in the series here dealt with), a tendency to escape work or to prolong his stay in hospital, together with an absence of signs was regarded as making the case too doubtful for any statistical purpose. The symptoms noted were not exhibited only by those in whom the parasite was found, nor did those who did exhibit them show signs of any other disease. All cases in which a complicating disease was present are excluded. I therefore believe that I was in fact dealing with cases of malaria and consider that a study of the symptoms which they presented may well be of use to the medical man dealing with an isolated case and requested to answer the questions set forth above.

Apyrexial Rigor.—Rigor means shaking visible to others and not under the control of the patient, but in malaria the word is sometimes used to cover all the symptoms of an acute attack—i.e., to include a hot stage and subsequent sweating. It is my belief that this may occur without any rise of temperature, and I have myself witnessed it on more than one occasion. It may at first appear that
one could not have a hot stage without rise of temperature. The researches of William Hunter, in which he showed that the heat of an inflamed part was never really above that of the rest of the body, and that the apparent heat was only due to an excess of blood in the part are a sufficient answer to this supposition. The following is an example of apyrexial rigor:

Private T., aged 28, had two typical rigors during which Plasmodium vivax was found, and he was then put on quinine and came under my care. Sixteen days later his morning temperature was 97°4 F., and his pulse 72. At 10.30 a.m. the same morning he was noted by the nurse to be shaking but not sweating. At 10.40 a.m. he was seen by me and was then sweating profusely, and showed marked rigor which he attempted to control in vain. His eyes were suffused, but he had no pain and no headache. His temperature was 98°6 F., and his pulse 114, regular and full. He appeared very flushed and was hot to the touch. At 11 a.m. he was still sweating, but the rigor had almost ceased. His temperature was 98°2 F., his pulse 96, and respirations 40. At 11.10 a.m., he was no longer shaking, but complained of nausea. His blood was negative for parasites. At 12.45 p.m., his temperature was 98°6 F., his pulse 88, and respirations 30. At 2.10 p.m. he had headache which had come on about an hour previously. He had no longer any sweating. Two days later he had a similar but less severe attack, characterized by feeling cold, then hot, and sweating, and finally by headache. On other occasions he had similar attacks, but his temperature never rose above 99°4 F., while he was taking quinine.

In this case rigor happened to be very marked, and the patient looked and no doubt felt as ill as a man with a temperature of 104° F. He lay curled up in bed covered with blankets and evidently very unhappy. He had already been promised evacuation to England, and had nothing to gain by simulating attacks. Nor would it be possible to simulate such an attack as he had. His temperature was taken with three different thermometers, and I counted his pulse and respirations myself. It may be noted that his haemoglobin two days before the attack was 76 per cent. During the attack it was 82 per cent., the sweating producing concentration of the blood. Two days later it had fallen to 58 per cent., and not till four days later was it above 80 again.

To such attacks as this I am accustomed to give the name "minor relapse." Minor relapses are not usually so flamboyant as that just described, nor are they usually strictly afebrile, more often the rigor is absent, and the temperature rises to 99° F. or just over. The following case is an example:

The patient had two rigors of the usual type before he came under my care, and the Plasmodium vivax was demonstrated in the blood. He was then put on quinine. As soon as he was allowed up he began to have evening
pyrexia between 99° and 100°F. Each evening also he had symptoms which he himself recognized as malarial—e.g., a feeling of cold followed by flushing, sweating and headache. His blood, taken on one such occasion, showed no parasites, but the leucocytes were increased and the polymorphs 80 per cent. — just as is commonly the case at the onset of an ordinary malarial relapse.

In a similar case, but one in which the blood was examined three or four times, a few parasites was found on one occasion only. This man had not been exposed to re-infection for fifteen years. The parasites were *Plasmodium vivax*.

The pyrexia-producing effect of getting a patient up or allowing him to take a walk is commonly well marked, and this has led some to argue that the pyrexia is not to be considered in any way directly due to the malarial parasite. The argument does not take account of three points—viz.:

1. That parasites are occasionally found in the blood and other abnormalities more frequently.
2. That the symptoms which accompany the pyrexia (chill, flush, sweat, headache, nausea, &c.), are exactly those of an ordinary rigor.
3. That the pyrexia frequently shows tertian periodicity.

![Chart I.](image)

Showing the irregular type of pyrexia which is often seen in patients taking quinine, also showing quartan and tertian periodicity. The arrows show days on which minor relapses of malaria occurred.
Chart I is that of a man who showed quartan periodicity, and later tertian. He had also attacks on other days, and on one occasion rigor. He was taking 10 gr. of quinine daily for the first eight days, 30 gr. for the next eleven days, and then 10 gr. again. During the last week he had four intramuscular injections of 9 gr. each. None of these made any difference to his symptoms. *Plasmodium vivax* was present in his blood just before admission. He showed no signs of any other disease. The headache complained of was confirmed by a marked hyperalgesia to pin prick in the area of the supra-orbital nerve, which was easily marked out by this method. Complaint of pain in the left side was considered to be genuine in view of the presence of an audible spleen rub in the seventh interspace due to perisplenitis.

*Chart II.*

Showing tertian periodicity: the arrows show days on which minor relapses occurred.

Chart II shows more regular tertian periodicity, and there were no attacks on the intermediate days. Here, again, increasing the dose of quinine had no effect, although, as in the last case, an increased absorption was shown by the tinnitus, &c., provoked. Here also the *Plasmodium vivax* was demonstrated on admission, and the presence of pains in the legs and head was confirmed by characteristic hyperalgesias in those regions. The blood did not show parasites after he had been taking quinine, but did show abnormalities in the red and white cells, such as one associated with malaria.
Chart III is from the case of a man who can hardly be said to have had any pyrexia while in hospital. Nevertheless the suggestion of tertian periodicity is obvious. He complained chiefly of pain in the left side—he had had a rigor before admission—and had some audible crepitations at the left base. This is very common in malaria. He had also well-marked tremor, much wasting and general pigmentation. His haemoglobin was 82 per cent.; pallor was moderate. This is so exactly the picture that one gets with chronic malaria when more or less apyrexial that the suggestion of periodicity in his chart is at least interesting.

When a patient can be kept under observation, such charts may assist the physician in diagnosing malaria, although quinine is being taken.

Chart IV shows that quinine will not necessarily prevent the more severe attacks, and also shows the mingling of rigors with high pyrexia and minor relapses with little pyrexia. In this case the *Plasmodium vivax* was found and the patient was under observation in France, having contracted malaria in Salonica twelve months previously. He was taking 10 gr. of quinine daily except during the fourth week, when he took 30 gr., with no particular effect except to cause severe tinnitus.

These cases demonstrate that there is an occurrence in the course of malaria to which the name "minor relapse" can properly be given, that in a minor relapse all the symptoms of a major relapse may be present except high pyrexia, that the parasite will only exceptionally be found in the blood, and that these attacks show a regular periodicity in many cases. They have been most often noted when the patient was taking quinine.

We can now pass to other symptoms which may assist a practitioner called upon to decide whether a patient has recently had an attack of malaria.

*Perisplenitis.—* The spleen is often enlarged in malaria, but it is not very often so easily palpable that there is no room for doubt about it. If it is palpable it affords useful confirmatory evidence of a patient's statement that he has been suffering from malaria. If it is not palpable it may still, in a few cases, be possible to detect some abnormality of this organ by auscultation. When the surface of the spleen is inflamed, as is often the case in malaria, there is as a rule only a small area affected. The process in this area is comparable to a pleurisy, and the same sort of friction sound may be heard. The exact position of this sound has been, in some of the cases, noted with accuracy as follows: (1) In the sixth intercostal space behind the nipple line; (2) in the
Chart III.

Showing tertian periodicity with the temperature not rising above the normal line.

Chart IV.

Showing the course of a case of malaria taking quinine.
Ward: *Apyrexial Symptoms in Malaria*

**Fig. 1.**
Charts from five cases showing apyrexial rigor. The extremes of temperature noted are shown and joined by a dotted line.

**Fig. 2.**
Charts showing the minor relapse preceding the major by a two days' interval. Black arrows show that definite malarial symptoms were observed; white arrows show that no note has been made with regard to symptoms.

**Fig. 3.**
Charts showing tertian periodicity of the lesser degrees of pyrexia and of minor relapses in malaria.
seven in the seventh intercostal space up to and behind the nipple line; (3) in the seventh interspace one inch behind the anterior axillary line; (4) in the seventh interspace just outside the nipple line; (5) in the seventh interspace half way between the anterior and posterior axillary lines. The pain, however, is often felt in front of the place at which the friction rub is best heard. When this amount of evidence of peri-splenitis is available it will often be found that certain other signs exist. Thus there are commonly a few rales to be heard at the left base, and the patient may say that the pain is worse on taking a deep breath. This will of course suggest that it is a pleurisy with which one is dealing, but the error can be avoided by hearing crepitations where the lung is not and where a pleuritic rub is unknown. In some cases the lung signs are very evident and may even progress to pneumonia or pleurisy. This is especially the case when the spleen is very much enlarged. When the condition quiets down some pleural adhesions are left and these give rise to pain in the side, often attributed to the spleen. A short course of deep breathing exercises will almost always dispel these pains if due to pleural adhesions; the same exercises have little or no effect on some cases, and in these one assumes that there are very likely splenic adhesions also. In the latter class of case, getting up will often produce a dragging pain that has been absent when the patient was in bed.

Pain in the Side.—It may often happen that a patient will present himself to a medical man, stating that he has had a malarial relapse on the previous night and has taken quinine, but that he has a pain in the side or a headache so severe that he feels unable to go to his work. In such a case any means of testing the truth of his statements could not fail to be of value. There is such a means, and it consists in a proper appreciation of the factors underlying the pain and of applying an objective test for the presence of those factors. We have already dealt with certain causes of pain in the side; there are, however, others. It is a fact, even if an inexplicable fact, that malaria is accompanied by certain areas of cutaneous hyperalgesia in a very large number of its victims (fig. 4). These areas are segmental—i.e., they correspond to the cutaneous distribution of the nerves derived from definite segments of the spinal cord. These areas show the positions now occupied by the various segments of which the body was originally built. Many of these segments have been so displaced by the outgrowth of limbs that the corresponding skin areas no longer have any segmental appearance, but in the case of the body this is not so and individual segments
can be traced as bands round the body. The area with which we are immediately concerned is the seventh dorsal, and this forms a band on the front of the body about midway between the xiphisternum and the umbilicus. In very many cases of malaria this segment is hypersensitive to painful stimuli, such as a pin-prick. Often lower dorsal segments are also involved. We test this in the following way: Pin-pricks are made at intervals of about one-sixth of an inch along a line drawn from the nipple to Poupart’s ligament. The pin should be so held that as it comes into contact with the skin the first finger also touches the skin. This enables one to exert even pressure and to avoid excessive pressure. When this is done the patient will indicate by an obvious flinch when the hyperalgesic area is reached. He may also be expected to wear an injured expression, for he naturally supposes that the pin has suddenly been pushed in more deeply than before. It is this flinch which
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constitutes the objective sign of the hyperalgesia. It is not under the patient's control, whereas his statements are and so are not to be relied on. The sign is commonly most marked on the left side of the body, and it has been thought that this betokens some connexion of the spleen with the seventh dorsal segment on its visceral side. Very possibly this is so, but the matter must not be taken as proved. This same segment is also affected in trench fever, and possibly in other conditions also, but this does not detract from the value of its demonstration as evidence that the pain of which a patient complains has at least a definite abnormal condition accompanying it. Where hyperalgesia can be demonstrated one can safely accept the patient's statement that he has pain in the area in question. Where hyperalgesia cannot be demonstrated the patient's statement can have only the value which one's impression of his character may allow it. This hyperalgesia is not found when a man has been free from malarial relapses for any considerable period.

Headache.—This is a very frequent symptom of malaria. It commonly affects the area supplied by the supra-orbital nerve. Hyperalgesia in this area may be detected by the method already detailed. This hyperalgesia is very variable in extent. As a rule the area is a large one, extending to the lower eyelid below and to the vertex above, while at the sides it is bounded roughly by a line drawn from the outer angle of the eye to the upper part of the ear (fig. 5). If this hyperalgesia is demonstrable the patient's statement that he has a headache may be safely accepted. It may be that the patient will complain of chronic headache rather than of the more acute form which follows a sudden relapse. In such cases certain other points may well receive attention. If the patient states that he has pain behind the eyes (which is due to a neuritis of that portion of the supra-orbital nerve which runs within the orbit), defective lateral movement of the eyes, nystagmoid jerkings and "pink eye" tend to confirm his statement. So also do pain on pressure over the supra-orbital notches and photophobia. The headache is frequently unilateral so far as the patient feels it, but even in such cases hyperalgesia is usually bilateral. Temporal headache is often associated with frontal, while occipital headache is rarer. The areas of these can be demarcated by the test for hyperalgesia in a considerable proportion of cases. The same is true of pains—usually described as "stiffness" by the patient—at the side of the neck. These have been two or three times noticed to accompany attacks, and to last for two or three days afterwards.
Pharyngitis.—It is stated in text-books that pharyngitis does not accompany malarial relapses. This statement is not borne out by the cases which I have had the opportunity of studying. Hoarseness, cough, or a "bad chest" were stated by several patients to accompany every severe attack of malaria. In all such cases there was either definite hoarseness present or else either throat or chest showed physical signs sufficient to cause a cough. In addition to this one often noticed mention of cough in the history of patients, and varying degrees of bronchitis were frequent. One has only the patient's statement for evidence that such symptoms actually recurred with each attack. It was only a minority who had taken sufficient notice of their symptoms to be sure on the point, but in this minority no reason was ever found to cast doubt on their veracity. Probably 10 per cent. of the malaria cases admitted to my wards had been diagnosed as influenza when first seen. It is therefore well to remember that pharyngitis and other
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catarrhs often complicate malaria. Of these catarrh of the conjunctiva is very common, of the throat and larynx less common, and of the lungs least common. The finding of any one of these is further evidence in support of a patient's statement that he has had an attack of malaria, and not, as might have been thought, evidence pointing in the opposite direction, and in favour of some such loose diagnosis as influenza.

The recent paper of Captain J. A. Thomson on "Pulmonary Spirochaetosis"\(^1\) demands some comment at this point. He has demonstrated bronchorrhoea apparently due to a spirochæte or spirillum in many cases of chronic malaria. With all due reserve he hazards the suggestion that the frequent presence of cough, of a slight irregular pyrexia, and of several other symptoms dealt with in this paper, may be due not to malaria but to this spirochaetosis. This view does not commend itself to me for the following reasons:

1. Most of the cases of cough encountered have been of brief duration and associated with acute attacks.

2. Blood-stained sputum has not been noted. This is probably because it was not looked for.

3. The irregular pyrexia seems to me to be malarial, because it is accompanied by symptoms such as one meets in malaria; it occurs in cases of very long duration (e.g., fifteen years) where there are no bronchial signs; it occurs in cases in which the disease was contracted in England or France, the patient never having been farther abroad; it occurs in those who have had salvarsan treatment; it is sometimes accompanied by the presence of malaria parasites in the blood and it frequently eventuates in high temperature with rigor when administration of quinine is stopped.

4. Further the irregular low pyrexia is often interrupted by classical relapses, and these relapses are often led up to by minor relapses two days previously.

Hyperidrosis.—Excessive sweating is of course usual during a relapse but we are now concerned with a chronic tendency to sweating which is often met with in old malarial subjects. I have found this more evident than patients are willing to admit. Thus many denied any such tendency and yet, even on a cold day, were found to be sweating heavily when examined at the time they made the statement. This is perhaps explained by the fact that a profuse perspiration is less frequent and therefore more obvious in those whose occupation is

sedentary, whereas the manual labourer has become so accustomed to it that he ceases to take any particular note of the condition of his skin in this respect. It is from the manual labourer class that the majority of the cases here dealt with were drawn. The perspiration has been most noticed on the chest and hands because these were the localities most often open to observation. No doubt the axillæ and certainly the feet were as commonly affected. Sweating would break out for no apparent reason or following the mild excitement produced by the fact that a patient was about to be examined. It is well to remember in hospital patients that much perspiration will be borne without complaint rather than risk the displeasure of the sister by throwing off a blanket and so untidying the bed. This is not necessarily hyperidrosis. Hyperidrosis is met with in many conditions but can properly take its place as confirmatory of a patient's statement that he has been suffering from malaria.

Pigmentation.—Pigmentation is common in subjects of malaria—and also in those who have been much exposed to the sun and yet have not suffered from malaria. One has therefore to exercise a good deal of care in estimating its value. In three or four cases in this series general pigmentation has been observed to deepen during attacks and to grow less between them. In these cases it was always pigmentation of the lips which most attracted attention although the rest of the body was also pigmented. This pigmentation affects the border of the red part of the lips. As far as my experience goes, it is very characteristic of malaria. Isolated patches of pigmentation—e.g., freckling of one shoulder or a cloud-like patch on the abdomen, have been often noted, and leucoderma (the edges of the white patches being pigmented) is by no means infrequent, but it is unwise to place much reliance on them as diagnostic points. At the most they suggest a previous residence in a hot climate.

Raynaud's Symptoms.—A moderate degree of vasomotor spasm is common in malaria. It takes the form of "dead fingers" and "cold feet." The following are a few examples:

1. Aged 39. Fourteen months' malaria. Feet cold and white below a line drawn across the instep.
3. Aged 21. Twenty-one months' malaria. Frequently dead fingers since having malaria.
4. Aged 31. Thirteen months' malaria. Frequently various degrees of
cold and dead feet. Sometimes only one affected. Inhalation of amyl nitrite somewhat diminished the "dead" area.

(5) Aged 32. Three months' malaria. Four outer toes on each foot and a part of sole and instep cold and dead. States that they go cold about 11 a.m. and are warm again by 2 p.m. each day.

(6) Aged 35. Twelve months' malaria. Often has cold fingers, only the ends being affected. Has also cold feet, about up to level of ankle, as a rule.

These cases were all seen and the dead fingers, &c., verified. The demarcation of the cold area was always easy by sight and touch. In many cases a coldness of the extremities is complained of, but there is no suggestion of actual spasm. Such cases are not included as examples of Raynaud's symptoms. These symptoms need looking for, as the patients do not commonly complain unless the condition is very persistent or well marked. It will often be found that such patients wear bed-socks. In one particular patient there were symptoms which combined Raynaud's disease, erythromelalgia, and the transient oedema presently to be described. He was aged 34, and had had malaria only five months. The lesions noted on different occasions were as follows:

(a) Transient oedema of the calves of both legs, usually on the outer side only. On one occasion there was a single area the size of a walnut, the swelling having disappeared next day.

(b) Redness of the feet and toes, made worse by hanging them over the edge of the bed. The feet were extremely painful, and the left especially painful to touch.

(c) The development of what appeared to be linear areas of erythema, in shape resembling a dilated vessel. These persisted during the period of observation, and a few new ones appeared during the same time. They were always acutely tender.

(d) The sudden development of swelling and bruising on one leg. When the swelling subsided what appeared like a small bunch of varicose veins was left; this had certainly not been there before.

This particular patient was a policeman before enlistment—a post which he will certainly be unable to occupy again unless great improvement takes place. He was under observation for two months, but it cannot be said that any treatment tried was of any use. He was better in bed and with his feet up. He had no albuminuria, but had frequent minor relapses of malaria. His haemoglobin was 96 per cent.

Transient Oedemas.—The following are examples of transient oedemas. It should, however, be stated that the condition is not necessarily such that pitting can be obtained; sometimes it more
resembles a giant urticaria, to which, indeed, it is almost certainly allied.

(1) Aged 21. Eleven months' malaria. *Plasmodium vivax* present. The following is the actual note made on this case when oedema was first seen: "Complains of swelling of fingers and feet. On examination, all fingers swollen about the proximal digits. Ball of great toe appears swollen. No pitting. Cannot close hand properly. Cannot replace ring which he removed this morning. Haemoglobin 73 per cent. Pallor of face, but tongue good colour. States he never had such symptoms before." It was ascertained that he had no albuminuria, and the index finger was measured then and again in the afternoon. By the next morning the fingers were normal again. The maximum increase in circumference was 0·55 cm. for the left hand and 0·4 cm. for the right, the index finger alone being measured in each case. The two distal phalanges of each finger were free from swelling throughout.

(2) Aged 29. Twice infected with malaria, with a clear period of three years between the two occasions. Total duration of malaria four years. *Plasmodium vivax* found after admission and six days before the symptoms described. One evening he noted swelling of his legs. It was noted that marks of the pressure of his socks could be seen. There was no pitting. Swelling was obvious, but slight, and affected only the area of the calves and the middle of the tibia. Measurements were made every day, morning and evening, the patient being allowed up meanwhile. The swelling lasted four days, diminishing slightly on the third day and vanishing on the fifth. Only twice were sock marks noted, and even on these occasions it was not possible to satisfy oneself that there was any pitting. There was no albuminuria. He did not appear anemic.

(3) Aged 31. Two years' malaria. Malarial parasite present, variety not stated. Six days before symptoms were complained of. Patient presented on the dorsum of the right foot a semi-fluctuant white swelling which occupied about one-third of the dorsum. It lasted four days, and the maximum difference in circumference of the foot was 1 cm. There was indefinite swelling of the same area on the other foot. No albuminuria. The swollen area was tender, and it was this that had directed the patient's attention to it.

Symptoms such as the above are of interest, not only from their possible importance in diagnosis but from the doubt as to their exact causation. It is customary to describe them as vasomotor, and to most people this implies that they are in some way of central nervous origin rather than that their cause is to be found in any local condition. I am inclined to believe that they depend, in part at least, on actual changes in the walls of the arterioles or venules. For this opinion there are two reasons. The first is that, in all cases of true erythromelalgia, the pathological condition present has been shown to be an
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arteriolitis, with nerve changes secondary, if present at all. The second reason is a new one, it is the finding in the blood of malarial patients of cells which I believe to be endothelial cells derived from the blood-vessels—sometimes to the number of 1 or 2 per cent. of all white cells present.

The value of a Negative Blood Film.—The finding of malarial parasites in a blood film is very strong evidence that the symptoms of which a patient complains are malarial in origin. It is not, however, absolute proof. In the same way the absence of malarial parasites is no evidence that the disease is not malarial, for the patient may have been taking quinine or he may be suffering from malignant malaria in which the parasite (Plasmodium falciparum) is present in the blood for a comparatively short time only. It must not be supposed that the presence of the parasite is the only factor which differentiates a malarial from a normal film. Malaria is responsible for various other changes which it shares in varying degrees with other diseases. The presence of pigmented cells—i.e., pigment-carrying cells—is very suggestive of malaria. These cells are either the common polymorph or the "transitional." The "transitional" cell, which rejoices in a great number of names, is larger than a polymorph and has a convoluted, but not partite, nucleus, in which the chromatin markings are relatively poorly developed. The pigment appears as small spots of a greyish material within these cells, and as a rule many cells are affected. The finding of an isolated pigment cell is unusual and inconclusive. The presence of over 10 per cent. of transitional cells is suggestive of malaria, but these also occur in very many other conditions. The total number of leucocytes is often increased when the parasites first enter the blood. With this leucocytosis the polymorphs are in excess. It gives place quickly to a moderate degree of leucopenia, in which the polymorphs are diminished so that the lymphocytes are apparently in excess. Either of these findings is extremely common in many other conditions, and no reliance can be placed on them except that a leucocytosis without parasites is probably not malarial. The eosinophils are often increased in chronic malaria—up to 5 to 10 per cent. Fig. 6 shows several drawings of endothelial cells made by me from a malaria film. These cells are characterized by a large oval nucleus of indistinct structure, but usually with a well defined nucleolus. The cytoplasm is frayed and indistinct—which is but natural, since the cell is not really a blood cell but a tissue cell that has become sufficiently decrepit.
to be detached from its normal attachments and shed off into the blood stream. These cells may be found actually joined together, but more often are displaced in the process of spreading the film. They are then found not actually joined together but in groups of two or three.

Fig. 6.
Endothelial cells from a single film drawn under the camera lucida. For comparison a polymorph (P), an eosinophil (E), and several red cells (RBC), all from the same film. (Drawn by the author.)

In a case of kala-azar I found as many as three and four of these cells joined together and others which showed mitosis. But for this case I should perhaps not have recognized their nature, but I have found
them in only one or two cases other than malaria. The one other case in which they were most plentiful was that of an old man who was subsequently found to have suffered from what he described as ague ten years previously in America. In malaria these cells are often encountered, and, in my opinion, they appear to supply valuable confirmatory evidence in a doubtful case. The red cells are also abnormal in malaria. Red cells are destroyed by the action of the parasite, and the products of this destruction circulating in the blood are the most potent stimulants to new red-cell formation. It thus happens that the blood film following a severe rigor will usually show signs of this new formation in the presence of red cells larger than normal, exhibiting polychromatic staining, and, perhaps, some of them nucleated. These things are not found in normal blood, and their presence may therefore be of value in a doubtful case. Taking all these points together, one sees that a critical examination of a blood film, even if parasites are not found, may be of distinct value.

**Tremor.**—Tremor is usual in chronic malaria: it may exist as a continuing phenomenon or be present only for a few days after attacks. This tremor is seen in the tongue, the hands, and rarely in the lips. The tongue tremor is not under the patient's control. The hand tremor is not under control if it is malaria, but it is of course possible for a patient to simulate some sort of tremor. This will be a coarse tremor, and will disappear or become disordered and irregular if he is asked to screw his eyes up tight while holding his hands out in front of him. The malarial tremor is a fine tremor such as one may see in heavy smokers, &c.

**Herpes labialis.**—This is very frequent in malaria, i.e., if the attack has been a moderately severe one. It does not occur at once, but may be delayed from twenty-four to forty-eight hours or more. In any case, if a patient present himself with herpes, it will naturally occur to the physician that this is a symptom for which some explanation must be found.

**Jaundice.**—In certain persons jaundice is very apt to follow attack, and may persist a week or so. As a rule, it appears after severe attacks, but there can be no doubt about the individual susceptibility to this development. It depends on increased viscidity of bile due to increased destruction and elimination of blood pigments, and is therefore strongly suggestive of a recent malarial attack when seen in a malarial subject.

**Tachycardia.**—This is frequent. Commonly it is most marked when the patient gets up for the first time. It may then disappear after a
week or so; or it may persist apparently for so long as he has malaria. In many cases it is best brought out on exertion. The paradoxical response to exertion in the development of slowing of the pulse has also been seen. In one case a condition which seemed to be auricular fibrillation was present for three days.

Weakness.—Complaint of weakness is commonly made and two charts (fig. 7) are here presented illustrating certain measures taken to estimate this with exactitude. As such steps cannot be taken in ordinary practice, weakness must be deduced from the usual appearances—e.g., flabbiness of muscles and general want of tone, dyspnoea on exertion, &c.

Wasting.—"Malarial cachexia" is a term commonly employed but not often defined. Most agree that wasting is an essential part of the picture. Wasting is certainly common in malaria, but it is also a fact that a man may remain in good condition and good health in spite of frequent minor relapses. An absence of wasting is not, therefore, of great value as evidence. Anaemia is also recognized as a part of cachexia, but the term is loosely used, and provided pallor is present the condition is held to be fulfilled. Now, there is a real difference between the two. The colour of the lips is a better guide, and that of the tongue better still. Pallor is very common in malaria, but a pale man is not necessarily a sick man; an anaemic man on the other hand almost certainly requires treatment. I have seen a diagnosis of pernicious anaemia made in the case of a malaria patient who was hardly anaemic at all but very pale. Anaemia cannot, therefore, be said to be a part of malarial cachexia, but pallor would be expected and in its absence cachexia could hardly be present. The third factor recognized as necessary for the completion of the picture of malarial cachexia (so far as my inquiries go) is a large spleen. I may say at once that those who look for large spleens will be disappointed. Perhaps in one case in fifty the spleen is one or two fingers' breadth below the costal margin for a day or two following the acute attack; in several a combination of pain, rigidity of muscles and indefinite dullness will persuade the unwary that the spleen is enlarged. In most the wise man will refuse to express a confident opinion on the point. These remarks, of course, apply to malaria as seen in temperate climates only. In hot climates malaria often wears a different complexion. It is, then, possible for a man to be very wasted and very pale, and even very anaemic, and still not to have a palpably enlarged spleen. I recollect a case in which the red cells were only just over two million and the blood full of parasites, and yet
The figures to the left show the time in seconds during which the patient was able to hold a deep inspiration; this is the respiratory test. The figures to the right show the time in seconds during which the patient was able to hold at arm's length a stone weighing 5 lb. 2 oz. Arrows show attacks of malaria. Both patients were taking ten grains of quinine daily during the period for which the curves are shown, but not previously.

"Chart I.—The patient had an attack prior to admission to hospital. He had not been taking quinine. Considerable weakness resulted. A quick recovery followed; a level, which appears to have been normal for the individual, being reached in a few days. Two subsequent attacks, much modified by quinine, had an appreciable but not serious effect. This is a normal response for patients who suffer but little between attacks.

"Chart II.—A patient, previously a stoker, who had no definite attacks but continual headache and tremor; parasites were present before admission. Very slow recovery and very low level, even after three weeks.
the spleen was not enlarged to palpation. The picture of malarial cachexia is thus indistinct and of little value. It certainly does not represent the sort of chronic malaria with which a practitioner will have to deal when the troops come home.

**Conclusions.**

The signs and symptoms set forth above have been arrayed in the hope that they will aid the practitioner in answering a particular question—viz., the query of an employer as to whether one of his employees has recently suffered from malaria as he claims or whether, on the other hand, he has just been having a day off for what we shall probably continue to describe as "urgent private affairs." To recapitulate, the following may be looked for: (a) Rigor, hot stage, sweating; (b) periodicity of symptoms or pyrexia; (c) perisplenitis; (d) pain in the side, confirmed by hyperalgesic areas; (e) headache, similarly confirmed; (f) pharyngitis, conjunctivitis; (g) hyperidrosis; (h) pigmentation; (i) Raynaud's symptoms; (j) transient oedema; (k) parasites in the blood; (l) pigmented cells, excess of transitional or eosinophil cells, endothelial cells and polychrome red cells of abnormal size in the blood; (m) tremor; (n) herpes labialis; (o) jaundice; (p) tachycardia; (q) weakness. These are all wholly or partially out of the patient's control and the finding of several of them would be very strong evidence in support of the employee's statement. Furthermore they represent a syndrome which is not met with in any other disease, although trench fever is obviously a very close ally and presents many of them.

Many other symptoms which are too unusual to include have been met with and many have been described by other authors but have not been seen by myself. Some symptoms have been mentioned incidentally but not fully described—e.g., leg pains I have encountered in such circumstances that the influence of malaria could not be denied; transient hemiplegia, tetany, exophthalmos, purpura, bleeding from the gums, epistaxis, and albuminuria (probably very frequent after attacks), severe gastritis, diarrhoea, enlarged glands. Other authors have described mastitis, urticaria, a specific rash and extensive oedema. These I have not seen.
DISCUSSION.

Lieutenant-Colonel Andrew Balfour, R.A.M.C., C.B., C.M.G.: These careful clinical observations are the very kind of thing we require in malaria, for we are too apt to rush, as it were, to the blood and form our conclusions on such factors as an increase of large mononuclears or of those transitional cells of which Dr. Gordon Ward spoke, or on the presence of pigment-bearing leucocytes, &c. Those accustomed to live and work in tropical regions are familiar with those larval types of temperature to which Dr. Ward has directed attention, and, like him, we realize that they are frequently the precursors of a well-marked relapse. The most important point about them is their periodicity, which should always excite suspicion whether it be of the tertian or quartan variety. I take it that these small fluctuations of temperature which may not cross the normal line, really indicate a sporulation of parasites in the internal organs. As regards the apyrexial rigor, I think the rectal temperature, if taken, would very likely be found elevated. In addition to those mentioned by Dr. Gordon Ward, there is another clinical sign which should be looked for in cases which have more or less recently passed through an attack of malaria, and that is a distinct hepatic tenderness on deep palpation under the right costal margin. We are, perhaps, too apt to think only of the spleen in relation to malaria and to forget that the liver is also frequently affected. I agree with Dr. Ward that the cells he found in the blood films are endothelial cells. They are not pathognomonic of malaria, and are not infrequently found in other febrile conditions in the Tropics, more especially in kala-azar. It is, of course, a little difficult to be sure that all the different symptoms to which Dr. Ward refers were really due to malaria. I think it quite possible that some of them, such as, for example, the localized oedemas, may really be evidence of a kind of sub-scorbutic state. In Macedonia, as in the other Eastern war areas, the dietetic conditions were naturally not always of the best. Many men suffered from debility. In part, this was to be attributed to deficiency in certain articles of diet, and it is quite probable that this debility left its mark on some of the men, as shown by such clinical signs as Dr. Ward has described. Still, considering the whole of these signs, together with the histories of the cases, I have no doubt that the clinical symptoms noted have been helpful in enabling a correct diagnosis of previous malaria to be made.

Dr. F. S. Langmead: It is a very common fault to ascribe all symptoms occurring in people coming from a malarial country or in malarial subjects to the malaria itself. A more patient examination will sometimes show that a patient who is regarded as suffering from an unusual form of malaria is really the subject of an independent or secondary infection. Despite this warning, I agree that malaria is almost as protean in its manifestations as Dr. Ward
Ward: *Apyrexial Symptoms in Malaria*

has declared. I have had facilities for observing about 10,000 cases of malaria, and agree as to the occasional occurrence of rigors without fever. Such rigors are frequently accompanied by malaise, headache, sweating, rapid pulse, mental depression, and even by vomiting. Cases have been seen of all degrees, ranging from those with rigors without alteration of temperature, through those with rigors where the temperature only attains the normal line and those where the rigor is associated with a very slight pressure, up to those with characteristic pyrexial attacks. Pigmentation may be so severe as to resemble that of Addison's disease, and with this I associate also cases with temporary or persistent low blood-pressure and small pulse, possibly ascribable to suprarenal defect. Defective action of this gland and also of the thyroid is suggested by the tachycardia, tremor, and exophthalmos, which have been referred to and which certainly occur. I have come to regard tachycardia in malaria as occurring in three clinical forms: (1) a transient form abating with or soon after an attack; (2) a more persistent form which subsides after several days or weeks; and (3) a chronic and protracted form which possibly will remain permanent. It is impossible to say when the last two forms are indications that the malaria is merely in abeyance. Statistics as to the efficacy of various forms of treatment in preventing relapses are unreliable, as what constitutes a relapse is often decided by rule of thumb, and apyrexial attacks are not considered.
Experiments on the Complement Fixation in Malaria with Antigens prepared from Cultures of Malarial Parasites (Plasmodium falciparum and Plasmodium vivax).¹

(WAR OFFICE RESEARCH.)

By J. GORDON THOMSON, Captain, R.A.M.C.(T.C.).

(Protozoologist to London School of Tropical Medicine; Officer in Command of Malaria Laboratory, No. 4 London General Hospital.)

This research was carried out for the purpose of finding a more accurate test for cases of malaria. The only certain method of diagnosing this disease is to find the parasites in the peripheral blood; this is only possible when the infection is heavy enough to enable the pathologist to find parasites after a reasonable search in a thick film.

Many patients are admitted to hospital with a diagnosis of malaria on clinical grounds only, and many have received treatment with quinine before they have been examined for parasites, the result being that a large proportion of cases are labelled as malaria on the history of the case alone. A differential leucocyte count where no parasites are found is of great assistance in diagnosing these, and when the large mononuclears show a marked increase we can be fairly certain that the case is one of malaria, especially when there is a marked anaemia and splenic enlargement, with a definite history of rigors and temperature. The blood picture, alone, however, with the absence of malarial parasites always leaves us in some doubt whether the case is actually one of malaria, especially if no parasitic relapse occurs while the patient is under observation without treatment.

Many soldiers in the Tropics have been diagnosed as cases of malaria which are not malaria, and it is a common error to send patients into hospital diagnosed as typhoid which are proved afterwards to be malaria. Comatose malaria has again and again been confused with heatstroke with disastrous results. A golden rule ought to be framed by physicians in a tropical country—namely, in all cases of obscure fever, before

¹ At a meeting of the Section, held January 28, 1919.
commencing treatment examine a blood film for parasites, and if that is negative have a blood culture done. If this were carried out many mistakes would be avoided. On treatment by quinine (10 gr. t.i.d.), malarial parasites rapidly disappear from the peripheral blood so that about the fourth day asexual forms are so scarce that as a rule they cannot be found even after prolonged search.

Many methods of treatment have been tried, but there is no accurate means of diagnosing whether the patient is absolutely cured or not. We know that treatment must be continuous and prolonged if relapses are to be avoided, but we are never able to guarantee that the fever will not return. If a disease is not cured and a specific organism is still present in the body in large enough numbers to produce a certain quantity of antibody, we know that if we can produce a specific antigen it ought to be possible to deviate the complement. The strength and sensitiveness of this reaction depends on the presence of sufficient antibody and its specific antigen. Little is known about the antibodies formed against the malarial parasites, but there must be some, and there is no doubt these play a great part in the production of a natural immunity, and eventually many cases of malaria recover without treatment, usually after many attacks of fever over a prolonged period.

Immunity, though not quite absolute, is established to a certain extent and in a specific complement-fixation reaction we are able to measure the defensive activity of the infected host. Complement is deviated or fixed when antibody and its specific antigen are present in the test, and in this series of experiments we have prepared a malarial antigen successfully in the following manner:

Preparation of Malarial Antigens.

In a preliminary note published in the British Medical Journal (1918) [2] we described shortly the technique for making antigen, so that other experimenters might work along similar lines and perhaps improve the method. Cultures are prepared of malarial parasites, either benign or malignant tertian according to the method elaborated by J. G. Thomson and D. Thomson (1913) [1]. Cases in which the peripheral blood is heavily loaded with parasites are to be preferred, and, if possible, they should have received no previous quinine treatment. After growing in the incubator at 37° C. for twenty-four to forty-eight hours, the supernatant serum is pipetted off and an excess of sterile
distilled water is added to the remaining sedimented corpuscles in the tube. Shake well up and so lake the corpuscles. Centrifuge and pipette off the red haemoglobin-stained water, so leaving a deposit of débris of corpuscles, leucocytes and parasites. Add again an excess of distilled water, shake up and centrifuge and so on until the supernatant water is practically colourless and thus the whole of the haemoglobin is removed. We now have a deposit of malarial parasites, and if this deposit is smeared on a slide and stained, we find, if the culture is a good one, that we have almost a solid mass of parasites with some leucocytes and the empty envelopes of red blood corpuscles. Dissolve this sediment in as small a quantity as possible of N/10, NaOH, and a viscid yellow fluid is obtained. Neutralize this carefully with normal HCl. If too much acid is added we cause a precipitate, and this must be avoided.

So far we have found this concentrated solution of parasites too anticomplementary to use as it is, and dilution is necessary with normal saline. The ideal aimed at is to obtain an as concentrated an antigen as possible which is not too anticomplementary, for it is by getting a strong antigen that we will expect to get more sensitive and reliable results.

Dilute this antigen ten times with normal saline and carry out a preliminary titration with complement (guinea-pig's serum). The actual test is that recommended by D. Thomson (1918) for gonorrhoea [3]. Take twelve Wassermann tubes and add 0·1 c.c. of guinea-pig's serum (complement), diluted as follows:

\[
\begin{array}{cccccccccccc}
(1) & (2) & (3) & (4) & (5) & (6) & (7) & (8) & (9) & (10) & (11) & (12) \\
1/10 & 1/20 & 1/30 & 1/40 & 1/50 & 1/60 & 1/70 & 1/80 & 1/90 & 1/100 & 1/110 & 1/120 \\
\end{array}
\]

= dilutions of complement.

Add to each 0·1 c.c. of antigen (diluted 1 in 10). At the same time when testing each new batch of antigen it is better to conduct similar tests, the antigen being diluted 1 in 20, 1 in 30, 1 in 40, 1 in 50, 1 in 60, 1 in 70, 1 in 80, 1 in 90, and 1 in 100. Thus we can find without waste of time the dilution which has not too much anticomplementary action. This complete test will take, therefore, ten rows of tubes, but it can be made shorter by making jumps—for example, the antigen could be tested 1 in 10, 1 in 40, 1 in 50, and if found to be unsatisfactory in these dilutions, higher dilutions could be carried out later.

Having added the complement and the antigen to the tubes, place
them all in the ice chest for at least one hour, and then give half an hour in the water-bath at 37° C. Add to each tube 0.1 c.c. of sensitized sheep's corpuscles (3 per cent. suspension). Place the trays immediately in water bath at 37° C., and read the result in fifteen minutes. The lowest dilution of complement which produces complete haemolysis in that time should be taken as the minimum haemolytic dose. Note the dilution of antigen which gives a satisfactory result, and dilute antigen accordingly. As I have pointed out, the stronger the antigen the better, provided it is not too anticomplementary. So far I am quite unable to explain why some antigens are so anticomplementary, but further experimental work may throw some light on this.

The actual test of the malarial sera is based on the technique of Captain D. Thomson (1918). Four tubes, A, B, C, and D, are employed for the serum of each suspected malaria. To A, B, and C, add 0.1 c.c. of serum (inactivated at 56° C. for ten minutes) and diluted 1 in 10 with normal saline. Sometimes it is better to dilute the serum only 1 in 5. To tube A add complement 0.1 c.c., containing three and a half minimum haemolytic doses. To tube B add complement 0.1 c.c., containing three minimum haemolytic doses. To C add complement 0.1 c.c., containing two and a half minimum haemolytic doses. To D add complement 0.1 c.c., containing two and a half minimum haemolytic doses. To A, B, and C add 0.1 c.c. of antigen, preferably a compound antigen made up of different strains of parasites and diluted according to what has been found necessary after titration against the complement used. To tube D add no antigen, but 0.1 c.c. of normal saline, so that this acts as a serum control. Place all the tubes in the ice chest over-night and add to each next morning 0.1 c.c. of sensitized sheep's corpuscles; place immediately in the water-bath and read the results quickly in exactly fifteen minutes.

With each batch of sera tested, introduce at least two known positive malaria sera and two known negative sera, that is the sera of persons who have never had malaria and which we know possess no anticomplementary action per se. As soon as the control tubes D show complete haemolysis and the negative sera show haemolysis, the results in A, B, and C ought to be read and noted at once and also at the end of fifteen minutes. If left longer, and the reaction is a weak one, we fail to detect weak positive reactions. An antigen control containing no serum but an equal amount of saline and two and a half minimum haemolytic doses is necessary, since antigen may absorb complement
during the night in the ice chest. If control sera and antigen are unsatisfactory it is quite impossible to read the test, and the experiments will be required to be repeated. As the reaction is a delicate one, and requires to be very carefully controlled, we avoid using old serum, which is perhaps contaminated, and thus develops anticomplementary power, apart from any specific antibody in it. Sera previously heated and kept for a few days, must be heated again immediately before the test.

Results of Experiments on the Complement Deviation on 200 Cases of Malarial Infection.

It was first necessary to test the sera of known malaria cases with parasites present in the blood in order to find out if positive results could be obtained with the antigen used. Antigens were prepared from single cases of malaria and used unmixed with other strains. Separate antigens were prepared from benign and malignant tertian malaria, and also from the spleen of a malignant tertian case. This latter method of obtaining antigen may prove to be the best, but, unfortunately, we have not yet been able to obtain a heavily infected spleen.

The spleen antigen prepared here did give positive results, but these were weak and unsatisfactory. The actual experiments carried out were as follows:—

(A) Preliminary Experiments carried out in known Cases of Benign Tertian Malaria, all with Plasmodium vivax present in the Blood, using Antigen prepared from a Single Strain of Parasites (Plasmodium vivax).

All these cases were done with an antigen prepared from a single strain of Plasmodium vivax (a Salonica strain). Seven out of the nineteen gave only weak positive reactions, and one serum gave a completely negative result, although the blood contained numerous parasites, the patient having had a rigor on the day previous. The remaining eleven cases gave positive results, and all, after standing in the tubes for a short time, became negative. For this reason the results must be read quickly after the control serum and the known negative sera show complete haemolysis.
The serum which gave a completely negative result must have been either very deficient in antibody or the antigen used was not specific to that particular strain. We shall discuss this later under a separate section.

(B) Experiments carried out in order to determine if Separate Strains of Parasites react differently to Various Antigens.

In this series of observations an attempt was made to determine if some antigens were specific to certain strains of parasites. The cases under discussion were all benign tertian, and all at the time of examination had parasites (Plasmodium vivax) present in the blood. It was thought that strains from Salonica, Mesopotamia, Egypt, East Africa or Palestine, might react differently with different antigens. This point we have been quite unable to settle, because it is possible that the real cause of these varying reactions is due to slight differences in the preparation of the antigens.

It is evident from these experiments that some antigen gave a much stronger reaction with these cases. This may be due to the fact that the antigen was stronger, or to the strain of the parasites used in its manufacture.

(C) Experiments conducted for the Purpose of determining if Specific Antigens could be prepared for Benign and Malignant Tertian Malaria.

The first two experiments conducted led us to believe that the reaction was a specific one, and we are still in doubt about this matter, as it is extremely difficult to determine whether or not a patient may at one time or another have been doubly infected. Experience of over 3,000 infections shows us that it is quite common for a patient to have a primary infection with Plasmodium falciparum and afterwards to return to hospital with a Plasmodium vivax infection, which seems to persist longer.

As there is frequently no record of the parasites in previous attacks, it is quite impossible to exclude double infections. Difficulties also in the sensitiveness and strength of the antigen prepared also play a part in these observations.

This series of experiments show that there may be a certain
specific action, but it also tends to show that there is probably a group reaction for malaria. Experiments conducted by Noguchi and Craig seemed to prove that there are specific group reactions in the way of complement fixation between Treponema pallidum, Treponema pertenue, and Treponema microdentium, as antigens prepared from pure cultures of these may give complement fixations in the presence of syphilitic serum.

(D) Further Experiments made by using an Antigen prepared from Spleen of a Malignant Tertian Case and Antigen prepared from Cultures of Plasmodium vivax.

These experiments show that certain cases give a specific reaction to the infection found, but in other cases a positive reaction is obtained when using either antigen.

(E) Series of Cases done using a Compound Malaria Antigen composed of ten cultures of Benign Tertian Malaria (Plasmodium vivax).

Cases of benign tertian were chosen which had been infected in different zones; the following strains of parasites were added in as far as possible equal quantities, to the same tube and mixed:

<table>
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<th>\textit{Plasmodium vivax}</th>
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<th>Salonica</th>
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<td>&quot;</td>
<td>...</td>
<td>Palestine</td>
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It was found necessary to dilute the original solution of the parasites 1 in 100 with normal saline in order to get rid of the anti-complementary action, and as a result our antigen was very much weakened. With this antigen we have obtained positive results with a large number of cases, both benign and malignant tertian malaria, all of which had parasites in the peripheral blood at the time the serum was examined. One case, a benign tertian, gave a completely negative result, indicating that there must have been extremely little antibody
present in the serum. Negative results in such patients are difficult to explain, but it is quite possible that at certain stages, even when parasites are present in the blood stream, antibodies are scarce, probably because they have not had time to form, or because the patient was deficient in reaction to the parasites.

(F) Experiments on Complement Deviation in cases of Malaria under various courses of Quinine Treatment.

These experiments show that it is extremely difficult to estimate what is a sufficient treatment for any individual case of malaria. Most of the patients had many attacks of fever and numerous admissions to hospital. On each admission they were treated with quinine and discharged after varying intervals.

A study of the results obtained in complement deviation shows that certain patients react very rapidly to the treatment, but others remain positive in spite of prolonged dosage with the drug. It would seem to us that we have here a parallel in syphilis, where it is well known that the best results are obtained by attacking the disease during the primary stage, and complete cure is not uncommon. If, however, the disease becomes chronic, in many cases it is incurable. Inefficient treatment of malaria during the initial attack only leads to difficulties and makes the condition much more resistant to cure afterwards.

Patients who come into hospital with malaria undergo a course of treatment with quinine and are discharged; some of these return after varying intervals with a fresh attack, but others disappear and seem to carry on without relapsing, or at least without having sufficient fever to make them ask admission to hospital. It was noted that many of the above cases gave a positive result after very prolonged treatment, and that others became negative, but so far we have not been able to trace whether the patients relapsed or not. In nearly every instance where a patient had been free from malarial attacks for a period of one year, the blood was found by us to be negative.

(G) Experiments done correlating the large Mononuclear Count with the Complement Deviation.

A few observations were made correlating the large mononuclear-leucocyte count with the complement deviation, and it was found that
cases showing a marked increase of the large mononuclears in every instance gave a positive deviation of the complement. The presence of quinine in the blood in detectable quantities in the serum did not seem to affect the test for the deviation of complement. A large number of known cases of syphilis were tested against malarial antigens, and were found to give positive results, and it is therefore necessary to exclude this disease, which is easily done by having an ordinary Wassermann done on the serum.

In another section we show that the Wassermann reaction for syphilis is in no way affected by an attack of malaria.

Conclusions.

(1) It is possible to obtain a deviation of the complement in cases of malaria by using a specific antigen prepared from cultures.

(2) How far the test will prove to be of practical value in diagnosis is difficult to estimate as it is a delicate one and subject to great error, especially if it is performed by an unskilled observer.

(3) The effect of treatment seems to vary greatly, some patients react more quickly to quinine than others. This may mean that the parasite is more resistant in some cases, or that the body of the host is able to react more strongly in certain individuals when quinine is administered.

(4) The method of preparing antigen is a new one; it is not a watery nor alcoholic extract but a complete solution of the whole parasite, and we would thus expect it to contain the specific substances necessary for a good reaction in the presence of antibody.

(5) Positive reactions are obtained with syphilitic sera, but this is probably due to lipoidal substances and is not in any sense a specific reaction.

(6) The experiments conducted so far point to the possibility of a specific reaction for benign and malignant tertian malaria, and there is evidently a group reaction as well. It is thus better to use a compound antigen made up of many different strains of malarial parasites.

(7) To every batch of ten sera tested it is advisable to introduce two known negative sera which are known to have no anticomplementary action per se.

A—6a
REFERENCES.


Further Notes on the Epidemic (Influenza), with Special Reference to Pneumonia, in Macedonia.

By Julius Burnford, M.D.

In "A Note on Epidemics" published in July, 1918, I pointed out some peculiar aspects of the epidemic in its inception. Since then unexpected opportunities of studying the epidemic in different centres have presented themselves. The observations arising therefrom are such that it seems to me to justify elaboration of these earlier remarks, and the placing on record of one's findings whilst the picture is vivid.

It is difficult at the moment to place the epidemic in proper perspective, and indeed the picture is as yet incomplete, for though the acute stage is now over we are faced with the possibility of sequelæ for some time to come. Some of these are even now obvious, and more especially those due to disintegrative or non-reparative changes in the lungs—some severe and presenting no difficulties in interpretation, others milder and indefinite, but likely to interfere more or less seriously with the life and work of the individual.

Material on which observations are based.

This has been confined to soldiers. My movements have brought me by chance into military districts at the time coinciding with the beginning of the epidemic. In France in the spring of 1918 the

1 At a meeting of the Section, held April 29, 1919.
earliest cases of a mild type, which I have described as a peculiar glandular type, were admitted in large numbers to the isolation wards under my care. Whilst doing duty at the Royal Herbert Hospital in June I noted at the beginning of the epidemic in that area numerous cases of a more severe and mixed though not very fatal type. Whilst in charge of the Medical Division of the Sixty-third General Hospital in Salonica in September, the wards were filled with cases in all grades of severity, many of them fatal. And when moving with the Army of Occupation to the Dardanelles in December a fresh and severe outbreak came under my observation in a number of Anzacs who had come from the Jordan Valley, where there was a shade temperature of 120° F., and who were now encountering adverse conditions of transport and climate on a bleak and exposed spot on the Peninsula. With these must be included a sprinkling of men who had come in January under similar circumstances from Bangalore, though these had stopped at Salonica en route, and had left the mass of their severe cases in hospital there. It is possible that these acquired the infection whilst quartered in a depot at Salonica. Some officers, who had been taken prisoners at Kut and had been confined in Asia Minor until the Armistice, had detailed in their diaries an account of an epidemic amongst the inhabitants and themselves in a village of Yosghad, which began on October 4.

To supply statistics would only be misleading. The physique and previous health of the men varied in different districts. In Salonica the men were often much debilitated, and furthermore the outbreak coincided with the season of malaria, which confused the diagnosis in many cases. Here the mortality was great, and out of 100 consecutive cases of pneumonia as many as fifty died. In the Dardanelles, out of about 900 Anzacs, over 150 were admitted with influenza; of these twenty-five developed pneumonia and twelve died.

Exceptional opportunities for observation present themselves in a general military hospital, and especially in Macedonia, where evacuation was necessarily slow—and patients were kept till convalescence was advanced. In some cases the disease actually developed in patients convalescent from other diseases. At the same time we must deplore many lost opportunities for research owing to the exigencies of the service. Indeed at the Dardanelles, in the absence of a pathological laboratory, it was necessary to carry on the work with the aid of a small travelling microscope and some stains acquired from a hospital ship and the local Turkish hospital.
Path of Infection.

It would seem that infection is carried by human traffic, judging from the times at which the epidemic originated at different parts of the world. Cases often fatal were evacuated from the leave trains en route. Amongst the Anzacs the disease broke out on a crowded transport, on which they were detained for several days before disembarking. But it is difficult to account for the outbreak in the middle of Asia Minor—120 miles east of Angora—unless as was suggested it had been brought from the Black Sea by caravan.

There can be little doubt that overcrowding and poor ventilation are potent factors in the spread, and these are difficult to avoid in transports and troop trains.

Clinical Manifestations.

The condition is essentially a toxæmia, which exerts its activity primarily and principally on the respiratory system, and to a much less degree and much more infrequently on the gastro-intestinal tract and occasionally on the nervous system. The milder cases present a striking similarity of symptoms: sudden onset with malaise and pains in head, back and limbs, pyrexia and intense congestion of pharynx and upper air passages without any increase in pulse-rate, and terminating in two or three days by crisis or brief lysis.

Increasing grades of severity are associated with more extensive involvement of the respiratory tract from above downwards. In the most serious cases there is general affection of the lungs, and a most fatal type of pneumonia in which the toxæmia symptoms are complicated by characteristic manifestations of pulmonary obstruction. In all cases however the respiratory tubes and tubules are first affected, and it is on the mucosa that the toxin particularly acts. Hence it is possible to pick out from a series of cases types in order of severity, though the types often blend. Clinically we find (1) pharyngitis with or without implication of the mucosa of the nose and sinuses; (2) laryngitis; (3) tracheitis; (4) bronchitis; (5) bronchiolitis; and (6) pneumonitis with involvement of all the lung tissue.

In any case the disease may terminate by crisis or rapid lysis, as in the mild type, after a few days. Occasionally there may be hoarseness or loss of voice for some time after the larynx has been affected, and some debility. But in the earlier cases seen in France this was not
a noticeable feature, and the men were returned to convalescent camp in a few days.

Further points relating to the milder cases are indicated in the charts appended, and it is not proposed to deal further with them here, save in one particular. The associated adenitis which was so marked a feature in the earlier cases as to suggest a diagnosis of rubella was not so much in evidence in the later cases, though the patients not infrequently exhibited many hard enlarged glands in the cervical, axillary, and even inguinal regions. The significance of this will be discussed later.

![Chart I](image1)

**Chart I.**—Two-day type with adenitis.

**Chart II.**—Adenitis. Rapid recovery. No leucocyte increase.

*Note. — The dotted line on the charts indicates the leucocyte content of 1 c.mm. blood.*

**The Pulmonary Type.**

The manifestations of this type are so varied in their clinical detail that it is difficult to give a general description. But it may be said that in the main the symptoms fall into two groups: (1) toxæmic, (2) mechanical, though the more or less asphyxial state produced thereby presents symptoms closely resembling some due to the toxæmia. Again the picture varies in different outbreaks. Sometimes the toxæmic symptoms predominate, producing a drowsy and lethargic state with indefinite physical signs of pulmonary disease. At others the obstructive lesions, such as coughing, cyanosis, and asphyxia, are more manifest. This variation in type has been noted frequently. In
Salonica there was a period when the incessant and for the most part unproductive coughing created a veritable and painful din during the night. But in the Dardanelles there was throughout an ominous quiet in the wards. In the diary of the Kut officers it is noted that the roaring and barking of the gasping dusky patients could be heard far from the huts.

The physical signs of involvement of the lungs are often most misleading at the outset; in a few days there may be definite evidence of consolidation of one or more lobes, but at the same time the entire lung tissue is in all probability affected. A central pneumonia may gradually reach the surface and present signs of consolidation.

A case may simulate middle-ear disease or cerebral disease during the first few days.

Physical signs must be interpreted in the light of general symptoms and with care; indeed it must be said that the physical signs of pulmonary involvement are of less importance than the objective signs and general condition of the patient.

*Symptoms in Detail.*

The recumbent position is usual, and only rarely is there any desire on the part of the patients to sit up. Indeed this is the only position of comfort, and attempts to make the patient sit up for examination may only lead to paroxysms of coughing. Some are particularly tranquil and cheery throughout, others have a sense of impending death which is nearly always justified.

*Cyanosis* is a frequent and pronounced feature. It is apparently in direct relation to the extent of the pulmonary mischief, and indeed is often a better index of this than the physical signs themselves. It is of great prognostic value. In the earlier stages and often during the long convalescence it is of a lighter hue, the cheeks may be flushed, whilst the ears are deeply cyanosed; later the cyanosis becomes more intense and universal, and in the still later stages there is a dusky black complexion—a condition that suggested to one medical officer the name of the black death. It is evidently symptomatic of an asphyxial state, and not connected with the toxaemia. It is only found in connexion with pulmonary involvement, and varies directly with this. It is increased by the most trivial exertion, such as coughing or drinking, or even talking, and in the convalescent can be excited by exertion. Though of grave significance, deeply cyanosed cases can and do recover
—often after many weeks. In some stages it produces the impression that the lungs are working to the limit of their reserve power, and in convalescence it must be regarded as an index of lung damage, and as an indication for the restraint of unnecessary exertion. Attempts to relieve cyanosis by venesection have failed, and indeed the evidence is against the cyanosis being of cardiac origin. In a few cases, however, cyanosis occurred in association with distended cervical veins and appearances of cardiac dilatation; these were occasionally relieved by venesection. The blood in severe cases is black and sometimes even tarry. In the most extreme case the patient died with asphyxia and severe agonal convulsions, he was black in colour, and the pulse remained good to the end.

Respiratory Distress.—The respirations are increased out of proportion to the pulse-rate, which in fact may remain normal. A rate of 40 per minute is common, and in some it may be even 60; in one case it persisted at 90 for a day before death. In the early stages it assumes an emphysematous type, the expirations being prolonged; and with increasing severity of symptoms the expirations become more and more prolonged in comparison with inspirations; or else there may be gasping and quick panting with hardly any movement of the chest wall, into which indeed little air enters. In other cases the breathing may be noisy and roaring in character, as if purely tracheal in origin. In many cases there is actual distress with or without exertion. It is important to note that (1) it is only in the pulmonary type of disease that these respiratory variations occur; (2) venesection fails to give any relief; (3) histological examination reveals destruction of the aërating tissue sufficient in amount to account for the symptoms; (4) the condition is often relieved, though only temporarily, by the administration of oxygen, which should be continuous if possible; (5) alteration in the position of the patient produces no improvement.

The respiratory changes must then be regarded as indicative of pulmonary mischief, and as such are of prognostic import. The combination of intense cyanosis and rapid breathing is of grave moment.

The Pulse.—As in milder cases the pulse-rate in the pneumonic type may be slow, but often is raised to 100 and sometimes more, but not in proportion to the respiratory increase. Only rarely is there dilatation of the heart and in grave cases the pulse-rate may be over 120. Death does not occur from cardiac failure, and in one series of cases at autopsy the myocardium was firm though in the Anzac
series there was distinct evidence of myocardial degeneration. In some cases indeed the pulse was good until the end and in one it was even felt for a brief period after respirations had ceased.

Other Symptoms. — Occasionally cerebral symptoms are seen. Sometimes delirium is an early symptom, in others it is a late one. Lethargy is characteristic of the toxæmia. Deafness may arise early, sometimes also indefinite signs of mastoid disease. Vomiting is frequently met with accompanied by furred tongue at the outset of the illness and diarrhœa occasionally. Albuminuria of slight degree often accompanies the febrile disturbance. Haemorrhages are not uncommon. Epistaxis may occur in the early stages. With the cough there may be pinkish (blood-stained) sputum; the typical rusty sputum is rarely seen. Occasionally the sputum contains dark coloured blood and clots for a time and sometimes more extensive haæorrhage results, with bright red blood at the onset. The intense engorgement of the mucous membranes and of the pulmonary capillaries and also the definite infarction sometimes seen will account for these symptoms. In two cases there was definite ulceration of the vocal cords. In several cases extensive haæorrhages were noted in the sheaths of the rectus abdominis muscles at autopsy.

Physical Signs.

These are anomalous and vary even in the same patient from time to time. They appear to be the result of obstruction to the bronchioles with more or less surrounding consolidation intermingled with areas of emphysema. The signs vary with the accumulation or evacuation of the secretion of the bronchioles, so that at one moment the lung may be silent and at another may present signs of consolidation. The most characteristic and frequent finding is a fine crepitation heard over a considerable area, often more marked on deep respiration or after coughing. Sometimes the signs are slight and suggest a central pneumonia with relaxed or emphysematous lung surrounding, and in a few days the consolidation reaches the surface with characteristic signs. But only occasionally are there definite classical signs of lobar consolidation; usually the mixture of lobular consolidation and emphysema produces indefinite signs. Hence we find varying signs as follows: (1) Crepitations; (2) Crepitations with absolute dullness; (3) crepitations with impaired percussion note; (4) crepitations with whispering pectoriloquy with or without tubular breathing; (5) silent
lung; (6) choked lung, into which little air enters with great difficulty, relieved sometimes on coughing; with resolution the sounds become coarser and râles appear. It may be taken that generally signs of "choking" of lungs or even spreading consolidation will be found posteriorly and laterally whilst anteriorly there is evidence of emphysema in a hyper-resonant lung with harsh breathing. The middle lobe is frequently implicated with the lower lobe. Occasionally, but rarely, the upper lobe on one side is consolidated with or without similar lesions in the lower lobe. Sometimes signs of spreading pneumonitis (for this is perhaps a better term than the more specific designation of pneumonia) are noted and the lesion beginning at the base may implicate in time the greater part of both lungs. At times one part may show resolution whilst another becomes affected, as in pneumonia migrans. But, as has been pointed out above, the signs of localized consolidation are always misleading inasmuch as the entire lung tissue is probably affected. Occasionally, but rarely, pleural effusion occurs even with whispering pectoriloquy and tubular breathing in connexion with absolute dullness, a condition verified by exploratory puncture.

Significance of Physical Signs.—There is little relation between the signs enumerated above and the severity of the disease. A fatal case may present crepitations throughout a brief illness. But perhaps a definite and early appearance of lobar consolidation may be more hopeful than signs of diffuse lesions. Here it is important to note that the signs of lobar consolidation do not necessarily imply lobar pneumonia. Clinically the following occur: Bronchitis, pneumonitis, broncho-pneumonia, massive consolidation, effusion and emphysema.

Sputum.—The sputum and the cough are very variable. Usually there is little sputum in the early stages; sometimes it is blood-stained and pinkish, rarely rusty; sometimes it is tenacious; sometimes it actually contains blood. There may be no cough, or a dry cough, or at times paroxysms of barking with little expectoration which often is only brought up with difficulty. In the later stages the cough weakens and the act is painful. Often the cough throughout is insignificant. In some cases large quantities of highly albuminous glairy fluid containing whitish muco-purulent matter are expectorated. With resolution the sputum changes to nummular type but is not profuse, and with breaking down lung tissue it becomes actually purulent. Quantities of purulent matter may be expectorated for several weeks in cases which die, showing evidence of suppurating and breaking down of large areas of lung tissue.
Progress.

In favourable cases the acute symptoms end by crisis in some, by lysis in most. But though it would seem that the toxaemia has been overcome the pulmonary lesions do not immediately resolve. Indeed resolution may be prolonged for weeks and is always slow. Consequently even when the temperature falls by crisis the respirations remain increased and only regain their normal rate with improvement in the pulmonary state. Similarly cyanosis may persist for a long time and be accentuated by slight exertions; hence a long convalescence is indicated.

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Chart III.


The silent and unresolved lung may persist for weeks and perhaps indefinitely. But the future state of the unresolved lung remains to be followed up. The affected side may be dull and the signs simulate fluid. In these cases a pyrexial curve is sometimes obtained, in some slight, with rise of one or two degrees, in others more marked. In one case (Chart XI, p. 63) the rise was only noted at 6 a.m. each day.

Where there is actually pleural effusion a serous highly albuminous fluid and occasionally thick purolymph may be evacuated. The unresolved lung may undergo further changes, either reparative with appearances of crepitations and coarse sounds accompanied by a
Burnford: Further Notes on the Epidemic in Macedonia

**Chart IV.**


**Chart V.**

Lobar type of consolidation developed during convalescence from malaria. Resolution delayed. Much cyanosis and respiratory distress. Leucocytosis (18,000).
disappearance of the radiographic shadow; or disintegrative, with abscess formation. Signs of cavitation with purulent expectoration and hectic temperature may herald a fatal result (Chart VII, p. 60). Or organization may lead to healing and recovery after a long convalescence. In this connexion it is justifiable to anticipate conditions of bronchiectasis and chronic interstitial fibrosis arising in the unresolved lung, but this remains to be seen.

The voice when lost often does not return for weeks and occasionally there is actual ulceration of the cords. In the fatal cases, from which alone is it possible to surmise the course of events in those that recover, the processes are all more intense. Death may occur from acute oedema of the lungs at the beginning of the illness, associated with respiratory distress, cyanosis and much frothy (in some cases blood-stained) fluid welling up and exuding from the lips and nostrils: the patient actually dying from drowning (Chart XVI, p. 69). In others there is more obstruction and asphyxia with little expectoration, but the complexion becomes dusky purple or black, and agonal convulsions accompany death.

Again, there may be more evidence of resistance and reaction on the part of the patient, but the lesion attempting to clear from one part spreads to another (Chart VIII). Here a surprising respiratory compensation is manifest, for with only a portion of lung tissue still
Malaria frequently. Both lungs affected; left more widespread, main symptoms however were cyanosis on exertion and respiratory distress; he felt well and could sit up and got out of bed when unattended. Autopsy: Universal consolidation, disintegration and suppuration, both lungs; left lung much enlarged and spread into right side of thorax, with large abscess cavity in upper lobe. Heart muscle firm. Sputum at first mucoid and profuse, became greenish and nummular and later purulent.

Wandering type of pneumonia spreading from left to right lung.
Cyanosis. Recovery.
aerating, life is often maintained for days notwithstanding that the acting lung tissue is itself highly emphysematous, so that respiration becomes more rapid, more laboured, the inspirations become gasps, the expirations drawn out sighs, and ultimately death results from exhaustion. The deeply cyanosed state with gasping noisy respirations may persist for hours and even days, belying all prognosis of immediate death.

Admitted with malaria; during convalescence developed pneumonitis both sides; then left lobar pneumonia and later pericarditis. Resolution was delayed, but pericardial signs cleared. Two months after admission there was no pericarditis, but left upper lobe still consolidated. Invalided, and still under treatment for unresolved pneumonia five months afterwards.

Pathological Findings.

These are significant of an acute irritation of the air passages, with a responsive reaction on the part of the mucosa. There is intense engorgement of the capillaries and an effusion of exudate into the lumina of the tubules and alveoli, accompanied by softening of lung tissue and a vicarious emphysema of the less affected parts. The trachea was frequently found to be deeply engorged and of a purplish tint, and in a few cases there was definite ulceration of the vocal cords. Briefly there occur tracheitis, bronchiolitis, consolidation, softening, and emphysema.

The stress in the first place appears to fall on the tubules, but the lesions are far more extensive than they appear at first sight at autopsy. Indeed there is actually present a generalized bronchiolitis which is to be seen even in the emphysematous and more normal parts. In more
advanced lesions there are peribronchitis with cell exudations, capillary engorgement and alveoli choked with serous fluid, and varying amounts of connective tissue and round cells with blood corpuscles. In many and in further advanced cases there are round cells, and, in some, collections of polymorphonuclear cells as in grey hepatization. Consequently areas of consolidation are found, either nodular and discrete, or

Brucho-pneumonia; cyanosis; toxæmia. Complicated by pleurisy and empyema. Pleural sac drained and washed out with eusol but ultimately resection of rib and recovery.
aggregated into pneumonia of lobar or massive distributions, and the picture varies with the intensity and stage of the reaction. Upon the chest being opened the lungs most frequently appear to be voluminous, bulging, pale, and emphysematous, filling the thoracic cavity. All their posterior and dependent parts have undergone oedematous consolida-

![Chart XI.](chart.png)

Consolidation of left upper lobe with general pneumonitis. Consolidation and cyanosis persisted throughout; signs of cavitation on thirtieth day; pyrexia only at 6 a.m.; general condition improved; invalided; still under treatment six months later with unresolved pneumonia. Sputum, at first nummular, became purulent.
tion, the substance being purple in colour. This process spreads more or less laterally and often involves the whole of the middle lobe. Rarely the upper and middle lobe are most affected, or there is a diffuse nodular consolidation of all the lung tissue. In addition there may be haemorrhagic areas or collapsed areas. In cases that die after a brief illness the consolidated process may not be evident, but there is engorgement and intense oedema.

**Chart XII.**

Influenza; relapse; left lobar consolidation; resolution delayed for weeks accompanied by cyanosis. Invalided; six months later at work in coal mine.
But, however varied may be the anatomical picture of broncho-
pneumonic consolidation, two conditions have been found at every
autopsy, and these are perhaps the more important factors—namely,
(1) universal friability of lung tissue, and (2) emphysema. The lungs
throughout, even in the parts apparently normal, have been soft and
friable, and have given way to the touch, often being of an almost
butter-like consistence. This is in no wise a post-mortem change, for
often the autopsy was done immediately after death. The emphysema
is both macroscopic and microscopic. In the anterior and uppermost
parts it is often very marked, and in a few cases large bullæ have been
found associated with the upper lobes and anterior border. Histologically
these areas show the fusion of alveolar walls usual in emphysematous
lung of long standing. But even in the consolidated parts one finds
microscopic areas of similar emphysematous nature.

It is to these two conditions that special attention must be directed;
the other changes may be considered of less importance. The pleura is
not often affected, occasionally it was seen to be dulled and injected over
more consolidated areas; pus was rarely, and fluid occasionally, present.
Not infrequently sub-pleural petechiae (Tardieu's spots) in large numbers
pointed to the asphyxial state.

The glands in the bifurcation of the trachea were in nearly all
cases much enlarged and deeply engorged; in some they were even
breaking down and diffusent to a less degree. The glands along the
trachea and main bronchi were similarly affected, and occasionally
the glands in the posterior triangle of the neck and the axilla. Histologically the bifurcation glands showed enormously engorged
capillaries with proliferation of endothelial cells and evidence of great
activity.

The heart showed varying changes. In a large number of cases the
muscle was firm and there was no naked-eye change, but in others, and
in all the cases amongst the Anzacs, the muscle was soft and friable,
and there was histological evidence of degeneration. In many the
cavities were filled with pale agonal and post-mortem coagula, but
dilatation was not noted. In a few cases of intense asphyxia the
cavities, especially on the right side, contained much blackish coagulum.
In two cases there was infective endocarditis, with large pendulous
vegetations on the aortic cusps and inflammation of the myocardium
at the base of these.

The remaining viscera showed the usual febrile changes, though in
many cases there was varying enlargement of the spleen, due to

ju—7
malaria, and in these the parasite had been found either in life or in smears of spleen at post-mortem.

Discussion of Post-mortem Findings.

It is evident that the specific changes occur in the lungs, and that the entire lung tissue is implicated. But the nature of the lesions is open to discussion. Is there a destructive activity exerted by the toxin on the lung tissue in the first place which facilitates the emphysematous change? Or is the widespread emphysematous change associated with so much destruction of elastic tissue as to produce of itself an undue friability?

These points have yet to be worked out, and in connexion with them one might recall here those cases of acute bronchiolectasis (with blistered lungs) occurring occasionally in children dying with bronchopneumonia.

Again, is the friability a condition compatible with recovery or is it invariably associated with fatal cases alone? This it is impossible to prove by direct evidence and the same line of thought has been applied to the condition of hepatization of the lungs in true croupous pneumonia. It would indeed seem impossible to expect resolution of so friable and emphysematous a lung as one meets with in the post-mortem room. But later investigation of cases in the light of these suggestions incline one to the belief that no case is hopeless until dead (see Chart XVIII, p. 73), and the recuperative power is indeed remarkable.

But, whatever may be the interpretation of these anatomical findings, there can be no doubt that the lungs are extensively damaged, and clinical evidence alone points to the permanency of the damage. This is the important factor to bear in mind in dealing with the many cases convalescent from the epidemic. It has been pointed out that the cyanosis persists long after recovery, and exertion increases this; many also complain of indefinite pains in the chest. Again we must add that it is time alone that will settle many of these points.

Reactionary Manifestations.

To determine the proper line of treatment it is necessary to appreciate the natural reaction of the system in those cases which recover, as well as the nature and action of the causal agent.
Notwithstanding that, after the failure to isolate Pfeiffer’s bacillus in earlier cases, many observers have found it in the majority of cases examined, there is still some uncertainty as to its being the sole causal agent. Nevertheless it is clear that we are dealing with a toxæmia of great and rapidly acting virulence, which exerts its activity on the respiratory tract. That it is not a septicæmia is evident; blood cultures for the most part have been sterile; some animal inoculations into rabbits with the blood of grave cases produced no general infection, indeed there is no evidence that the bacteria exist in the blood stream. It is obvious also that the toxæmia can be successfully resisted by the patient who recovers. An understanding of the processes at work must determine specific treatment.

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**Chart XIII.**

Admitted ninth day of illness with consolidation of right lung; developed empyema, followed by respiratory distress and death. Shortly after admission vomited round worm. Leucocytosis (45,000). Autopsy: Abscess surrounding thyroid cartilage and in right lung; suppuration bifurcation gland; empyema; collapse of lung; friable lungs; ulcerative endocarditis; myocarditis.

**Blood Changes.**—There is no definite inflammatory response as seen in lobar pneumonia. On the contrary in influenza alone a leucopenia is the rule. In some cases of pneumonia which exhibit a crisis a leucocytosis of 25,000 was frequently found, whilst in others, in which the patients died, a distinct leucopenia was noted (see Chart XV). In one case (Chart XVIII, p. 73), there was an initial leucocytosis of 60,000, which fell to 20,000, and for some time presented marked daily
Toxæmia, cyanosis; consolidation entire left lung, leucocytosis (38,000). Death from suppuration and breaking down of entire left lung.

variations. It is suggested that these changes are associated with secondary infections. During prolonged resolution, and more especially in abscess formation, there is some increase of the polymorphonuclear cells. In the sputum, also, the pus cell is not a marked feature in uncomplicated cases nor is there any degree of cell exudation comparable with that in grey hepatization.

The changes noted are: (1) A relative lymphocytosis; (2) activity of adenoid tissue; (3) intense engorgement. The significance of this lymphatic activity is not obvious at present. But as previously described there is often a polyadenitis and, in later phases of the epidemic, where cervical adenitis was marked, prognosis seemed better.

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**Chart XVI.**

Diffuse broncho-pneumonia, with delirium, cyanosis and respiratory distress. Death from edema of lungs and asphyxia. Marked leucocytosis preceding death (30,000). Pneumococci in sputum. At autopsy: Tracheitis; bronchitis; diffuse broncho-pneumonia; edema of lungs; hemorrhage into rectus muscle.

In the post-mortem room marked changes have been noted in the mediastinal glands but it has yet to be discovered whether Pfeiffer's bacilli are present in the glands. It is probable that this lymphatic activity plays a great part in the reaction to infection. The intense capillary engorgement and exudation of a highly albuminous serum must also be concerned in the reactionary process. And general observation suggests an elaboration of an antitoxin in the patient's blood. We have slight clinical evidence in one case in which treatment was based on this supposition.
Reparative Reactivity.

The questions of the damaged lung and the possibility of repair of the friable and emphysematous lung, &c., have been dealt with above. Our treatment therefore must be based on the hypothesis that the condition is one of toxæmia which leads to production of antitoxin and yet causes a certain degree of anatomical change.

Causes of Death.

These are (1) oedema, (2) asphyxia, (3) exhaustion, and (4) rarely, cardiac failure. For the most part death comes from failure of the respiratory system.

Treatment.

A survey of the track of the epidemic demonstrates that we are dealing with a mass infection which is spread along the lines of, and by, human traffic. The question whether this can be controlled is too big to touch upon in these notes and it is highly improbable that prophylaxis could ever be complete, especially so in war time. But much could be done to alleviate such conditions as overcrowding in huts, barracks and offices and, what is of great import in the light of demobilization, the condition of life in transports and troop trains. The movement of troops to localities where adverse climatic conditions prevail and to unsuitable depots and rest camps should also be avoided. Suitable ventilation, &c., at local centres will do much to diminish the incidence.

Individual Treatment.—At the moment this is purely symptomatic and though many specific remedies have been advocated none has proved satisfactory. The general management of the patient determines much and can be summed up as follows: fresh air, rest, sustenance and nursing. At the Sixty-third General Hospital our pneumonia cases were collected into five marquees each containing fourteen beds. The sides of the marquees were taken off, and only put up when required to afford protection from the occasional bleak winds; this was practically open-air treatment. The diet was suited to the patients' fancy and no restrictions in it were made. Brandy, 6 oz. or more during the day, according to previous habits, and champagne, were freely given, and port wine in convalescence. Medicinal treatment was of little avail. Salicylates certainly relieved the pains in the toxæmic stage. Expectorants were useless and later discarded. The inhalation of menthol and eucalyptus afforded much relief.
Morphia and, where obtainable, heroin were given at all times to produce sleep. Dover's powders were repeatedly given at night time, and judging by the rest the patients obtained there can be no doubt as to the benefit derived from the use of these drugs and in no case was there any evidence of their having done any harm. Sleep was an absolute necessity in the most serious cases, and this means of producing it was never refused. But as soon as the patients became convalescent morphia was stopped. This unconventional use of morphia, as was to be expected, aroused some anxiety on the part of my more timid colleagues but, notwithstanding the theoretical dangers, the repose that resulted from its use could only have been productive of good. So, after putting the matter before the consulting physician we no longer spared the drug.

Camphor was given in small and large doses with no appreciable result. Atropine was used freely in cases of marked œdema but was soon discarded. Digitalis and strychnine were used to a great extent at first, especially with a rapid pulse, and continued in smaller doses later, but no appreciable effect was noted.

Cyanosis was relieved by oxygen in many cases. At first venesection was tried but without success, except in those rare cases presenting signs of cardiac dilatation.

Quinine was given in large doses, often intravenously, in cases complicated by malaria and in delirious cases mistaken for cerebral malaria, but it appeared to have no effect, either good or bad.

After a time linseed poulticing was introduced and used extensively; most of the patients were considerably relieved by it and it seemed to do much good. After this the routine treatment followed was to give Dover's powders, 15 gr., on admission, and to apply linseed poultices as soon as signs of pulmonary involvement appeared.

Relation of Malaria to the Disease.

In Macedonia there was always the possibility of malaria to be considered both from the point of view of diagnosis and treatment. The presence of malaria was determined often by haematological examinations but more often at autopsy from the pigmentary and splenic changes. Some of these cases recovered, but for the most part the combination of malaria and pneumonia was a very fatal one.

At first the cerebral symptoms of influenza were mistaken for those of cerebral malaria and treated accordingly. But there was some
hesitation as to administration of quinine in view of its effect on the reactionary processes. But later all suspected cases were given quinine, as stated above (Charts VI, p. 59; IX, p. 61; XI, p. 63; XVII, p. 73).

Specific Treatment.

In less acute cases which lingered for days or weeks and presented signs of spreading involvement of the lung tissue it seemed that the primary infection was complicated by secondary infections presumably of pyogenic nature. Hence one naturally turned to vaccines for assistance. Two vaccines were employed. In the first I punctured a lung immediately after death and obtained a pure culture of a minute coccus of undetermined nature, and from this the vaccine was made. The other was a mixture of streptococci supplied from the Central Laboratory. Series of cases were taken and though it seemed at first that satisfactory results ensued, it was obvious that identical results obtained in the control series.

With the growing conviction that the essential process was one of toxæmia attention was directed to serum therapy and we had in mind the idea that in the blood of convalescent patients would be found the antitoxin itself, and we are still of this opinion. Unfortunately various difficulties presented themselves and it was not until the end of the epidemic on the Dardanelles that we found an opportunity to put these ideas into practice and then only in one case. The patient was gravely ill and indeed we had seen no other case of such gravity ever recover. But he was given serum from several picked convalescents and slowly improved. It is not presumed to offer this as proof of cure, though from the experience of this one case we have regretted not having attempted the treatment earlier. But it is put on record as suggestive and also as a proof that the use of the serum was at least harmless—an essential factor in initiating further investigations on these unconventional lines (Chart XVIII).

In working up these notes I have to acknowledge many kindnesses and much assistance at all times on the part of Colonel Phear, C.B., A.M.S., Consulting Physician to the Salonica Army, and to Major Elworthy, O.B.E., whilst O.C. of the Central Laboratory, and to my colleagues, Captains R. G. Willson, Carruthers, and Fowler, of the Sixty-third General Hospital and Twenty-eighth Casualty Clearing Station respectively.
Section of Medicine

**Chart XVII.**

Malaria (mixed benign tertian and malignant type). Cerebral symptoms; vomiting. Treated by intravenous quinine. Developed pleuro-pneumonia, and died with involvement of entire right lung; hiccough preceded death. Slow pulse throughout.

**Chart XVIII.**

Pneumonia, with severe toxemia and mental symptoms: marked cyanosis and gravely ill; leucocytosis (60,000). Serum taken from convalescent patients and inoculated subcutaneously, on several days. Marked improvement and recovery with resolution.

JU—7a
[The following specimens were exhibited: Two specimens of ulceration of vocal cords; specimens of marked enlargement and inflammation of bifurcation, bronchial and peritracheal glands; a case of infective endocarditis with aortic vegetations and abscess in myocardium; specimen of breaking-down lung; microphotographs illustrating alveolar exudations, bronchiolitis and surrounding changes, emphysema, and capillary engorgement of lymphatic glands.]
DISCUSSION ON EPIDEMIC ENCEPHALITIS.

Dr. F. W. Mott, F.R.S.

I will commence my remarks and demonstration with a résumé of the results of the examination of two brains made by M. Marinesco, Professor of Neurology at Bucharest, who has been working for some time past in the Pathological Laboratory of the Maudsley Neurological Clearing Hospital. Disseminated miliary or punctiform hæmorraghes, visible to the naked eye, existed in the grey matter in the neighbourhood of the floor of the fourth ventricle, the aqueduct of Sylvius and even the third ventricle, and were also found in the posterior part of the pons and of the peduncles. The cerebral cortex, except for congestion of some of the vessels of the leptomeninges, showed in these two cases neither macroscopic nor microscopic lesions. On the contrary, the first segment of the spinal cord, which was the portion of cord available, presented the same histological lesions as the pons, bulb and peduncles. Microscopical study of the above-mentioned regions demonstrated the existence of four kinds of lesions: (1) Infiltration of the walls of the small vessels and especially the veins, consisting of lymphocytes and plasma cells in the adventitia,
disposed in several layers. The endothelium and fibroblasts may also have taken part in the inflammatory process. (2) Foci of interstitial inflammation consisting of neuroglia cells of several kinds, including large cells with voluminous excentric nucleus and many fibrillar prolongations, lymphocytes and polynuclears. The foci of interstitial inflammation appear sometimes to be altogether independent of vascular infiltrations and may occur in the roots of the nerves—e.g., hypoglossal, pneumogastric. (3) Lesions of the nerve-cells which do not correspond with those usually seen in infantile paralysis. There is dissolution of the soi-disant Nissl bodies, relative achromatosis, reduction in volume of the cellular body and of the number of prolongations and multiplications of the satellite cells, but only exceptionally is there evidence of neuronophagia, as described by Economo. (4) The foci of hæmorrhage, the most obvious microscopic change, are seen to be much more numerous when microscopic examination is made. These hæmorrhagic foci remain circumscribed around the walls of the small vessels, and red corpuscles are mingled with the cells of inflammatory infiltration or they constitute a kind of covering and float about around the vessels. In spite of the very great number of hæmorrhages the vessel wall does not appear to be necrosed, but sometimes a solution of continuity of the vessel wall can be seen.

From microscopic investigation of these two cases M. Marinesco has drawn some conclusions as to the nature of the disease. He regards lethargic encephalitis as a disease entirely different from botulism, from the hæmorrhagic poliomyelitis of Wernicke, from the poliomyelitis of Heine-Medin, and from sleeping sickness. Like these two last diseases it is an infectious inflammatory disease, but the nature of the infectious germs has not yet been determined. It is distinguished from botulism by its symptomatology and by the four above-mentioned histological lesions. The vascular lesions, due to the presence of an infiltration of the walls of the vessels by migratory elements, are absent in botulism, and the readily recognizable Bacillus botulinus has not been found in these cases.

I will show photomicrographs to illustrate the chronic inflammatory conditions of the perivascular lymphatics in sleeping sickness and cerebro-spinal syphilis, in which there is definite infection by a specific organism and which may be accompanied by somnolence. In epidemic encephalitis and the bulbo-pontine encephalitis of Heine-Medin disease, which, as Wickman has shown, is a form of anterior poliomyelitis, somnolence is a frequent symptom, passing on to coma. Epidemic
encephalitis is an acute inflammation of the subadventitial perivascular lymphatics, and is characterized by naked-eye haemorrhages in the pons, the medulla, the peduncles, and around the third ventricle. Microscopic examination shows not only lymphocytes and plasma cells in the lymphatic sheaths of the arteries and veins, but large numbers of polynuclears, and the inflammation is so acute that there are haemorrhages, not only into the lymphatic sheath and periadventitial space, but into the tissues around. There is proliferation of neuroglia cells around the vessels and in the nervous tissue in the proximity of the inflamed vessel. I can find no essential difference between this epidemic encephalitis with its characteristic clinical symptoms of fever, headache, oculo-motor, facial and bulbar symptoms of difficulty in swallowing, paralysis of soft palate and respiratory symptoms, and the ponto-bulbar encephalitis of Oppenheim and Cassirer, observed frequently in the epidemic described by Wickman. In the Heine-Medin disease, as in cerebro-spinal syphilis, different forms arise according to the maximum seat of the lesion and this may be explained by supposing that the primary seat of invasion of the lymphatics of the nervous system varies in different cases and in different epidemics. Thus, the spinal cases may start from the intestines and find access to the spinal cord by the lymphatics surrounding the lumbo-sacral arteries; lesions in the bulbo-pontine and upper spinal region may start from infection of the lymphatics surrounding the vertebral artery when the throat and nose are the primary seat of invasion of the periarterial lymphatics, or by the infection of the internal carotid when in the encephalitic or meningeal forms. It is said that this epidemic of ponto-bulbar encephalitis cannot be the same as Heine-Medin disease, for the cerebro-spinal fluid obtained by lumbar puncture contains no lymphocytes. Dr. C. R. Box has had cases in which lymphocytes have been found. It appears that when the meninges are affected lymphocytes and albumin are certain to appear in the cerebro-spinal fluid.

Dr. P. N. PANTON.

At the London Hospital, the blood and the cerebro-spinal fluid were examined as to the protein content and the cellular constituents in about twenty typical cases of this condition, and, apparently the diagnosis was correct in eighteen of them. The blood showed an average leucocyte count of 7,600. The cerebro-spinal fluid was in all
cases perfectly clear. The protein was present in very slight excess. With regard to cellular content, and the existence of a lymphocytosis, very few such cells were found. In a few cases the cellular content was normal; in some others there was a trifling excess of leucocytes. The finding of other lesions appeared to be the best aid to diagnosis. We had a good many cases of meningococcal inflammation, but we could scarcely deny that a case was one of epidemic encephalitis because we found no changes, as the changes were so small and it was hardly possible to distinguish between this condition and poliomyelitis. In the American cases there was a high degree of leucocytosis and an increase of lymphocytes in the spinal fluid.

Dr. F. G. Crookshank.

My view of the late epidemic is that the cases we then saw represent what I may call only part of a "spectrum," of which one end is constituted by "infantile paralysis" and the other by "acute delirious mania" and various forms of "encephalitis." Fifteen years ago, Wickman pointed out that, in Heine-Medin disease, some epidemics differ in type from others, just as do cases in a focus and foci in an epidemic. This is true not only clinically but also pathologically, as is shown by the American records—a point not without bearing on Dr. Mott's remarks. So, too, with reference to blood counts. In one epidemic (in Germany) Müller found leucopenia almost constantly, whereas in New York, in 1916, leucopenia was very rare.

Again, in some epidemics, examination of the cerebro-spinal fluid has shown a high cell count. But in England, this year, the reverse was the case, though some lymphocytosis was the rule.

With reference to Dr. Panton's reports it is important to indicate, as he has not done, the day of the disease on which the cerebro-spinal fluid is examined. The American observers have shown that, after the initial rise, the cell count rapidly falls, and many of Dr. Panton's examinations were made late in the first week, or even later.

Another point—that of the causal organism. Sufficient attention has not been paid in this country to the work of Rosenow and his associates who, in 1916, found a certain pleomorphic coccus (which apparently, under certain conditions, splits up into the "globoid bodies") frequently associated with poliomyelitis. On drawing the attention of bacteriological friends to Rosenow's papers, it appeared clear to them that the organism described by von Wiesner, in 1917, as the cause of
“encephalitis lethargica” in Vienna was, in fact, Rosenow’s coccus. The same organism was then found in London in several cases of “encephalitis,” and in associated abortive “influenzal” cases. Moreover, the “globoid bodies” were produced in accordance with Rosenow’s accounts and directions. It appears also that a peculiar coccus, very closely resembling this most remarkable organism, has been lately identified by various observers in many parts of Europe (in association with “influenzas”) and given various names.

Dr. J. A. Murray.

Dr. McIntosh, of the London Hospital, in the course of his bacteriological investigations, was unable to find the organism of botulism, and a number of monkeys were inoculated. In no case was a true paralysis of the poliomyelitis type produced. In the only monkey in which paralysis occurred it was due to a pressure lesion of the cord. From the experimental side the work has, so far, resulted in no real evidence as to the nature of the disease, but further investigations with the serum are to be carried out.

Dr. C. Da Fano.

I have had the opportunity of seeing some preparations from Dr. Ingleby’s case which we are now studying. So far as I have been able to see, the lesions are, up to a point at least, different from those of polio-encephalomyelitis. It is, however, better to wait until our investigations are completed, and we are able to examine the nervous system carefully by modern and exact methods before pronouncing on the subject.

Dr. Helen Ingleby.

I had the opportunity of observing a well-marked case of the meningeal form of the disease, and the cerebro-spinal fluid showed no abnormality.

Dr. W. H. Hamer.

Early in 1915 a meeting was held here to discuss the outbreak of cerebro-spinal fever then prevailing in London, and certain epidemiological points were brought under notice. Thus it was contended that
the cases of cerebro-spinal fever constituted only a part, indeed, relatively quite a small part of the epidemic then prevailing; in illustration reference was made to Sydenham's "new fever" of 1685; it was shown that between this and the outbreak of 1915, considered as a whole, a strikingly close resemblance existed, and it was urged that the demonstration of "species," "varieties," and "strains" of organisms associated with particular symptom-complexes should not be allowed to blind us to the fact that we were face to face with an epidemic. We are now, once more engaged in considering a particular group of symptoms, and there is again a risk that the wood may be obscured by the trees.

Since then, however, two new developments have occurred. In the first place the feeling that epidemics may advantageously be studied from an epidemiological or macroscopical, as well as from a bacteriological or microscopical point of view, has gained ground; secondly, there has been some searching of heart with regard to debatable questions arising out of recently advocated segregation methods.

Medical officers of health have been much interested in these two developments. They have seen how, in recent experience, the epidemiological teaching that influenza is a protean disease has been once again verified, and they have had borne in upon their minds the impracticability of applying in civilian life, on a large scale, methods which have been prescribed for use in some instances in the Army and Navy. It should be specially noted, moreover, that during the last ten years evidence has been collected clearly showing a close association between outbreaks of poliomyelitis, polio-encephalitis, cerebro-spinal meningitis and prevalences of influenza.

In November, 1916, I read a paper before the Section of Epidemiology, in which Brorström's demonstration of the influenzal nature of poliomyelitis, and the London evidence as to the identity of origin of influenza and cerebro-spinal meningitis were referred to. Following upon this my colleagues and I had an opportunity of discussing the whole question with an Army epidemiologist, and we found that he had already worked out in Army camps the problem with which we had been confronted in a civilian population. As his report has not yet been published, and our own conclusions have encountered criticism, there is some risk at the moment of the work being lost.

The early epidemiologists advocated the extensive view of their subject, but the latest laboratory methods, described in a recent number
of the Journal of Hygiene, are based upon "a process of balancing, dependent upon a series of very accurate measurements and quantitative adjustments." Six weeks' hard work are necessary "before even a trained bacteriologist with considerable serological experience can sufficiently master the technique to obtain consistently satisfactory results," and, if the expert goes for a holiday, "even for a week, it required at least another week's work before the necessary unconscious manipulative dexterity returns."

Under such a method of study how are we to know an epidemic when we see it? In place of broad epidemiological inquiry there are substituted laboratory methods, concerning which, however, there is no agreement, for some workers advocate serological and others fermentation tests.

Epidemic encephalitis is not a new disease. Certain extracts from Hecker's account of the sweating sickness prove this, inasmuch as they show that stupor or lethargy is writ large over the earliest "posting" influenzas of the historical records—those of the fifteenth and sixteenth centuries. It was associated with the "sweating sickness," which prevailed in England contemporaneously with the widespread continental outbreaks of cerebritis, cephalitis, "hauptwehe" "trousse-galant" and influenza.

And this brings us to the great epidemiological event of the year 1918. Hard on the heels of the "epidemic lethargy" came the "Spanish influenza." Is it not permissible, therefore, to plead once again for the epidemiological point of view? The fact of the matter is that these "new diseases" cannot possibly be fully comprehended by cell counts in cerebro-spinal fluid, or by inquiring as to the absence or presence of strains of organisms resembling one another as closely as Francis Galton's twins, who, you will remember, "exhibited a quaint interchangeableness of expression that often gave to each the effect of being more like his brother than himself." Again, these new diseases cannot be completely understood by insisting upon the fact that particular structures have been picked out in the cerebro-spinal nervous system, and that the lesions are here at one level and there at another—nor can they be satisfactorily explained by concentrating attention upon some particular sign or symptom, whether it be sweat, lethargy, paralysis, an exanthem, or some evidence of involvement of the pulmonary, nervous or gastro-intestinal systems, and by considering each phase of the epidemic prevalence in relation thereto, and studying it apart from all the other phases. There is a time-honoured method
which cannot be ignored—viz., the epidemiological method, and recent experience has shown that a clue to the enigma is to be found in a return to that method and to the doctrine of "epidemic constitutions." The teaching of Sydenham should be once again studied in the light of the subsequent epidemiological history. Creighton has told us that there is "something more than accident in the association between epidemics of influenza and epidemics of ague." Examination of his "History of Epidemics" shows, I submit, quite clearly that the remarkable related "agues," which occur in the years round about all the great "posting" epidemics of influenza, throughout the whole of the recorded history, are nothing more than those very gastro-intestinal, pulmonary and nervous manifestations which actually constitute, as every epidemiologist realizes, part and parcel of the influenza prevalences themselves.

Lieutenant-Colonel S. P. James, I.M.S.

Sir Arthur Newsholme has authorized me to give a brief summary of the results of an inquiry into encephalitis lethargica recently carried out by the medical staff of the Local Government Board in collaboration with the Medical Research Committee. Printing difficulties have greatly delayed the issue of the report of this inquiry, but it is hoped that it will be published during the coming week, when we shall be glad to send a copy to anyone interested. The following summary is abbreviated from Sir Arthur Newsholme's general review which prefaces the report.

In its initial stages last April the inquiry was concerned chiefly with an investigation of the hypothesis that the illness was a manifestation of botulism. In connexion with that view Dr. McIntosh, on behalf of the Medical Research Committee, carried out a complete bacteriological investigation, and Dr. Hancock and Dr. Pearse, of the Food Branch of the Board's Medical Department, personally investigated, from the point of view of a possible food origin, fifty-eight cases reported up to May 7. The result of these preliminary inquiries was that neither on the bacteriological nor on the epidemiological side could any direct or indirect evidence be obtained of an association of the illness with the Bacillus botulinus or with infection from food. In addition to proving that the illness is not due to food, the results of the preliminary inquiry pointed to a possibility that it might be one of the many forms of the
disease—or group of diseases—to which nosologists at present attach the indiscriminative label “Heine-Medin” disease, of which infantile paralysis—officially termed acute poliomyelitis—is the commonest type of illness.

It was found, however, that the present illness differs very strikingly from infantile paralysis, not only in the localization of the paralysis and some other equally obvious signs, but in its age-incidence, seasonal prevalence, course, duration and fatality. In these circumstances it was decided to carry out a more complete clinical and epidemiological study of the illness than had been attempted in regard to the first fifty-eight cases. To this end the Public Health Department of the London County Council, which had collaborated from the beginning of the inquiry, agreed to investigate all cases in the London area, thus enabling the Board's medical inspectors to be deputed, as opportunity offered, to investigate the provincial cases. Up to the end of June the number of cases in the London area inquired into was 107, and the number in the provinces was 121. Apart from these special inquiries Dr. MacNalty was deputed to undertake a detailed personal study of the clinical symptoms of the illness. In this task he has had the advantage of conferring with Sir William Osler, and with Major George Draper of the United States Army Medical Corps. Major Draper's services were lent to the Board through the courtesy of the Surgeon-General and Chief Surgeon, American Expeditionary Force, and he examined and reported on cases in Birmingham, Leicester, Oxford and other localities. His report is printed in the publication from which my present remarks are taken.

On the bacteriological side, in addition to the research in connexion with a possible food origin, the problem of the nature of the illness was attacked from the point of view that the virus might be closely allied to or perhaps identical with that of acute poliomyelitis, and in this line of research animal experiments were begun as soon as the necessary monkeys could be obtained. In addition Sir Walter Fletcher, F.R.S., Secretary to the Medical Research Committee, was enabled to secure the services of Professor G. Marinesco, of Bucharest, for the examination of specimens from fatal cases of the disease.

The accounts given by all these observers serve both to describe the illness and so far as present knowledge permits, to elucidate its nature: and it is satisfactory that as regards the solution of the vexed problem whether the illness is or is not a form of acute poliomyelitis the conclusions which emerge from several methods of study carried out
independently are in effect the same. I may summarize them very briefly as follows:—

(1) For identification and description it was decided to follow Economo in terming the illness encephalitis lethargica, a name which has the right of priority and indicates a characteristic clinical feature.

(2) Dr. MacNalty, from his clinical study, concludes first that in its essential primary features the illness has a characteristic and constant symptom series of its own: second, that between this symptom series and that of the rare forms of poliomyelitis with which alone it could be confused the clinical differences are more prominent than the resemblances.

(3) The results of the epidemiological inquiries were striking and permit no ambiguity of inference. They are to the effect that encephalitis lethargica is not a form of acute poliomyelitis and that its presence and epidemic prevalence depend on conditions other than those necessary for the presence and epidemic prevalence of that disease.

(4) Both Professor Marinesco and Dr. McIntosh, as a result of their separate researches, arrive independently at the conclusion that encephalitis lethargica as it appeared in the present outbreak is identical with the illness described by Economo in Austria and Professor Netter in France, and that it is a disease sui generis anatomically, and is clinically distinct from analogous affections.

By all these findings an important part of the problem was cleared up; and it now remains to ascertain what the nature of the disease is, rather than what it is not. On account of the speedy cessation of the outbreak this has not yet been possible, and it must now wait until a larger incidence of the disease supplies material for further research.

In the meantime various hypotheses have been suggested by different workers engaged in the inquiry, and these will come up for consideration if another outbreak is experienced. They are enumerated in the report but time does not permit me to state them here. I will therefore conclude by stating the view which appears best to agree with present knowledge. It is that encephalitis lethargica is one of a group of diseases in which, as in cerebro-spinal fever and acute poliomyelitis, the pathogenic agent is much more generally present in the human organism than the clinical evidence implies. As regards cerebro-spinal fever this is no longer an hypothesis but a well-established observation. In that condition the specific reaction named cerebro-spinal fever arises
in one of two ways: first, as the result of a breakdown in the immunity to the effects of the virus which the individual who harboured it had up to that time enjoyed: second, as the result of a non-immune person becoming infected with a strain of the virus which has attained the degree of pathogenic action described as specific. During severe epidemics evidence can sometimes be obtained that cases of cerebrospinal fever are arising in both these ways, but during inter-epidemic and mild epidemic periods it is seldom or never possible to obtain evidence that the illness is infectious; at such times cases of the disease are always scattered sporadically, and they cannot be traced to any known source of infection except the patient himself. This is the view that best explains the irregular widespread, sporadic distribution of encephalitis lethargica. And until further research yields precise information we may assume that many people harbour the organism of the illness, and that in certain of these persons there occurs for some unknown reason either an enhanced virulence of the parasite or a lowered resistance of the tissue cells, or both, the result being that the stimulus of the parasite overcomes the resistance of the tissue cells and the host suffers from the effects of the virus which previously he had harboured with impunity. This explanation implies that the key to the problem rests not in the purely bacteriological view of the causation of disease, but in the wider view that disease results from the interaction of several factors, of which changes in the properties of the tissue cells on the one hand and in the provoking stimulus or pathogenic agent on the other are the chief.

This view emphasizes the rôle of the individual in the origin and progress of the disease; and the practical indication would seem to be to enlist all the resources of personal and public hygiene in an endeavour to influence favourably the potential energies of body and tissue-cell resistance, especially in individuals who may seem to be predisposed to a disease of this nature if they happen to become a host of its parasite. At the same time it is clearly of importance to pursue research both to ascertain what pathogenic agent is concerned, and what are the factors of individual predisposition and correlated resistance, the variations of which are subject to so many influences.

The first necessity for these studies is early and complete information of all cases of the disease in different parts of the country, and for this reason the Local Government Board has decided, as a temporary measure, to make encephalitis lethargica compulsorily notifiable for a period of one year.
Dr. F. G. Crookshank.

Careful inquiry has satisfied me that Dr. Hamer is right in stating that the association of diverse epidemics of myelitis, poliomyelitis, mesencephalitis and encephalitis, with epidemics of "influenzas," has obtained not merely for years, but for centuries. Change of type there has been, no doubt, but changed conceptions of disease have also arisen, and we have got into the habit of speaking as if a new conception of disease corresponded with a new appearance of disease, or the appearance of a new disease. Particular diseases are conceptual only, and not natural objects. Certainly no one would claim that the late epidemic—chiefly one of polio-encephalitis—was one of "poliomyelitis." But I do claim that epidemic encephalitis and epidemic poliomyelitis are made of the same material, and form part of the same suit. The observed difference (entirely quantitative and not specific) does not warrant the belief in two different "diseases," and is perfectly compatible with what Wickman has said concerning the protean nature of Heine-Medin disease. It is not a case of discovering or observing a new disease, but of re-arranging or extending our concepts of an older one—namely, the Heine-Medin disease.

Colonel James has spoken of the types of Heine-Medin disease other than "infantile paralysis" as being very rare. But is this really the case? Is it not rather true that observation had been hampered by too narrow a conception? It is barely thirty years since Medin first showed that we have to deal with something more than "infantile paralysis"—an acute and specific disease affecting only the anterior horns of the grey matter of the cord. In 1892, Sir Thomas Barlow pointed out how like the early stage of infantile paralysis often was to influenza; and this at a time when poliomyelitis was common, and Sir John Rose Bradford was urging the view (advocated eight years earlier by Strümpell) that polio-encephalitis was a part of the "same disease" as poliomyelitis. In those days of influenza there was plenty of polio-encephalitis and poliomyelitis, and in 1892, the late Dr. Vivian Poore had, in his wards, more than one case of stupor that would now be called "encephalitis lethargica." Some diagnosed these cases as typhoid, others as this and as that; but, looking back, the diagnosis becomes clear. Indeed, reference to medical journals shows clearly how, in those years of influenza prevalences, encephalitis and poliomyelitis were, as now, constantly associated. This coincidence was
very notable in 1910-11-12. A remarkable case was that of a lady who died of an "obscure cerebral condition," in stupor, with anomalous paralyses. Post-mortem, a mesencephalitis was demonstrated by the late Dr. R. G. Hebb, and we afterwards traced the infection as having been almost certainly contracted, three weeks earlier, at Hitchin where poliomyelitis was then prevalent.

The necessity is, not for further analysis and discrimination, but for a synthetical approach to a solution of the various questions raised.

Sir Arthur Newsholme. K.C.B.

I agree entirely with what Colonel James has said. There appears to be a misunderstanding or a fallacy underlying the remarks of Dr. Crookshank. These seem to claim identity when mere grouping together is all that is indicated. He wishes to throw us back to a single disease with very variable symptoms, when the more probable scientific explanation is that we are dealing with a group of diseases occurring under similar conditions and possibly excited by similar external circumstances. We know that in a succession of dry years the prevalence of scarlet fever and diphtheria, and of puerperal fever even, is much greater than in other years characterized by excessive rainfall. These facts were shown, as to two or three of the conditions, by Dr. Longstaff, and in my Milroy Lectures I showed that this applied on a wide scale to rheumatic fever. But although these diseases have occurred in conjunction with each other and under similar conditions, it does not follow that they are identical diseases. It is true that the diseases we are now discussing are not differentiated from one another clearly at the present time, but the differentiation appears to be in process of being made. And the reports by Colonel James and Dr. MacNalty show that the clinical as well as the pathological effects, both being consistent, conform much more nearly to the idea that we have to deal, in lethargic encephalitis, with a disease which, although it occurs under similar conditions with poliomyelitis and others of the group, is not pathologically the same.

Dr. P. N. Panton.

It seems to be taken for granted in some quarters that in these epidemics spread over centuries we are dealing always with the same disease. There is not the smallest scientific evidence in favour of that
theory. In the various influenza epidemics, how can anybody say that the disease is invariably the same? Even in this epidemic one must have seen cases which have seemed to be encephalitic, and were afterwards proved to be nothing of the kind. The most competent amongst us sometimes cannot distinguish between one and other of these conditions. It will not do on such evidence to take it for granted in these epidemics that we are dealing with the same disease.

Dr. John Robertson.¹

In the cases which occurred in the Birmingham area this spring, the feature which was most prominent was that they occurred suddenly and without any previous occurrence of anterior poliomyelitis or influenza. Quite a large number of reports came to hand within a few days, and within a period of three weeks nearly all the cases that were reported occurred. These reported cases ceased, almost as suddenly as they appeared. The explanation that Dr. Hamer gives does not prove to be an explanation of the sudden occurrence in a large city population of cases having a fairly definite train of symptoms.

Dr. F. G. Crookshank.

Colonel James has insisted that, whereas encephalitis attacks both old and young, poliomyelitis—by which he means, I take it, acute anterior poliomyelitis with resultant flaccid atrophy—is pre-eminently met with in young children. But surely this observation does not warrant his conclusion, that we are dealing with different "diseases." It is only as it should be, for, in children the anterior horns are far from being organized, and are obviously more liable to attack than in older people—at any rate so far as the production of resultant flaccid atrophy is concerned. The question is a biological one. Moreover, it is not always so much that actual atrophy is produced as that the child "grows out" of the stunted limb, which ceases to develop after the injury done to the anterior horns. That cannot well take place in adults.

The facts are obscured by our habitual terminology: for indeed we speak sometimes much as do parents of a growing lad who say "his

¹ Medical Officer of Health, Birmingham.
clothes are getting too small for him." The point was noticed by Laborde so far back as 1856. We cannot describe as "different diseases" symptom groups differing from each other merely because of the structural peculiarities of the persons concerned at different age groups.

The fallacy in Colonel James's argument appears at bottom the same as that involved in the pretended distinction between scurvy in adults and scurvy in children who have not cut teeth—namely, that in the latter the gums are not affected.

Dr. Farquhar Buzzard.

Colonel James is right in stating that everything points to the fact that encephalitis lethargica is an entirely different condition from that form of encephalitis due to the virus of poliomyelitis. I do not regard cranial nerve palsies as one of the characteristic features of the disease. Certain cases at post-mortem examinations have shown that the site of encephalitis may be either in the cerebral hemispheres, producing hemiplegia, hemianesthesia, hemianopia, &c., in the mid-brain producing a picture like that of paralysis agitans, or in the brain-stem with ocular palsies, &c., forming the most marked features of the clinical picture. Probably the encephalitis occasionally gives rise to severe haemorrhages and causes symptoms and signs of increased intracranial pressure which frequently prove fatal.

Dr. A. J. Hall (Sheffield).

Sixteen undoubted cases have been under my immediate observation. The epidemic in Sheffield differed from the whole, in that there has not been one fatal case throughout. My cases have included persons of each decade up to 70 years. Only two have been under ten years. The sexes are about equally represented. The epidemic began early in March, and was practically over by the end of April.

Symptoms and Signs.—The three cardinal signs in a typical case are lethargy, general asthenia, and cranial nerve palsies. One or even two of the cardinal signs may be slight or absent, but as a rule the clinical picture is chiefly due to the asthenia and lethargy. In one of the cases the lethargy was slight and the cranial palsies limited to a slight ophthalmoplegia, but the general asthenia was so extreme that the
patient gave a typical picture of immobility, being unable to turn over in bed and hardly able to move arms or legs. Another case within twenty-four hours developed almost complete facial diplegia, whereas there was no lethargy or asthenia throughout. I emphasize the importance of considering the three cardinal signs as a whole; any one of them may predominate or may be slight or absent. In most cases the cranial nerve palsies formed but a small part of the whole symptom-complex.

Prodromal Period.—In most cases there is a distinct interval between the onset of illness and appearance of characteristic symptoms, and this varies widely. It is usually a few days—about one to four or five—but in some of the cases it has been not less that ten days. The onset of the cardinal signs may be sudden, but is usually ingravescent, and this is particularly true of the cranial palsies. They take some time to develop fully, and may vary in extent from day to day. The general asthenia tends to make the facial palsies less obvious than usual. Ophthalmoplegias: Strabismus of variable extent, inequality of pupils, and nystagmus are commonly present. The last named is sometimes of an irregular and incoördinate kind. Fever is noted in many cases, sometimes severe and prolonged, but may be slight, even in a severe attack. Lethargy is perhaps the most striking feature of most of the cases. It is not true sleep. Often the patient is surprisingly awake to what is going on. Many who are stuporose all day become delirious at night, returning to stupor next day. Sometimes the delirium is of a foolish kind and suggests hysteria. Tremors: In one case tremors with asthenia were the chief symptoms. Speech: During the lethargic period this is often affected. In one case there was a distinct hurried or festinating speech, such as is sometimes observed in paralysis agitans.

Results.—All the sixteen patients are still alive, most were seen within the past few days, and reports from their medical attendants have been received of others. Group A consists of seven cases in which recovery was complete and absolute. Group B contains six cases in which recovery was practically complete, some slight trace of illness being left behind. In Group C there were three cases, and these after six months are still far from recovery. In each case the present incapacity is due to involvement of the trunk and limb muscles generally, the cranial palsies having disappeared completely or almost so.

The points for discussion may be summed up as follows: Was this or was it not an epidemic of poliomyelitis? If it was not, then it may
have been either an entirely new disease or one that until recent times has not been observed in epidemic form. Lethargy and asthenia, so severe and prolonged in most of the cases of encephalitis, has not been recorded as occurring in typical cases of poliomyelitis, sporadic or epidemic. I cannot think that anyone would consider the term "sleeping sickness" descriptive of poliomyelitis in general, yet it would not be an inappropriate name for this epidemic. Even the palsies present striking differences in the two diseases. In poliomyelitis the onset tends to be rapid and maximal; the regions affected are usually those innervated from the spinal cord; the distribution is often unilateral, and they frequently leave permanent residual effects of greater or less extent in the areas first attacked. In the recent epidemic the paralyses were typically of gradual ingravescent onset; they almost always affected regions innervated by cranial nerves and were often bilateral. So far as present evidence is available they usually left no permanent residual effects in the area first affected. We know that many nerve poisons, e.g., diphtheria, Bacillus botulinus, lead, alcohol—show a peculiar predilection for certain particular parts of the nervous system; in these cases of epidemic encephalitis there seems to have been such a definite selective action at work. It has been pointed out that poliomyelitis in its epidemic form is characterized by the multiplicity of the sites affected. The cases of the recent epidemic were notable for the uniformity of the sites affected. With regard to the possibility of this being an old disease which has only recently been recorded in epidemic form, I have no proof to bring forward. A similar epidemic appeared about the same time in 1917. In course of time records of sporadic cases of a similar type may be found as abnormal or exceptional varieties of influenza or poliomyelitis, &c. Such a line of inquiry seems worth consideration, but in any case the final decision rests with the pathologist. The photographs displayed by the epidiascope showed the mask-like countenance, the lethargy, and the posture of patients in bed.

Dr. A. Salusbury MacNalty.

From anatomical considerations and a survey of the clinical evidence, it has been found possible to classify the cases of encephalitis lethargica, occurring in the recent outbreak, as follows:—

An acute illness in which nervous localizing signs may or may not be present.

N—5
This illness is comparable with acute poliomyelitis (Heine-Medin disease), syphilis, enteric fever, &c. Of this malady there exist the following types:—

(1) A type displaying general disturbances of the functions of the central nervous system, but without localizing signs.

(2) Types with nervous localizing signs.
   (a) Clinical affection of the third pair of cranial nerves.
   (b) Affections of the brain-stem and bulb. Local lesions of other cranial nerves.
   (c) Affections of the long tracts, e.g., pyramidal (as shown by the presence of Babinski's sign), pre-pyramidal (von Monakow), as evidenced by tremors, &c., and up-coming afferent tracts.
   (d) Ataxic types (involvement of the cerebellar mechanism).
   (e) Affections of the cerebral cortex.
   (f) Types indicating some evidence of spinal cord involvement.
   (g) Types indicating possibly an affection of peripheral nerves (polyneuritic cases).

(3) Mild or abortive types of the disease, with or without localizing signs in the central nervous system.

The various types show signs of common identity. This resemblance is not only evinced by the general symptomatology, but is shown by the gradual merging of cases of one type into cases of another type.

The disease may be defined as a general infectious disease, characterized by manifestations originating in the central nervous system, of which the most frequent and characteristic are progressive lethargy or stupor and a lesion in or about the nuclei of the third pair of cranial nerves.

In the first place brief reference may be made to the description of the general disease, which is recognizable in all types of encephalitis. Like other infectious diseases, it may be assumed that encephalitis lethargica has a period of incubation, followed by a period of prodromal symptoms, then by acute manifestations, and finally by a stage of regression. The duration of the incubation period, like that of acute poliomyelitis, is probably variable. The prodromal period commonly ranges from the first to the seventh day, but may be protracted as long as three weeks. In a few instances a distinct interval of remission occurs, often of some duration. The severity of the prodromes bears no relation to the clinical type subsequently declared, or to the subsequent course of the disease. To a certain extent the prodromes are specific and indicate an affection of the central nervous system.
Among such symptoms, lethargy, headache, giddiness and diplopia rank chief. There are also general symptoms such as lassitude, fatigue, vomiting and diarrhoea; in a number of cases the first general symptom is a simple catarrhal conjunctivitis. After a prodromal period of variable duration the declared symptoms of a general infectious disease become manifest. There is an early febrile period, usually lasting for two to three days. A common variation of temperature is between 101° and 102° F. Marked asthenia is present. The patient lies in bed, often immobile, with an expressionless, mask-like face. Catalepsy, including flexibilitas cerea is not uncommon and may last for several weeks. At this stage the lethargy of the prodromal period has passed into the characteristic stupor. This stupor may be of all grades of severity. Early in the illness it often alternates with periods of nocturnal delirium ranging from slight rambling to maniacal seizures. Rigidity is present, sometimes restricted to the muscles at the back of the neck, frequently having the character of "plastic tone," described by Sherrington, sometimes approximating to the so-called "late rigidity," the extremities being rigidly held and hypertonia being present in all the muscles. Emotional changes are present and these come out in cases with slight stupor or when the patient is aroused from stupor. There are highly characteristic speech changes, the voice being nasal and monotonous, sentences being uttered very slowly and words slurred into one another like the voice of a general paralytic, and uttered hesitatingly. In some patients, the speech for a few minutes becomes chattering, rapid and unintelligible. Fibrillary movements, twitchings and tremors are common. Not infrequently irregular, non-rhythmic spontaneous movements of the face, trunk and limbs, resembling those seen in chorea and thalamic affections, are present late in the disease. Muscular pains, hyperæsthesia, retention or incontinence of urine, incontinence of faeces, sweating, skin eruptions and dysphagia are other symptoms that may occur. Marked constipation is present in practically every case. Such are the chief features of the general symptomatology.

In the type without localizing signs in the central nervous system, the general symptoms such as pyrexia, lethargy, stupor and asthenia are prominent. In one case in my series, that of an adult, which proved fatal, pathological proof was given of the accuracy of the clinical diagnosis. From the clinical standpoint, the inclusion of a case in this group can be justified only by pointing out its close approximation in symptomatology to proven cases of encephalitis without localizing signs.
In this type, the manifestations of the disease are severe and the proportion of deaths high.

In clinical types of encephalitis with nervous localizing signs, paralysis of various cranial nerves, notably the third and the seventh are most common. The clinical character of the lesion may be supranuclear, nuclear or infranuclear. The onset of cranial nerve paralysis is variable; usually, however, it begins in the first week of declared illness. It has the following characteristics: (1) It may be complete or incomplete and unilateral or bilateral; (2) when bilateral, it is not strictly symmetrical, the extent to which paralysis has occurred being always greater on one side than another; (3) the paralysis is progressive up to a certain point and on its first appearance is never at its maximum. This progressive character does not only appear in isolated and bilateral lesions. When more than one cranial nerve is involved the resultant palsies hardly ever appear simultaneously. Finally, the rapid complete or partial clearing of the paralysis is perhaps the most remarkable and distinctive feature of the types with local nervous manifestations.

The prominent features of the remaining clinical types are revealed in their nomenclature. Mild or abortive cases are distinctly rare.

Recovery from the disease is gradual and tedious, as a rule, chiefly on account of the great prostration and muscular weakness.

In fatal cases untoward signs are an intensification and frequency in delirious attacks and deepening of the stupor. The patient then passes into deep coma and death ensues, apparently from paralysis of the respiratory centre in the medulla. In some instances death is due to a respiratory complication, usually broncho-pneumonia. Death most often occurs within the first three weeks and commonly during the third week of the illness.

As regards after-effects, those that have come under notice in a limited number of cases are: (1) An alteration in the mental condition; (2) the persistence of cranial nerve palsies; (3) the subsequent appearance of paralysis, apparently of spinal cord origin; (4) athetosis.

I venture to draw the following conclusions in regard to the clinical nature of encephalitis lethargica and its relation to Heine-Medin disease: (1) The forms of disease seen in the outbreak, although resembling poliomyelitis in certain features, differed from that disease in more important respects and displayed a characteristic clinical syndrome. (2) It is possible that the accepted classification of poliomyelitis has been made on too wide a basis and that certain cerebral,
ponto-bulbar and ataxic types described as occurring in rare instances in epidemics of poliomyelitis may in reality have been examples of the clinical forms observed in this outbreak. (3) In our study of cases in the outbreak there are clinical indications that poliomyelitis and encephalitis have occurred side by side. (4) Assuming the independence between encephalitis and poliomyelitis the relationship between them would be comparable to the relationship that is known to exist between typhoid and paratyphoid fever.

The identity or non-identity of the two diseases must be finally established by pathological research. Nevertheless, even if the virus of poliomyelitis is found in the future to be the aetiological factor in encephalitis, the evidence is sufficient to establish the existence of a clinical syndrome, hitherto undescribed in epidemics of Heine-Medin disease.

**Dr. F. G. Crookshank.**

With regard to treatment, in a certain number of cases I was able to secure the trial of a mixture of urotropin, benzoate of ammonium, and salicylate of sodium, and the combination of these drugs appears to be useful.

I have notes of 127 cases, 120 of which were seen at the London Hospital, four at the North-West London Hospital, and three in private. Some cases occurred early in January, others in February, but, by March, an outbreak of polio-encephalitis of Heine-Medin type had been confidently predicted. Deaths were twenty-six, or 20.39 per cent.; post-mortems were made in eleven cases; seventy-seven patients were male, fifty female; twenty-eight were under 5 years of age, twenty-nine between 6 and 10, and seventy over 10. These figures, when compared with Ruhräh's table of age-distribution in the New York City, up-river States, and New York Rural outbreaks of 1916, show that the differences between London (1918) and New York Rural (1916) are less than those between New York Rural (1916) and New York City (1916)—a fact which I presume Colonel James would not deem to warrant the notion that different diseases were occurring side by side in New York City and New York Rural in 1916.

Lumbar puncture was done in fifty-one cases; seven times without result. Protein was found in excess in twenty-six out of twenty-seven cases; lymphocytes were present in excess in twenty-five, and in marked excess in six cases. Leucocytes were present in excess in four cases,
and in marked excess in two. Gram-negative organisms were found twice; Gram-positive organisms twice.

Concerning the mode of onset: There was a history of injury to the head in nine cases; tooth extraction was mentioned twice. There was a two-stage history in forty-six, a history of sudden onset of nervous symptoms in thirty-seven, and a gradual onset in seventeen. The general symptoms included profuse sweating in thirteen cases, marked diarrhoea in seven, constipation in twenty-nine, glycosuria (without any previous history and with complete recovery) in one case, acetonuria (without glycosuria) in four cases. There was marked vomiting at the onset in thirty-eight cases, and pain in the hypochondrium in thirteen cases. (The eighteenth century physicians used to speak of pain in the hypochondrium, in the "nervous fevers," marked before the nervous system was affected.) The rashes observed were erythematous in twelve cases, miliary in two cases, and petechial in five, while there was herpes in eight cases, desquamation in two, joint swelling in two, oedema of the legs in one, lymphadenitis (marked) in three cases, and bed-sores in one; Draper's sign—the separation of the central incisors—was noted in many cases, but recorded only in ten. Emotionalism was marked in five, mutism was recorded in seven, delirium in nineteen, acute delirious mania in one, hallucinations in three, and nocturnal sleeplessness (marked) in three. Drowsiness was prominent in thirty cases, stupor in twenty-six, coma in twenty-five, and epileptiform "fits" in fifteen cases. Head retraction was marked in twenty-three cases, and limb spasticity in twenty-nine. Tremors, twitching, or chorea were marked in twenty-two, trismus was very marked in two, flaccid paralysis (apart from asthenia) was noted in forty-seven cases, atrophy of muscle groups (apart from generalized atrophy) in ten. Facial paralysis (other than transitory) in eight cases, amaurosis with slight disk changes in five, ptosis in twenty-three, strabismus in nineteen, nystagmus in fourteen, deafness in five, hypoglossal palsy in five, and dysphagia in five. (The old physicians used to describe "hydrophobia without the bite of a dog": this also has been reported in England this summer.) Pain and tenderness in the limbs were noted as marked in eleven cases, and in six there was definite acute ascending paralysis of the "Landry" type. In one case there were trophic changes with gangrene of the finger-tips, and there is reason to believe that an acute central myelitis, "ascending" in type, was present. This case has not however been otherwise included in the above notes.

I have not arranged my cases in groups corresponding to any
definite types, but was much interested in Dr. MacNalty's classification, which does not appear to differ materially from that made by Wickman in relation to Heine-Medin disease.

I confess I was somewhat astonished to hear the suggestion made that cases of encephalitis had never been recorded in association with cases of poliomyelitis. Such have been repeatedly described during the last thirty years (since the time of Medin) as a study of Wickman's and other writings clearly shows, whilst Koplik commented on the many "ponto-bulbar" cases he saw in New York (1916). I have not noticed to-night the mention of a single point not touched upon in Wickman's book; and, although the "clinical differences" between "encephalitis lethargica" and poliomyelitis have been repeatedly referred to, I am unable to gather that there are any to be put forward which are qualitative and not quantitative.

Dr. A. Salusbury MacNalty.

Dr. Crookshank spoke about the points of differentiation and the need for laying stress on clinical evidence in the examination, and was surprised that these did not figure in my paper. For the sake of brevity I did not mention them, but these points had been already touched upon by Dr. Hall, and I have set them out in detail in the forthcoming report of the Local Government Board.
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Section of Neurology.

President—Henry Head, M.D., F.R.S.

PRESIDENT’S ADDRESS.

Some Principles of Neurology.¹

By Henry Head, M.D., F.R.S.

To-night we hold the first meeting of this Section after the close of the greatest war in the history of the world. We have scarcely wakened from the long nightmare in which for more than four years we have lived. As with many of its victims who came under our care, the horror has been so oppressive that the dream still haunts our first waking hours. We of all members of the community have cause to recognize that this victorious peace has been won by the sacrifice of innumerable young lives; and for us older men the future must be dedicated to making the world a better place for those who are to take up our burden.

It was of these young men I was thinking when I meditated the subject of my Presidential Address. For it is exactly twenty-six years since I had the honour of reading my first paper, on "Pain in Visceral Disease," before the Neurological Society. Looking back over a quarter of a century, it seemed to me that I might employ my discourse most profitably by considering certain principles which underlie the standpoint of neurology to-day. I am not going to weary you with an historical epitome. In this quarter of a century there have been no startling discoveries; but I believe that to-day the outlook of the

¹ At a meeting of the Section, held November 14, 1918.
younger generation is fundamentally different from that in which we were brought up, and I shall attempt to indicate the direction in which it seems to me the science of neurology is moving.

The cataclysmic events of the last four years have shaken men's belief in the old order, and medicine has not escaped the universal demand for a restatement of current values. The young are looking to us to enunciate the principles on which our teaching is founded. They are not disposed to accept without criticism conventional explanations. They regard with scepticism the statements, current in text-books, that the manifestations of an epileptic convulsion are the expression of "irritation of the cortical centres," or that aphasia is explained by "the destruction of visual, auditory or motor images." Their curiosity is no longer satisfied by attributing the sensory changes, caused by a peripheral nerve injury, to a "paraesthesia" or "hypo-aesthesia." And indeed words of this order are but anodynes to the conscience. They explain nothing; they serve only to put to sleep the salutary feeling that further investigation is necessary.

Satisfaction with words, that are in many cases nothing more than a restatement of the problem in bastard Latin and Greek, was one of the most remarkable characteristics of the last quarter of a century. No better example could be found than the misuse of the word toxæmia. We are assured that certain diseases are due to this cause; but, although we know much about the pathological changes in the central nervous system, the symptoms they produce, and the course of the disease, the "toxin" is purely hypothetical. Everywhere we find this remarkable tendency to explain the known by the unknown.

Now every student of natural science should retain something of the simplicity of the child. You all remember the story of the Emperor and his new clothes. Two weavers asserted that they had woven a cloth of extreme beauty and fineness; it had, however, one unusual characteristic. It was invisible to those who were stupid, or unworthy of the post they held. Neither the Emperor nor his courtiers could, it is true, see the stuff, but each was anxious not to betray himself, and all were loud in its praises. Royal robes were made of this wonderful material; but, as the procession wound through the streets, a child called out: "Why he has nothing on," and the child's saying passed from mouth to mouth among the crowd. So the young to-day are calling to us to recognize that many of the terms we use so glibly mean nothing. They do not cover the nakedness of our explanations.
This satisfaction with names is apparent in our conceptions of disease. No better example could be found than the customary attitude of clinicians towards syphilis of the central nervous system. Tabes dorsalis and general paralysis of the insane are thus spoken of as "diseases." In reality, there are two factors only in the morbid manifestations produced by syphilis of the nervous system, the character of the infective activity and the site of its anatomical incidence. The *Spirochæta pallida* may attack almost any structure, and the "disease" expresses the functions of the tissues affected. Tabes dorsalis is a convenient term to express the fact that the principal focus of the destructive process lies in the posterior columns, and "general paralysis" simply indicates that it has fallen mainly on the higher cerebral centres. Year by year the line, once so firmly drawn between tabes dorsalis and dementia paralytica, grew fainter as the identity of the pathological process underlying the two conditions was recognized. With the invention of the term "tabo-paresis," the boundaries between the two diseases fell. We now know that any nervous function, from the lowest reflexes to the highest intellectual activities, may be disturbed in consequence of syphilitic infection of the nervous system; and it is only by arbitrary selection that these diverse signs and symptoms can be erected into "diseases." Then again "parasyphilis" was supposed to be the manifestation of a process, syphilitic in origin, but not strictly syphilitic in nature. We now believe that "parasyphilis" is nothing more than an anaphylactic reaction in the tissues of the central nervous system, which have been rendered hypersensitive in accordance with their peculiar lymphatic supply. "Parasyphilis" does not differ from "chronic cerebral syphilis" in any essential pathological particular. It is merely a similar reaction in a different tissue. No better example could be given of the law that, in morbid conditions of the nervous system, the form assumed by the disease is determined by the site of the destruction, whilst the course it runs is dependent on the natural history of the pathological process.

During the last few years the attitude of the neurologist has undergone a subtle change, in regard not only to disease, but also to its general effect upon the patient. At one time everyone was seeking for a sign or a syndrome. Happy the man who could discover a new reflex; it did not matter that he left it unexplained, and failed to correlate it with other ancillary functions. It was a "fact," and to it his name was attached.
But the discovery of such a phenomenon is nothing more than the statement of the terms of the problem awaiting solution. To find its explanation we must look to the behaviour of the nervous system as a whole, and observe how its reactions are changed. In many cases, this leads to the consideration of phenomena which would have been thought frivolous by the older generation. Take, for example, the importance attached to the patient's account of his own sensations, or the diagnostic value now attributed to certain dreams.

In 1901, I described certain mental changes, associated with disease of the heart and lungs, and showed that they formed another aspect of referred visceral pain. This work fell dead; for those who were interested in morbid conditions of the internal organs cared nothing for changes in the mental state of the patient; on the other hand the alienists denied the facts, because of the remoteness of Asylum life from the conditions of Hospital experience. They made no effort to discover whether such remarkable examples of the dependence of mental states on disturbances of bodily functions were really open to their investigation. To-day, however, this work is falling into its place as a small contribution to the relation of body and mind.

Interest in the functions of the nervous system, as revealed by disease, is steadily growing. The English school has always been essentially physiological, from the days of Marshall Hall onwards; but this growing attention to function is mainly due to the teaching of Hughlings Jackson. He sprang from the physiologists of the middle of the last century, and was deeply imbued with the principle of evolution, not only of structure, but of function. But his contemporaries became increasingly obsessed with the study of anatomical and topographical details. As years passed by, the teaching of the day moved away from Jackson's conception of an evolution of function, still demonstrable in the activities of the nervous system. Finally his doctrines were scarcely comprehensible to those of us who were brought into daily contact with him in the wards, seduced as we were by the glib generalities of more popular teachers. But, when we came to think for ourselves, we found that his conception of a functional hierarchy, in which one form of activity was dominated by another, standing higher in the evolutionary scale, explained much that was otherwise inexplicable in the phenomena of nervous disease. This swing back to the physiological aspect was quickened by the exquisite researches of Sherrington, and the issue of his "Integrative Action of the Nervous System," in 1906, marks an epoch in the progress of neurology.
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We then began to appreciate that the existence of the central nervous system was due to the necessity of producing coherent action in a plurisegmental organism. Out of the primitive materials at her disposal, Nature was forced to develop a mechanism capable of integrating the physiological forces evoked by relations with the external world. As the animal rose in the evolutionary scale, the functions required of the nervous system became more and more complex, and out of the anatomical structures of a lower organism had to be developed a machinery capable of exercising these higher activities.

This was only possible by integrating the crude physiological processes excited by external stimulation, or arising in response to the action of internal forces. A prick excites a universal tendency to withdraw the part exposed to stimulation; it can also evoke a highly organized movement, such as the scratch-reflex, capable of removing the noxious object.

But such reflexes are preordained and admit of no choice. It became necessary, therefore, to develop a controlling mechanism that would permit the animal either to attack the stimulus locally, or to evade its action by flight. Finally, with the development of the neopallium was given the power of voluntary choice; the reaction was no longer unconscious, but could be preceded by a definite act of discrimination.

This adaptation to a clearly determined end demands a multitude of adjustments, and such integration is the business of the central nervous system. It can only be carried out by utilizing, in a modified form, functions originally developed for a somewhat different purpose. Thus, the withdrawal movement, which forms the basic reaction to pricking the sole of the foot, is controlled by higher centres in the mid-brain and forms the flexor aspect of the stepping reflex. But, when this higher influence is completely removed, as for example by total transection of the spinal cord, the lower reflex becomes manifested in its primitive form as a pure movement of withdrawal, accompanied by evacuation of the bladder and rectum.

Structure is anchored in the past, and expresses in its anatomical arrangements functions which have fallen into abeyance. Many of them have been carried up to centres of more recent origin, whilst others are exercised normally, in a refined form, under the control of those higher in the evolutionary scale. But nothing remains in its old form, unless it is of utility to the organism under some possible conjunction of circumstances.
Thus the afferent impulses from the viscera do not normally enter consciousness. The eupeptic knows nothing of the processes of digestion, beyond a certain gentle sense of well-being. But, under abnormal conditions, afferent impressions from the internal organs are capable of arousing pain and discomfort. When this occurs, one form of this sensory disturbance is referred to the superficial structures of the body and is associated with areas of tenderness, corresponding to a segmental arrangement of the central nervous system. This is not a "hyperæsthesia," but a tendency to react more violently to any stimulus capable of evoking discomfort. The threshold is not materially lowered, but the vehemence of the response is greatly increased.

Now this over-reaction is associated with reflexes which still reveal their old defensive character. As an answer to visceral pain, the abdominal muscles become tonically contracted, and the leg is drawn up on the affected side. The sufferer is forced to creep into some sheltered place, and to lie curled up, till he either dies or recovers. All capacity to move as a whole is abolished; the reactions of the part affected now dominate the activity of the complete animal. Visceral reflexes, of this order, are antagonistic to general movements, and this is expressed, not only in muscular immobility, but in the mental attitude which accompanies visceral pain.

Pain is the oldest defensive reaction, and potentially painful stimuli are the basis of all primitive reflexes. It is, therefore, of importance for higher development that these impulses should be rendered less effective in favour of those impressions which lead to a more general and discriminative response. But, although they are controlled and even abolished, the mechanism underlying the production of pain must remain in full physiological activity, ready to play its part, should occasion arise, in the defence of the body against noxious influences.

The functions of the central nervous system are not a palimpsest, where a new text is written over an earlier manuscript, partly erased. The more primitive activities have been profoundly modified by the advent of the new centres, which utilize some of the faculties originally possessed by the older mechanism. In many cases the higher function could not be exercised without the existence of these lower powers which it dominates and controls.

When, however, the higher mechanism is thrown out of action the functions of the lower centres are free to exhibit their activity unchecked. Jackson insisted that the manifestations, due to a lesion
of the central nervous system, must be considered from a negative and a positive aspect. Injury to the pyramidal system was shown, negatively, in the loss of the finer voluntary movements; but the activity of the lower centres, released from control, was evident in the spastic rigidity of the paralysed parts. In the same way, removal of the control normally exercised by the cortex over the activity of the optic thalamus leads to the remarkable condition known as thalamic over-reaction; all stimuli capable of evoking discomfort, and in some cases even pleasurable excitation, cause an exaggerated response on the affected half of the body. This is not due to "irritation"; it is due to release. It is the direct consequence of loss of that control normally exercised by the cortex over the activities of the optic thalamus. Once this control is removed by disease, the lower centre reacts unchecked to any stimulus capable of exciting a response.

It has been the custom to call upon a hypothetical "irritation" to explain all such positive manifestations of nervous energy. The violent flexor spasms and the outbursts of excessive sweating, which occur when the spinal cord is injured, have been attributed to this cause. In reality, they are the expression of a diffuse and massive response to stimuli, applied to parts below the level of the lesion; they are manifestations of a primitive mode of reaction, due to removal of control from above. Involuntary movements in hemiplegia, automatic acts following an epileptic attack, the emotional utterances in aphasia, are all examples of the same phenomenon. The condition of sensibility, during the protopathic stage of recovery from a peripheral nerve lesion, is another instance of a primitive mode of reaction, released from control by the tardy restoration of the epicritic mechanism. Normally the massive "all or nothing" response of the more primitive system is held in check by the localized and discriminative sensations, due to excitation of the epicritic end-organs. But, when the one set of afferent impulses is permitted to play on the ultimate sensory centres unchecked by those arising in the higher mechanism, the response becomes massive and diffuse, and extensity becomes of greater import than intensity.

Had we applied Jackson's law, that the functions of the nervous system are integrated on evolutionary principles, neurology would not have made so many excursions into the wilderness. He taught us that a lesion of the cerebral cortex caused disorder of movement, not paralysis of the muscles. This lesson, however, was not applied to the
other functions of the cortex. Much time was wasted on topographical localization of various sensory centres, before the nature had been determined of the functions exercised by the cortex. Now we know that, as far as somatic sensibility is concerned, the cortex is responsible for the appreciation of spacial relations, for the power of responding to different intensities of stimulation, and for the capacity to recognize similarity and difference in external objects, brought into contact with the body. The cortical centres are not concerned with the crude appreciation of touch, pain, heat and cold.

Such principles are not only of theoretical importance; they are perpetually thrust before the neurologist in his daily work. Clinical diagnosis is a by-product of scientific investigation. It is impossible to expose every patient to laborious scientific examination, nor would it serve any useful purpose to do so; but the simple tests, employed in the wards, are valueless until they have been calibrated by more elaborate investigations. The man who says he can obtain all the information he wants, in cases of injury to peripheral nerves, by means of a pin and a piece of cotton wool, depends on someone else to teach him the significance of these empirical tests. They have no scientific value, until the data they yield are correlated with results, reached by methods capable of measurement.

The charm of neurology, above all other branches of practical medicine, lies in the way it forces us into daily contact with principles. A knowledge of the structure and functions of the nervous system is necessary to explain the simplest phenomena of disease, and this can be only attained by thinking scientifically.

Let us then consider some of the leading principles, which the candidate of the future must master in the course of his initiation:—

(1) When any level of activity is attacked, the most complex functions, and those which have appeared most recently, are the first to suffer; they are also disturbed to a greater degree, and to a wider extent, than those which are simpler or more inevitable in their expression.

An excellent example of this rule is seen in the effect of a lesion of the sensory cortex on the appreciation of the spacial aspects of an

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1 The word "level" is employed throughout in a strictly functional sense. Any one anatomical organ of the central nervous system may exercise functions of more than one physiological level. Thus the optic thalamus contains, not only the termination of the fillet, but also the centre for the affective aspects of sensation. For the same reason, a lesion of the post-central cortex not only produces loss of certain sensory faculties, but may also cause hypotonia in the affected parts of the body.
external stimulus. Tests which demand recognition of movement in three dimensions, are more gravely affected than such a comparatively simple one as localizing the position of the stimulated spot; for, with the precautions we habitually adopt, this is an exploration in one-dimensional space. Division of the spacial aspects of sensation according to one, two and three dimensions is obviously arbitrary; but the tests we apply become more or less complex and difficult according as they demand recognition of relations in space of three, two or one dimensions. Consequently, the records obtained by measuring the extent of passive movement which can be appreciated in the affected limb are more gravely and extensively affected than those yielded by our method of testing localization.

(2) The negative manifestations of a lesion appear in terms of the affected level.

Thus, if those parts of the cortex which are associated with speech are injured, the consequences are not an affection of some totally different function, such as visual or auditory images, but are shown in terms of defective speech. The business of a clock is to show the time. Faulty action of the main-spring may cause it to go fast, to go slow, or to stop. But we cannot look at the face of the clock and say "That is strong main-spring time"; all we can say is that the clock is fast. So the negative manifestations of a cortical lesion affecting speech are presented to us in the form of a disturbance of speech, not as a destruction of images.

In the same way, morbid conditions at the reflex level appear as changes in the reflexes; a disturbance of cerebellar activities is seen in inco-ordinate movement; whilst an injury to the sensory cortex affects the discriminative aspects of sensation, a purely mental function.

(3) A negative lesion produces positive effects by releasing activities, normally held under control by the functions of the affected level.

I have already given many illustrations of this law. The phenomena of the mass-reflex after injury to the spinal cord, and of spastic rigidity in hemiplegia, are examples on the motor side; protopathic sensibility and thalamic over-reaction illustrate similar conditions amongst afferent activities.

(4) The functions of the central nervous system have been slowly evolved by a continuous process of development. The methods by which this gradual progress from lower to higher efficiency has been reached are still manifest in the phenomena of its normal activity.
Thus the lowest reflexes give the most definite response; there is little or no choice, the answer is inevitable. They are, as Jackson expressed it, "highly organized." But further development of the needs of the animal demanded a reaction that was less fixed; some variation must be permitted. This led finally to the domination of many reflex manifestations by consciousness. What at lower levels appeared a variable response, became, with the progress of evolution, voluntary control.

(5) Integration of function within the nervous system is based on a struggle for expression between many potentially different physiological activities.

Let us consider the following example, drawn from the afferent side. A metal test-tube, containing water at 45° C., normally evokes a sensation of pleasant heat. But it is easy to show that this temperature, suitably applied, stimulates the cold-spots and then appears to be cold. If, by chance, both the end-organs for the reception of heat and cold are absent, pain is produced. Thus the same temperature is capable of exciting heat, cold and pain; and yet under normal circumstances it evokes a sensation of pleasant warmth. Evidently, the incompatible impulses, due to stimulation of the cold- and pain-spots, are prevented from reaching consciousness.

Such integration occurs at several stages, during the passage of afferent impressions through the central nervous system. Those alike in their qualitative potentialities are gathered together, whatever may have been their origin, at the periphery. Thus all impulses capable of arousing pain come together in the spinal cord. In the same way, there are separable paths for those associated with sensations of heat and cold. Finally, on higher physiological levels is fought out the struggle for final dominance, and, of the warring impulses, one group only excites a conscious response.

The processes by which this integration is rendered possible are three in number. First of all those impulses, potentially of a like sensory quality, are gathered together. Secondly, all impulses capable of exciting sensations of a different quality are rejected by the receptors, which guard each functional level. The third method is manifested in the phenomena of adaptation. Water at 30° C. may appear under suitable external conditions to be neither hot nor cold. But, when the hand has been previously soaked in water at 45° C. for some time, a temperature of 30° C. will seem to be cold. Conversely adaptation to
water at 15° C. converts 30° C. into a warm stimulus. This power of adaptation to the conditions of the environment, and consequent shifting of the neutral point, is one of the most important factors in the mechanics of integration. No stimulus acting over a long period can remain continuously at the same level of efficiency; it leads to a state increasingly favourable to the appearance of the opposite phase of activity. This is the essential condition underlying the tendency to biphasic reaction so characteristic of the central nervous system.

These three processes, acceptance by similarity, rejection by difference, and biphasic dispositions, form the main features of the physiological functions of the central nervous system. They are factors which lead to integration, whether we are dealing with reflexes or with such psychical acts as sensations.

They are interposed between the vital consequences, produced by the impact of a physical stimulus on peripheral end-organs, and the final processes of consciousness. We have no right to speak of psychophysical parallelism; we ought rather to consider on the one hand physiologico-physical responses and on the other psycho-physiological relations.

Thus, between the impact of a physical stimulus on the surface of the body and the movement or sensation it evokes, lie the innumerable reactions of the physiological level. Here the form assumed by the conscious response is prepared to a degree scarcely suspected until recently. Many of the impressions produced by a single external stimulus are incompatible with one another, and some of them must give way in the conflict. But the result of the struggle is not fixed or pre-ordained; it may be reversed by some previous occurrence, which has changed the disposition of the central nervous system. The form assumed by a reflex may be determined by the character of the movement which preceded it, and the phenomena of sensory adaptation depend upon the active influence of the past in present events.

Moreover, the general resistance of the central nervous system to certain impulses varies greatly at different times and in different persons. This is particularly evident in the case of pain. We are all subjected to innumerable painful impressions, which under normal conditions never reach consciousness; but if the resistance, either local or general, is lowered, these impulses may underlie definite sensations of pain.

We neurologists are brought into daily contact with these diverse
functional reactions. We work in the passage-way between the physical universe and the dwelling-place of the mind. We can watch the processes of evolution, visible in the actual behaviour of the central nervous system. We see the coming and the going, and we alone can record which of the many aspirants has conquered the right to enter or to leave that council chamber of human activities.
Section of Neurology.

President—Dr. Henry Head, F.R.S.

Certain Inter-relations between Peace and War Neuroses.¹

By T. A. Ross, M.D.

In the treatment of the neuroses there is one point which is of capital importance—the question as to how relapse is to be prevented. The literature of the last four years has confirmed, what was known before, that any treatment in which the physician believed would be followed by apparent cure.

Many writers, apparently little familiar with the pre-war work, have been satisfied when they had evolved methods which removed the symptoms in hand, and have believed that by accomplishing so much they had done all that was necessary or possible. Their publications, however, have not done much to solve the real problem. Janet [1], writing with prophetic insight a few months before the outbreak of war, said: “Le temple d’Esculape a guéri des milliers de malades, Lourdes a guéri des milliers de malades, le magnétisme animal a guéri des milliers de malades, la ‘Christian Science’ a guéri des milliers de malades, la suggestion hypnotique a guéri des milliers de malades et la psycho-analyse a guéri des milliers de malades; ce sont là des choses incontestables.” Others had written in this strain, yet we have had long discussions about the importance of electricity, about the necessity of accomplishing cure at a single sitting, about the value of an atmosphere of cure—the advocates of a particular method being unaware that though these things might be of moment in their own practice, they were not so of necessity in that of anyone else. The briefest reflection on Janet’s words satisfies us that belief will remove

¹ At a meeting of the Section, held January 3, 1919.
many nervous symptoms, that the success of the method employed is no criterion of its foundation on truth, and that, if we are to have a rational therapy, we must endeavour to ascertain what is behind these manifestations, which are the outward sign of something. We must cease to be content with mere primary cures. If we can do this we shall be in a better way to prevent these too frequent relapses, which have so greatly marred the war work of all the belligerents.

In a recent paper [2] on war hysteria, published in the Lancet, an attempt was made to show that there was no fundamental difference between hysteria and what is termed neurasthenia or anxiety state, that the mere removal of obvious hysterical manifestations was an inadequate method of treatment, and that there was an underlying anxiety to be dealt with. It was pointed out that this could commonly be accomplished by ordinary conversational methods—the elaborate technique of psycho-analysis being rarely called for. It is proposed now to demonstrate that these propositions are as applicable to civilian as to military cases, although the causes of anxiety in them are often further to seek.

Every neurosis is preceded by anxiety, usually by one which tends to be prolonged or to recur frequently. The individual does not succeed in adapting himself to it, and one of two things occurs. Either he struggles to continue in the environment of his misery, in which case he will acquire the symptoms often called neurasthenic, gradually becoming more inefficient and feeling more ill; or some sudden symptom appears such as paralysis, which obviously unfit him for the environment and he escapes from it. This sudden symptom, called hysterical, may be the prolongation of a defence or emotional reaction, or be the continuation of the disability caused by a real, though perhaps slight, injury. Its removal will probably, actually or potentially, place the patient once more in the impossible environment—hence its presence gives little trouble. The patient does not feel ill or concerned about his state. If the symptom is removed, as it can be by any form of persuasion that is believed in, and the anxiety is not dealt with, it will soon recur or the patient will present neurasthenic symptoms.

The suggestion has been made that when an hysteric becomes a neurasthenic after the disappearance of his obvious disability, the phenomenon is not a natural one but an artefact produced by the physician. Such a possibility is conceivable, and requires consideration. In the article referred to stress was laid on this, and on the necessity of
avoiding any suggestion, whether by word, tone or look, which could lead the patient to suppose that any symptom was expected. It would, however, be difficult to prove that one had not suggested subsequent neurasthenic symptoms in any given case where one had removed the hysterical manifestation. Fortunately examples may be found where a spontaneous cure of hysterical manifestations has been followed by the appearance of neurasthenic manifestations, no doctor being in attendance to make evil insinuations.

Case I.—N. W. came under care in October, 1918, suffering from feelings of being run down, poor memory, difficulty of concentration, and depression. These symptoms had been present since September, 1917. He had been buried in December, 1916, and had become aphonic. The aphonia persisted, but he was transferred to the R.A.M.C. in June, 1917, and worked as orderly in a home hospital. The aphonia improved gradually without treatment: when asked when it finally disappeared he replied "about September, 1917," the date of the onset of his neurasthenic symptoms. At the time he was happy and contented in his work, stated that he had no anxieties nor had he had any illness. Confronted with the fact that the onset of the present symptoms coincided with the disappearance of the aphonia, he readily admitted that this was true; but the coincidence seemed to him meaningless. He was told that there was some connexion, and was invited to search his memory to try to ascertain in what frame of mind he was at the time. He remembered that he had been feeling extremely well until the aphonia had cleared, that he had then thought that his life had been rather dull, and that as he would soon be sent back to France and get killed, he would now have as good a time as possible before that occurred. Then his neurasthenic symptoms developed.

Here at any rate the proof is clear that medical interference had nothing to do with the change from an hysteria which did not incommode to an anxiety state which did so exceedingly. Such histories are common enough. The case illustrates also that, without deep analysis, the complex which underlies the whole neurosis may be brought to light. The method employed is as follows: a full ordinary history of the patient is taken. He is listened to for as long as he wishes to speak; he is given the chance of telling everything. He is questioned about any functions he may have omitted. His past history is inquired into. He is then examined clinically from top to toe, and any special examination, bacteriological or otherwise, is undertaken before the attempt is made to follow up any particular symptom by the method about to be described. If the history is taken in such a manner as to give the patient the chance of saying everything he wants to say,
all the facts necessary for comprehension may be obtained by it alone. A patient who sees that his doctor regards his history as important, becomes very communicative and, in the midst of talking of some indifferent matter, may blurt out, "I wish to tell you something I have never told anyone before," and may there and then make a communication which explains everything. The followers of Freud have never tired of saying that an anamnesis is a totally different thing from an analysis, that an analysis reveals matters which no history possibly could. In his book on psycho-analysis, Brill tells a story in support of this. The outcome of a long investigation into a young lady's dreams showed that she wanted to marry a young man of whom her mother did not approve, and that she had sometimes wished that her mother was dead. *Partiuriant montes, nascetur ridiculus mus.* Everyone who takes histories at all is told stories quite as shocking. In most cases, however, the history does not finish the case, and if it has been proved that the symptoms do not depend on physical disease, the patient is told that this is so, and he is asked whether he had any anxiety at the time of their onset. He will probably say that he had not, and is told that he most likely had some anxiety but that he has forgotten it. The investigation so far has probably taken up about an hour, which is enough for one sitting with any patient, and he is invited to think over this matter of anxiety before the next meeting. When this takes place the date of onset of the symptoms is sought for. The patient is asked when he thought that they began. He is then asked whether he is sure that he did not have some of them, say a month or longer before that time. To find this out he is asked to remember where he was at such anterior time, to think what he was doing, and to picture the whole circumstances without any reference specially to the symptoms themselves. Almost always he will report that he had some previous attacks. He is then made to go back in the same way till something is lighted on which is a satisfactory explanation of the phenomena.

*Case II.*—C. P., aged 32, always fainted in hot weather. He believed that this was due to some effect of heat on the strength of his heart muscle. He was asked to think back in the manner described, and it was discovered that ten years previously he had undergone a great strain. At that time he was 23 years old; his father had died suddenly, he was the eldest of a large family, and had become responsible for the others. On a very hot day as he sat pondering over his difficulties he fainted, and afterwards had always done so in hot weather. The cause seemed inadequate, and he was instructed to pursue his research. In a few minutes he said that he recollected that he had often fainted before that date, but that then it had always been in cold weather.
This discovery surprised him very greatly, and of course shattered the weather theory at a blow. He now went back winter by winter, and remembered many faints in cold weather, until he came to a bad day at about the age of 6. He was now asked to recall all about the house he was then living in, and to associate all the surroundings in his mind. Soon he recollected that he had had terrifying dreams the night before, but he could not recall what they were. Then he remembered being awakened out of sleep by a cat jumping on to his face—a sufficiently terrifying experience for a little boy. The next day he had had paroxysms of terror and had fainted often. The weather at the time had been very cold. It is not very difficult to understand why he had changed over to hot weather when his father died. In itself the incident would not have been a strong enough stimulus to initiate faintings, but he had by now learned to meet all difficulties by this reaction—it had become the easy way of escape from a disagreeable environment. He had always ascribed it to weather of some kind: extremes of all sorts are held responsible for illness, and so he passed easily from cold to hot. This is a more plausible explanation of the phenomena than the obvious Oedipus' complex to which the Freudian could soon have led this patient after the first hot weather faint, associated as it was with the father's death. These memories were worked out in about half an hour, which may be looked on as an average time for a task of the kind. When he arrived at the cat episode, he was certain that no recollection of it had come into his consciousness since the time of the event itself.

The question arises whether it was essential to work out the history: in this case it was. The weather was warm when he came under observation, and the intermittent hysterical symptom of fainting was frequent. Vigorous persuasion—i.e., the strong assurance that his heart was healthy, and that he had no disease that could cause fainting, had already been tried without benefit; but after the matter had been sifted to the bottom, though the weather continued hot, there were no more attacks.

A further question arises, why should it be helpful to ascertain the original cause? At the outset these faints were beneficial. They enabled the child to escape for the time from an intolerable memory. Later, anything associated with this memory might have brought it into consciousness; the faints prevented this undesirable occurrence. Though the importance of the incident got less as the years went on, the day when the patient was prepared to face this unknown horror never arrived. As the matter had never been permitted to come into consciousness, as he really did not know that it was there, this is not surprising. But when he was told that undoubtedly something was there, he succeeded in remembering it, and saw that it had no relevance to his life now. With that, fear departed.

It is not always necessary that the physician should be with the patient while the search is being carried out. If there is much pressure of time, and the method is explained, many a patient will find the answer to the problem by himself, and bring the answer in a day or two.
Case III.—C. S. suffered from enuresis. It had been present since childhood at various periods of his life. While he was being examined he was asked to lie on his back. He said that he could not, that whenever he tried to do so, he experienced such a choking sensation that he would be obliged to get up at once. He could assign no cause for either symptom, but in a couple of days he was aware of the explanation. About twenty years before, when he was a little boy, he had been set upon by two bull dogs. He had been pinned to the ground, and his throat had been seized, so that he had felt he would be choked. He had wetted himself, and since then had been liable to repeat this in times of stress, while the other symptom had been present whenever he tried to lie on his back. At this interview he lay on his back with ease.

Case IV.—A. B. came under care on account of neurasthenic symptoms. He underwent a rest cure, in the course of which the explanation was given that his symptoms would disappear if only he would not regard them with anxiety. They disappeared—i.e., he was treated by a method dependent on belief, with the usual result—relief from the symptoms for a time. He then got up and went for a walk. On returning he said that he was as bad as ever: the cure had been a failure. In the course of his walk he had been in a country lane with trees on either side, in which he had been uncomfortable; there he had come across a child; and this encounter had finished him: he had to turn and come home. He then said that a wooded lane was always a difficult place for him; he further said that he could travel in a train only if he got a corner seat: he could not sit on a middle seat. He was instructed in the method of thinking back, and next day he told this story: Some ten years before he had been an engineer in charge of a tropical railway. He lived for months almost alone beside the railway in a lane in the jungle—in a wooded lane. One day when he was recovering from an attack of malaria he had an unpleasant interview with a subordinate. He shuddered because he was weak from his illness, was aware that his face showed tremor, and conceived the idea that the other man thought he was afraid. He was ashamed of this, and knew that for some time after when he had to give orders he did so with his face averted in case it would show tremor. In the gradual development of his illness, these facts had become overlain by secondary symptoms, and in the original history they were missed; but after the removal of these secondary symptoms by persuasion, he still had the primary difficulty of showing his face in a place where the associations were strong. Hence he could not meet the child in the lane; hence he preferred the corner seat in a train, for there one can hide one's face with a book or paper, in a middle seat only if one makes oneself more conspicuous than ever. Next day he went in the lanes and in the middle seat of a train, and his other symptoms again disappeared.

Attention to detail in the history will in a very simple way often destroy the inhibitory beliefs which are preventing the patient from being a useful member of society. The following case, though it
occurred in the Army, is really a civilian case. It is of a kind that was common twenty years ago, that will become common again, one of the class of post-influenzal neurasthenias.

Case V.—The patient was a boy of public school type, who enlisted in September, 1914, and trained till Christmas, when he had influenza, followed by nervous breakdown, which continued for eighteen months. He was discharged from the Army in April, 1915. He was very depressed, could do no work and wished he were dead. With great care and prolonged rest he recovered sufficiently by the autumn of 1916 to take up work in an office, and to re-enlist in July, 1917. He continued to do well till May, 1918, when he was attacked again by influenza. This was followed by a recurrence of the old symptoms, and when he came under care in August he felt that he would soon be as bad as ever; he wished he were dead and contemplated suicide. This sort of case was common in the great influenza years of the nineties, and was then usually ascribed to some effect of the toxin of the disease on the nerve centres; and this boy had been assured that he was one of those who could not stand the influenza poison. Nevertheless he returned to duty, feeling quite well two weeks after coming under care, all that was done being to take his history. The salient points were these: At the original attack he was home on leave: a telegram was sent to his unit to say he was too ill to travel. The police called to verify this. He was overwhelmed with shame, thought he was being looked on as a fraud, got up and returned to the camp, where he was put in bed for a few days. On the journey he thought all eyes were turned on the deserter. After about a week he went on parade and collapsed, his legs giving way under him. When he got to the parade ground he felt that everyone could see through the miserable pretence of his illness and that he was irretrievably disgraced. Now it might be said that the influenza toxin was enough to account for all this. He had got up too soon, he fell because he was weak, and one feels more emotional if one is weak. Something however happened that day which makes this view untenable. He was carried off the parade ground in the morning, in the afternoon he took a ten-mile walk. As soon as these facts were put into juxtaposition, the patient saw that no influenza toxic theory would account for the two halves of that day. He saw that the walk was undertaken to escape as far as possible from the scene of his shame, and that shame was the cause of the ensuing eighteen months of misery. He saw that his recent symptoms were due to fear and that there was no reason why they should continue. He offered to return to duty in a couple of days, but was kept for two weeks for safety.

It is hoped that these examples may help to show that psychological treatment is neither so impossible nor so tedious as some have been maintaining, that the neuroses are not meaningless conglomerations of symptoms, but the expression of well defined anxiety, that the prevention of relapse is bound up with the discovery of this anxiety
and with its abolition or perception by the patient in another light. The hidden memories are often not buried deeply, but they are not lying on the surface.

It may be pointed out that none of these patients showed a sexual complex. They were all relieved of their troubles without touching on this aspect; and as was suggested in the case of C. P., it would have been easy to have insisted on a sexual origin. It is safe to say that a follower of Freud on finding that the series of faintings had begun immediately after the father’s death, would have pursued this clue. Whether he would ultimately have insisted on the validity of the father complex or not is immaterial. If he had he would have missed the point; if in the end he had not, much harm would in the meantime have been done. The case is a warning that there should be no preliminary prejudices about the nature of the complex to be discovered. This is not to say that a sexual complex is not a frequent cause of a neurosis, but that there are many other powerful instincts, which have their effect in the production of these conditions.

I have to thank Major Worth, R.A.M.C., for permission to quote the military cases in this paper.

REFERENCES.


Section of Neurology.

President—Dr. Henry Head, F.R.S.

The Hysterical Element in Organic Disease and Injury of the Central Nervous System.¹

By Arthur F. Hurst, M.D., and J. L. M. Symns, M.D.

(ABSTRACT.)

[Printed in extenso in the Lancet, March 8, 1919, pp. 369-374.]

It has long been recognized that hysterical symptoms may be grafted upon symptoms caused by organic disease. Our experience with soldiers during the past four years has led us to believe that this association is much more common than is generally supposed. We would even go so far as to say that there are few symptoms caused by organic disease which are not liable to be aggravated and perpetuated by suggestion, so that it becomes necessary in almost every case of impaired function to look for an hysterical element which can be removed by psychotherapy.

Disseminated Sclerosis.—It is not an uncommon occurrence to find an extensor plantar reflex, ankle clonus, exaggerated knee-jerk and abdominal reflexes absent in a patient who seeks advice for some early symptom of disseminated sclerosis, such as impaired vision, in spite of the fact that no symptom of paraplegia is as yet present. These physical signs are accepted as proof that the disease has involved the pyramidal tracts, and experience shows that sooner or later the legs will become weak and that severe spastic paraplegia will ultimately develop. Many patients suffering from disseminated sclerosis have a peculiar state of mind, one feature of which is an abnormal degree of

¹ At a meeting of the Section, held January 9, 1919.
suggestibility. It is not surprising, therefore, that hysterical symptoms—symptoms produced by suggestion and curable by psychotherapy—may develop. When the lesion to the pyramidal tracts in such a suggestible individual becomes sufficiently marked to cause some stiffness and weakness in the legs, the stiffness and weakness may give rise to the idea of paralysis, and hysterical paraplegia may rapidly appear. If the patient is seen at this stage it may be impossible to make an accurate diagnosis, for we are face to face with a case of hysterical paraplegia with all the physical signs of organic paraplegia, although only a very small proportion of the incapacity is a result of the organic lesion. Any treatment which is followed by improvement must really act as a form of psychotherapy, and the hysterical paraplegia disappears, leaving behind the physical signs of organic paraplegia and the slight degree of weakness and stiffness which were present before the onset of the hysterical symptoms. This we believe to be the chief explanation of the occurrence of periods of more or less spontaneous improvement, which is such a characteristic feature of disseminated sclerosis. It applies equally to the improvement of other symptoms, such as amaurosis; the slight impairment of vision which results from the earliest changes in the optic nerves sometimes even before any change can be recognized in the disks, suggests a grave loss of vision to suggestible individuals, so that almost complete blindness may occur long before definite optic atrophy is present. The vision may greatly improve again either spontaneously or as the apparent result of some form of treatment, but really as a result of suggestion.

Tabes.—It is very common to find lost ankle-jerks with feeble or lost knee-jerks in patients who have sought advice on account of gastric crises or other symptoms which are due to early tabes, but who have so far had no ataxy nor other symptom which would indicate that the posterior columns are diseased. It is clear, therefore, that the physical signs of disease of both the lateral and posterior columns precede the onset of symptoms. Physical signs of organic disease of the central nervous system are thus qualitative and not quantitative. We have seen numerous cases in which much of the incapacity in a man obviously suffering from tabes was proved to be hysterical by its rapid disappearance under psychotherapy, the symptoms having been suggested to the patient by the slight incapacity which resulted from the actual organic disease. In addition to this auto-suggestion, hetero-suggestion often plays a part, symptoms being unconsciously suggested by the medical officer in the course of his examination. It is, for example,
very easy to suggest Romberg's sign, and we have now seen a number
of cases in which a well-marked Romberg's sign was obviously hysterical.
The improvement in the gait of tabetic patients, which results from the
methods devised by Frenkel, does not in our opinion always act solely
by educating the patient to use his eye, to help his deficient muscle-
sense and to make the most of such muscle-sense as he still has. The
results obtained are sometimes too rapid and too dramatic and can
scarcely be explained except as a result of suggestion, the inco-ordination
being largely hysterical and the nature of the incapacity having been
suggested by the slight degree of unsteadiness actually caused by the
organic disease. One of us (A. F. H.) in 1913, saw a man with all the
physical signs of tabes, who had been unable to walk for six years. He
was brought in a chair to the Guy's Hospital Neurological Department
at 9.30 a.m. After he had been examined he was told that he would
probably learn to walk again if he carried out certain exercises, which
were shown to him. He continued to practise these, and by 12 o'clock
he had improved to such an extent that he could walk the length of the
room, and in a week he was walking about normally. This was regarded
at the time as a triumph of re-education of the deficient muscle-sense,
but the re-education must really have been re-education of the patient's
mind—in other words, psychotherapy—as, if the total inability to walk
had been entirely due to organic changes in the cord, it is inconceivable
that the little muscle-sense still present could have been re-educated to
such an extent in a single morning after lying dormant for six years.

Friedreich's Ataxy.—The following case was a typical example of
Friedreich's ataxy, and until recently we would have accepted all the
symptomatic as the result of the organic changes in the central nervous
system without further discussion. We would have said that Friedreich's
ataxy is one of those nervous diseases in which very little can be done,
and that the patient could hope for no improvement, but would slowly
and steadily get worse. So convinced have we become of the enormous
importance of looking for an hysterical element, even in the most unlikely
places, that Captain Reynell proceeded to treat the patient as if his
incapacity was hysterical, although there was nothing in his mental or
physical condition which gave any grounds for such an idea.

Hysterical Ataxic Paraplegia, associated with Friedreich's Ataxy.—Private B.
aged 25, gradually became unable to walk in the dark two years ago. In
October, 1917, he began to have difficulty in the daylight, and was stopped
several times by the military police on suspicion of being drunk, as his gait
was unsteady. From June, 1918, the ataxy was so severe that he could only
go out in a bathchair. The inco-ordination of the arms and legs then steadily increased up to the time of his admission to Seale Hayne Hospital on October 12, 1918. Dr. W. H. Haupt informs us that the patient's brother, aged 27, is a complete cripple with every symptom and sign of severe Friedreich's ataxy. Our patient's speech was slightly affected, and on admission he could scarcely do anything owing to extreme inco-ordination. He fell frequently when he tried to walk without assistance, and was very unsteady on standing, falling immediately if he closed his eyes. There was slight but definite kyphosis, and the plantar arches were abnormally high on both sides. The knee- and ankle-jerks were completely absent on both sides. The plantar reflexes were extensor. A diagnosis of Friedreich's ataxy was made. Treatment by persuasion and re-education was given, and he learnt to walk fairly well the first day; further improvement followed exercises practised for half an hour three times a day. A week after treatment was begun the gait was almost normal, and unsteadiness could only be detected when the patient changed his direction suddenly. The hands soon became so steady that he developed into a competent potter.

Just as the physical signs of an organic lesion of the pyramidal tract may precede the development of paralysis due to the lesion, and may be associated with hysterical paralysis, persisting after the cure of the latter by psychotherapy, so may these physical signs persist after recovery from organic paralysis, and be associated with hysterical paralysis which develops as the organic symptoms disappear.

Injuries and acute diseases of the brain and spinal cord may result in changes which are to a great extent evanescent. The initial changes may, however, be sufficient to block the transmission of nerve impulses and consequently to cause complete loss of function in the parts which receive their innervation from the affected portion of the nervous system. But the permanent results of the lesion may be so slight that no loss of function persists, although, corresponding with the converse conditions in disseminated sclerosis, the damage may be sufficient to give rise to the permanent presence of organic physical signs.

In the majority of cases the gradual improvement in the actual lesion is accompanied by a corresponding functional improvement. Occasionally, however, especially among suggestible individuals, such as soldiers who are mentally and physically exhausted as a result of the stress and strain of active service, the patient may not realize that the lost functions are returning. The initial incapacity gives rise by auto-suggestion to the idea of permanent incapacity, often aided by the unconscious hetero-suggestion of the physician, and whilst a less suggestible man might recover the use of his paralysed limbs in a few days, the paralysis is perpetuated in the suggestible man by the
development of an hysterical element, which has been produced by suggestion and which can be removed by psychotherapy. In such a case the paralysis remains complete, and although at first it is entirely organic in origin, the proportion of the organic to the hysterical element in its make-up becomes steadily less, and in some cases a stage is reached in which the incapacity is almost entirely hysterical and independent of structural change, although the latter may still be sufficient to give rise to physical signs. A condition may thus occur which is primarily organic, but is ultimately hysterical. Everything of organic origin may disappear, or the residual lesion may be sufficient to produce organic physical signs without any loss of function, or to produce both organic physical signs and some loss of function.

The numerous symptoms and physical signs which are supposed to help in the diagnosis between organic and hysterical paralysis fall into three groups. The first consists of the phenomena which afford visible and conclusive evidence of structural changes in the nervous system, such as optic atrophy and abnormal cells in the cerebro-spinal fluid. Equally conclusive are the second group of physical signs—those which are entirely beyond voluntary control, such as the Argyll Robertson pupil, the reaction of degeneration, and loss of knee- and ankle-jerks. The third group of signs are those which could be imitated more or less accurately by anyone who had studied them, but which would not be likely to occur as a result of auto-suggestion, as the individual would be unaware that such signs accompanied the disease he believed he had. The signs belonging to the last group lose much of their value in the distinction of organic from hysterical paralysis, when the latter has followed organic paralysis, as the characteristics of the hysterical paralysis have been suggested by those of the organic paralysis. An ordinary individual, who develops hysterical hemiplegia, shows no paralysis of his platysma muscle (Babinski's platysma sign), because he is likely to be unfamiliar with the action of the platysma, and, being unaware of its existence, would continue to use it when the rest of the same side of the face was paralysed. But if the hysterical paralysis were a sequel of an organic paralysis, the characteristics of the latter, including paralysis of the platysma, would be perpetuated. Thus in hysterical hemiplegia and paraplegia following organic hemiplegia and paraplegia respectively, most of the third group of physical signs, which are regarded as characteristic of organic disease, may persist. Being caused by suggestion, they are
just as much a part of the hysterical condition as the paralysis itself, and like the paralysis they are completely removable by psychotherapy. Thus we have seen cases of organic paralysis followed by hysterical paralysis, in which the platysma, pronation and fan signs of Babinski, combined flexion of the thigh and pelvis, ankle-clonus quite indistinguishable from that present in organic disease, and various other signs were present; but the condition was none the less hysterical, as the paralysis together with these physical signs disappeared rapidly and completely under psychotherapy. The diagnosis of such cases may thus be extremely difficult, as hysterical paralysis following organic paralysis may not only be associated with permanent physical signs of organic disease, such as the extensor plantar reflex, caused by the residual organic disease, but also with the accessory signs, which are supposed to indicate the presence of organic disease, but which may themselves be really hysterical, being produced by suggestion and removable by psychotherapy.

Combined Hysterical and Organic Hemiplegia of Two Years' Duration following Nephritis; almost Complete Recovery with Psychotherapy.—Private R., aged 29, reported sick on September 29, 1916, with nephritis. On October 1 he had several fits and was unconscious for a few hours. When he recovered consciousness he was suffering from severe right hemiplegia, involving the face, arm and leg, and he was also aphasic. In July, 1917, as his urine was now free from albumin, he was transferred to a neurological hospital in London. The physician, under whose care he remained for more than a year, reports that on admission "there was complete right hemiplegia with aphasia. All deep tendon reflexes much exaggerated, right greater than left, well-marked ankle clonus right side: plantar reflex indefinite; tongue deviation to paralysed side." After a time he regained his power of speech with re-education. In May, 1918, a tenotomy was performed to overcome the flexion of his right knee. As this was not successful, his leg was subsequently twice moved forcibly under anaesthesia. The physician and several colleagues who saw him in consultation agreed that the hemiplegia was entirely organic. In August, 1917, he was transferred to another neurological hospital in London, as an attempt to gain him admission to the Star and Garter Hospital, Richmond, had failed. There was still no improvement when he came under our care at Seale Hayne Hospital in October, 1918. The right leg and arm were totally paralysed and absolutely rigid, the elbow, wrist and fingers being flexed, and the knee semi-flexed. The face, including the platysma, was paralysed, but, as in ordinary organic hemiplegia, the upper part was not involved. The deep reflexes of the arm and leg were much exaggerated on the right side and slightly exaggerated on the left, and well sustained regular ankle clonus was present. The abdominal reflex was absent on both sides. The plantar reflex
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could not be obtained. The paralysis was treated by persuasion and re-education, and in two-and-a-half hours the patient was able to extend his leg and arm and move them slowly in all directions. At the end of another hour he was able to stand by himself, and next day he was able to walk. This result was obtained without causing any pain to the patient in spite of the extreme rigidity. The exaggerated deep reflexes and ankle clonus remained unaltered, and an extensor plantar reflex was now obtained on the right side. An attempt was next made to overcome the facial paralysis, and in ten minutes there was marked improvement. After forty-five minutes treatment the facial paralysis had disappeared and the platysma was contracting normally. The patient is now able to use his right hand for all ordinary purposes—e.g., writing and needlework, and he walks with only a slight limp.

Hysterical Hemiplegia with Persisting Signs of Organic Disease following Concussion by Shell Explosion, cured by Psychotherapy after persisting for eight months.—Private T., aged 22, was admitted to Scale Hayne Hospital on June 20, 1918, for hemiplegia of the left side, which developed as a result of being blown up by a shell eight months before. He could only stand with assistance and was quite unable to walk. There were definite signs of an organic nervous lesion: the left plantar reflex was extensor, the abdominal reflex was absent, the ankle-, knee-, wrist-, and elbow-jerks were much exaggerated on the left side, and well sustained ankle clonus was present. In spite of this the patient was treated by vigorous persuasion and re-education. Within an hour he was made to walk and run, but it took five or six days to develop a normal walk. A fortnight later a distinguished neurologist, who was visiting the hospital, watched him playing billiards, and was asked to guess which had been the hemiplegic side. But the functional recovery was so complete that he was unable to do so, although all the signs of organic nervous injury were still present, and had not altered when the patient was discharged from the hospital feeling perfectly well two months later.

Combined Hysterical and Organic Hemiplegia of Eleven Months' Duration, following Gunshot Wound of the Skull; great improvement with Psychotherapy. —Lance-Corporal B., aged 23, was wounded in the right parietal region in December, 1917, and was admitted to a general hospital in France with left-sided hemiplegia. On December 28 an operation was performed and a small crack in the skull was found; some bone was removed, but no injury to the dura mater was discovered, and pulsation was normal. The bone was replaced and the wound sutured. On July 17, 1918, he was transferred to Scale Hayne Hospital. The arm was rigidly extended at the elbow, the fingers were extended at the metacarpo-phalangeal joints, but flexed at the interphalangeal joints. The leg was rigidly extended at the hip and knee, and the foot was fixed in a position of extreme plantar flexion. The deep reflexes were increased on the left side and well-sustained and regular ankle clonus was present. The big toe did not take part in the plantar reflex, but the fan sign was present on the left side. The degree of rigidity was extreme and the strongest effort
was required to bend any joint. The patient was treated by persuasion and re-education; movement in all joints except the shoulder was obtained in one sitting of four hours without much discomfort to the patient. The next day he developed pleurisy with effusion, which necessitated the postponement of further treatment for over two months. Psychotherapy was then continued, and he is now able to walk well, and his arm movements are almost perfect. At the present time (December 30, 1918), there are no signs of organic disease, the ankle clonus and increased deep reflexes having disappeared.

A series of cases of paraplegia were next described, the majority of which had followed spinal concussion, but a few of which had followed actual wounds of the spine. Rapid and complete functional recovery occurred in all as a result of psychotherapy; in some no physical signs of organic injury were ever present, in others they were present at first, but later slowly disappeared, often long after the paralysis had been cured; in the remainder the physical signs persisted to the end. The more common type was with extensor plantar reflexes, ankle clonus and exaggerated knee-jerks; in a few these signs were associated with evidence of lower neuron paralysis of the hands and forearms. The other type showed lost knee- and ankle-jerks, although the cerebro-spinal fluid was normal, the damage to the posterior columns being clearly a result of the spinal injury.

It is usually taught that incontinence of urine is never hysterical. But although the idea of incontinence is very unlikely to suggest itself to an individual spontaneously, we have seen three cases in which severe hysterical incontinence occurred as a sequel of the incontinence caused by the temporary organic changes resulting from concussion of the spinal cord.

_Hysterical Incontinence of Urine and Hysterical Paraplegia following Concussion of the Spinal Cord with Organic Physical Signs._—Private M. P. was buried in a trench in France in July, 1916. He was dug out and admitted to hospital suffering from weakness of his legs and incontinence of urine. This condition continued until he was admitted to Seale Hayne Hospital on August 2, 1918. On admission he could only walk with a shuffling and tremulous gait, and was wearing a urinal day and night on account of incontinence. The abdominal reflexes were absent on both sides, the knee-jerks were slightly increased and slight ankle clonus and a definite extensor plantar reflex were present on both sides. The incontinence was treated by persuasion and re-education. In a short time it was controlled during the day, but at first persisted at night. He was then treated by hypnosis, and after three weeks was completely cured. With the relief of the incontinence the paraplegia also disappeared, and he can now walk quite well, although the physical signs are unaltered.
In the following case blindness of a character generally supposed to be typical of organic disease was perpetuated as an hysterical symptom after the initial organic changes in the brain had disappeared.

Partial Hysterical Blindness, following Organic Blindness caused by a Wound in the Occipital Region and associated with Hysterical Deafness.—Private W., aged 22, was wounded over the right occipital region on June 7, 1917. He was unconscious for five days and was then trephined. On admission to Netley on July 6, 1917, he was completely deaf in both ears, but as the vestibular reactions on rotation were normal, the deafness was regarded as hysterical. He had difficulty in seeing and held anything he wished to read low down on the right side. On further examination it was found that he was also totally blind, except in the right lower quadrant of the field of vision of both eyes, the blindness being what might be expected to result from the wound over the lower part of the right occipital lobe near the middle line, which would be likely to involve the left lobe also to a less extent. An attempt was made at the end of August to cure the hysterical deafness by a pseudo-operation, the patient being told that a cut behind his ear would certainly restore his hearing. Nothing was said to him about his blindness, which was regarded as organic. The "operation" resulted in immediate improvement in his hearing, as it was now possible to carry on a conversation with him by shouting. Quite unexpectedly it was found that his vision was now absolutely normal, the blindness having been cured by the suggestive effect of the "operation." It must, therefore, have been due to perpetuation by auto-suggestion of the organic blindness, which was caused by concussion and not by destruction of the occipital lobe.
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President—Dr. Henry Head, F.R.S.

Case of Syringomyelia (alleged Plumbism). ¹

By F. Eve, M.D. (Hull).

(Introduced by Dr. H. G. Turney.)

The patient was a fitter, aged 26. Manifestation of the disease first appeared three years ago when his present rather falsetto hoarseness began. This symptom is due to paralysis of the left vocal cord. He worked in lead occasionally from March, 1917. Nine months later his left wrist “dropped,” this preceded by weakness and wasting in both hands and forearms, œdema of the hands and “claw hand.” On the strength of this he was awarded compensation for plumbism. Under splinting and massage the left “dropped wrist” was cured in nine months. Last July, a slight spastic paresis of the legs showed itself by a dragging of the left foot and ankle clonus. He now shows none of the anaemia, blue line or supinator palsy usual in lead palsy. There is much paralysis and wasting of the hand and forearm muscles, but the left flexor carpi radialis is remarkably spared. Heat and pain are not felt over the hands, arms, the left side of the head, and tongue, but touch is well recognized. As he does not feel pain, he has got into the habit of gnawing his finger ends and nails, which are raw and atrophic. The left pupil is much smaller than the right. Rotary nystagmus appears when the eyes are directed to the left. There is slight paresis of the left palate. The plantar reflexes are absent.

The case is of interest from the well-marked bulbar involvement; it also raised the question whether lead had anything to do with his illness. The only opinions expressed have been negative.

¹ At a meeting of the Section, held February 13, 1919.
A Rising-floor Cell to facilitate the Microscopy of Cerebro-spinal Fluids.

By F. Eve, M.D. (Hull).

(Introduced by Dr. H. G. Turney.)

This little device consists of a cylindrical cell, 1 cm. in depth, on the floor of which a cover-glass is placed. This is filled to the brim with cerebro-spinal fluid (fresh and uncontaminated with blood cells). This is put aside for two hours, till the leucocytes and bacteria sink on to the cover-glass, or until the spider-web clot has formed throughout the fluid, as occurs in many cases of tuberculous meningitis. The milled head is then slowly turned without shaking. This causes the floor of the cell to rise gently and the cover-glass to be left high and dry. The residual fluid has no option but to retire beneath the rising floor of the cell. When the screw has been fully unscrewed it is detached and placed on the hearth. The cover-glass now finds itself on a little metal table where it can be dried in front of the fire. It is then well fixed by heat and absolute alcohol. Staining should be done gently and without warming, lest the cells should be washed off. Under the microscope every cell and bacillus contained in a column of fluid 1 cm. in depth can then be scrutinized. If absolute figures are desired the area of the microscope field can be found by counting the number of squares it contains when viewing a haemocytometer chamber. The apparatus is almost indispensable for dealing with the spider-web clot in tuberculous meningitis, as the clot contains the tubercle bacilli.
and ravel up into a hopeless mass when any attempt is made to spread it evenly on a microscopic slide.

My thanks are due to my engineering friend, Mr. F. M. C. Lewis, for the improved design. It is made by Messrs. Allen and Hanbury.

Two Cases illustrating Effect of Severance of the Brachial Artery.

By A. Rocyn Jones, M.B.

Case I.

A. S., private, March 21, 1918: Gunshot wound of right upper arm; fracture of humerus; brachial artery severed. Operation same day; brachial artery ligatured; limb put on to splint with elbow fully extended. This type of splintage was kept up for four months by which time healing was complete. Massage was started about four weeks after healing. From the beginning the forearm and hand were cold, blue and numb, but at no time has the patient complained of severe discomfort or pain. He was evacuated to a military hospital at Glasgow and whilst he was under treatment there, the pulps and terminal phalanges of the four inner fingers came away.

He was admitted to the Royal National Orthopaedic Hospital on January 7, 1919. At that time there were two linear scars in the right upper arm, one of which was on the ulnar aspect with its centre 2½ in. above the internal condyle of the humerus. A mass of callus was felt on the outer side of the arm: the head of the radius was dislocated forwards interfering with flexion movements at the elbow and almost abolishing pronation-supination. The semipronated forearm and hand were cold and blue and dorsiflexion at the wrist was limited owing to slight forward subluxation of the hand. The fingers were stiff and flexed. The terminal phalanges were not present but the nails were brittle and atrophic and strongly curved towards the palmar aspect of the hand. All the muscles of the forearm were wasted, those of the lower half to a greater extent than those of the upper half. The inter-osseous spaces were very prominent and all the muscles of the hand were ill-developed and flabby. Adduction and abduction of the fingers and thumb could not be performed. In neither radial nor ulnar arteries was any pulse palpable. To all forms of sensory testing (pin-prick,
pressure, cotton wool, hot and cold tubes) the patient responded slowly, but the answers obtained were correct except over the terminal phalanges of the hand where neither prick, pressure nor cotton wool were appreciated. Between the thumb, little finger and the other digits no gross differences in sensibility were discovered. No over-reaction of the protopathic type was present.

Treatment with hot water baths and massage was now commenced. At the present time a pulse can be felt in both the ulnar and the radial arteries. The range of possible voluntary movement at all the joints has increased. The muscles have filled out considerably, more especially those supplied by the ulnar nerve. In sensory testing in the distribution of the ulnar nerve and of the radial nerve there has been much improvement, but in that of the median nerve this improvement has been delayed relatively and now there is a state of "protopathic over-reaction" in the part of the palm supplied by the median nerve and a considerable amount of sensory loss to all forms of stimulation over the distal portion of the median fingers.

It is proposed to continue the treatment with the hot water baths and massage until the circulation is still further restored and then to remove the head of the radius with a view to improving the movements of flexion at the elbow and those of pronation and supination.

**Case II.**

A. T., private, October 14, 1918: Gunshot wound just above the left elbow. Directly after his wounding the patient noticed that his hand had become "dead" and "as though electric shocks were passing down the forearm and hand"; the brachial artery was spurting blood. He at once applied digital pressure to the brachial artery above the wound. After a few minutes he was relieved of this by an officer, who kept up digital pressure until a pebble and bandage as a tourniquet were applied.

Operation: On the same evening he was operated upon at a casualty clearing station, the field card stating that the brachial artery was found to be lacerated and the median nerve bruised; the artery was tied. The limb was kept wrapped in cotton wool for some weeks.

On January 31, 1919, he was sent as a pensioner to the Royal National Orthopaedic Hospital. There was a star-shaped, keloid-like scar internal to the insertion of the tendon of the biceps and a small entrance scar 2 in. above and slightly behind the internal condyle of the
humerus. There was no evidence of any fracture. Full flexion of the forearm was possible, but full extension fell short by about forty degrees; supination was also incomplete. The fingers were extended, blue and tapering; the nails were oval, dull, and extremely pale. The finger-joints were stiff and only allowed of a moderate amount of flexion and that at the metacarpo-phalangeal joints; at the interphalangeal joints little or no movement was possible. Adduction and abduction of the digits was possible. The radial pulse was difficult to find and almost imperceptible; no ulnar pulse could be felt. To sensory testing there was an area on the index and middle fingers completely insensitive to pin pricks and outside this, extending over the distal portion of the palm, a larger area insensitive to cotton wool. Pressure could everywhere be appreciated.

Dr. George Riddoch showed a Case of Acromegaly, which will be reported later.
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President—Dr. Henry Head, F.R.S.

Some Biological Effects due to High Explosives.

By Alfred Carver, M.D., Captain R.A.M.C.(T.C.), and A. Dinsley, Lieutenant R.A.O.C.

Quite early in the course of the War the symptoms of nervous origin exhibited by a group of the soldiers who had been subjected at close range to the detonation of high explosives excited widespread interest and attention. Never before had such a mass of material been presented for investigation, but the underlying changes of the conditions, then termed "shell shock," were wholly obscure. Mott, in two cases which came to autopsy, described multiple punctate hæmorrhages in the substance of the brain; and the view that the condition was due to more or less gross structural damage of the central nervous system was, for a time, widely held. As time went on experience showed such a view in its entirety and for the majority of patients at least, to be untenable, and the pendulum has gradually but steadily swung to an opposite extreme. At the present time the very term "shell shock" is taboo, and in the Services has been replaced by the equally unsatisfactory one of "neurasthenia." This change in terminology indicates very clearly the present general attitude of workers and observers on this subject, an attitude which evokes emotional factors as the sole cause of all neuroses, both those of peace and war. Far be it from us to deny the important influence of the emotions in such conditions; for the evidence that the emotions in many, if not in the majority of patients play a fundamental part appears conclusive. At the same time one meets

1 At a meeting of the Section, held March 18, 1919.
with a large number of patients suffering from the neuroses of war in which the emotional factor does not appear to be primary, and cannot be considered as causative. In such patients, therefore, a purely psychogenic explanation will not suffice. Observation of this group reveals that physical or "commotional" factors have been operative.

First, one can distinguish a subgroup in which the ætiological factor is "direct concussion" produced by the impact of missiles, either fragments of shell or earth—upon the tissues overlying the central nervous system, but in which the brain, cord, and membranes have not been damaged by the missile. The existence of this group has been noted, but, in this country at any rate, its importance has been largely overlooked, and the symptoms which distinguish it from the psychogenic group have not been sufficiently generally recognized or clearly perceived to insure its acceptance as a definite type. We consider that the symptoms of "direct concussion" are distinctive and shall refer to them in detail later on.

A second subgroup which has been discussed, mainly by the French, is that which may be termed "indirect concussion." Here, although there is no direct concussion in the ordinary sense of the word, the patient was sufficiently close to the bursting of a shell to be subjected to the violent variations in pressure which accompany the detonation of high explosives.

A third subgroup, and one to which I have failed to find any reference in the literature, is that in which the ætiological factor appears to be the fine, extremely rapid vibrations which are one of the less known results of the detonation of high explosives. These vibrations are capable of agitating the nervous system in such a way as to produce a condition closely resembling that which results from excessive emotion.

If the above contentions be verified the neuroses of war may have a psychogenic, a physiogenic, or a mixed ætiology. The thesis to be put forward in this paper as a result of experiments is, that not only is the hypothesis of a purely psychogenic origin inadequate to account for all the neuroses of war, but that definitely physical factors also play an important pathogenetic rôle.

Whatever be the origin of the "shock" it must be assumed to have some physical basis just as must all normal processes, though this simple deduction seems often to have been lost sight of. As Sir Purves Stewart has put it, "the old definition of a neurosis as a nervous disease devoid of anatomical changes is inadequate. Disease without
some underlying physical basis is inconceivable. The lesion need not
be visible microscopically; it may be molecular or biochemical."

As to the nature of the changes continually taking place, either in
so-called "normal" or in diseased processes, practically nothing is
known; yet that emotional shock does in some way, no matter how
slight, modify them is generally conceded. It is not difficult then to
allow that the grosser physical agitations, such as those referred to
above, are likely to exert an even more profound influence upon the
delicate adjustment within the nervous system. Whether this be
brought about by rendering the subcortical centres more excitable,
or by inhibiting the higher centres, or by any other means, the clinical
picture is one of a relative inefficiency of normal cortical control.

The fact that, at first sight, the immediate syndrome in typical
effects of purely emotional shock is opposed diametrically to that in
the commotional group—viz., excitation as opposed to stupor or loss of
consciousness—need not be interpreted as implying that the underlying
physiological events differ widely either from one another or from the
normal. It would seem more helpful to regard them as a matter of
degree rather than of quality. This speculation is merely offered as a
suggestion, and one must frankly admit that, in spite of many efforts,
no knowledge which can be regarded as final has been acquired in this
line of investigation.

The conditions under which "shell shock" arise in man render
accurate observations extremely difficult. It, therefore, seemed advisable
to carry out experiments upon animals in such a way that the weight
of the explosive used could be kept constant; its nature and composition
known but varied at will; the actual distance of the animal from the
centre of detonation measured; and other factors also accurately taken
into account. With this object in view the experiments detailed below
were undertaken. Before setting them out, however, it will be well to
consider briefly what is understood by an explosive and what are its
essential properties.

An explosive may be defined as a substance capable of being
suddenly transformed into hot gases, which at the moment of their
formation tend to occupy a far greater volume than the original
substance, and consequently exert a great pressure, in every direction,
upon their immediate surroundings. The pressure exerted by some
explosives exceeds 300 tons per square inch. The amount of destruc-
tion which can be accomplished by an explosive under the most
favourable circumstances is known as its "power." A rough empirical
measure of this "power" is obtained by multiplying the volume of the
gas produced by the heat generated in the process. The shattering or
disruptive effect, "brisance," is, however, mainly dependent upon the
rapidity with which the change takes place. A slow explosion has the
effect of a relatively prolonged push, a rapid one that of a sudden sharp
blow as from a hammer. In the case of "high" explosives the
chemical change is propagated as a wave—"the wave of detonation"—
through the mass, at a velocity exceeding 10,000 metres per second.
The criteria which determine whether an explosive shall be called
"high" are thus the production of a great volume of hot gas in so
exceedingly short a period of time as to be practically instantaneous;
the shorter the time factor, the "higher" the explosive. The force
that must be transmitted to any given explosive in order to cause
detonation is termed its "tonal standard." For example, if a sudden
blow approximating 250 tons per square inch is required, to
detonate an explosive, this constitutes its "tonal standard" or
degree of sensitiveness. The disruptive effect of high explosive may
be calculated by multiplying its "power" by its "velocity of detona-
tion." When a high explosive is detonated there arises first a sudden
terrific blow, which exerts a compressing and shattering force upon
its surroundings in every direction. This is instantaneous and is
immediately followed by an equally sudden decompression; thirdly,
rapid oscillatory or vibrating movements are set up, which only
gradually die down. The nature of these vibrations requires further
investigation, but it seems probable that in addition to the material,
molecular vibrations, there arise also ethereal vibrations of somewhat
the same order as those utilized in wireless telegraphy. However this
may be, each of the three results just noted requires analysis when we
are considering the effects of detonation upon living organisms. Another
interesting and important property of explosives is that when a wave
of detonation, started at a considerable distance away, is transmitted to
a second suitable source, detonation of the latter may result. Even if
the distance be too great or the transmitted detonating wave, for any
other reason, inadequate to excite immediate detonation in the second
substance its physical state becomes altered in such a fashion as to
render it more unstable; and a series of detonating waves just below
the tonal standard will eventually cause its detonation.

The above are briefly some of the more essential general charac-
teristics of high explosives. For military purposes these properties have
been taken advantage of in the filling of shells, bombs and grenades in
order to secure (1) "fragmentation" of the container, shell or bomb, the lethal effect in this case being dependent upon the flying fragments; (2) "brisance," or disruptive effect due to the violence of the detonating wave; (3) a deliberate combination of (1) and (2).

There is a further effect which will be discussed later—viz., the purely demoralizing effect which is produced by the wave of detonation outside the sphere within which it causes any demonstrable physical destruction or wounds. Evidence will be brought forward to show that the Germans paid special attention to this peculiar effect and arranged their shell-fillings in such a way as to enhance it. This is fully in keeping with other of their methods of warfare and might be described as "frightfulness by detonation." With the obvious effects of "fragmentation" this paper does not concern itself. Attention will be directed entirely to the effects of detonation, and even here mainly to the indirect rather than to the more obvious direct effects; the latter of which will receive merely passing mention. If a high explosive be detonated at a point X certain zones can be mapped out in an area around this point. Three such zones may roughly be distinguished, their radii depending, amongst other things, upon the weight of explosive employed. It will, perhaps, be helpful to give a short general summary of the findings which led to the establishment of these zones before entering into details of actual experiments. Zone A is delimited by the extent of obvious gross disruption, and may be termed the "zone of brisance"; within it a crater is formed, barbed wire and other obstacles are blown aside, broken and distorted; animals usually killed and lacerated; other high explosives immediately detonated. Outside this zone is a second one, B, which must be rather more fully considered. It may be termed the "zone of decompression," for the disturbances in biological processes which take place within it seem largely attributable to this factor, though coarse jarring and shaking movements are also of importance. In zone B the gross effects of shattering and disruption are no longer evident; animals placed in it and protected from flying fragments arising from zone A are generally "concussed" without showing signs of any external injury; only sensitive explosive substances, the "tonal standard" of which is somewhere near that of the primary explosive detonated, and, even their explosion does not invariably occur. The outer boundary of this zone cannot be sharply defined, but beyond it there can be named a zone, C, in which the effects of detonation are modified further. In zone C the effects upon animals vary considerably, but as
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a rule a short transitory state of stupor followed by a stage of excited confusion is witnessed. High explosives placed in zone C only detonate if they have the same tonal standard as the substance employed at X, yet if they are left in position during a series of observations the fact that their stability has been thereby diminished can easily be demonstrated experimentally. Explosives after subjection to this treatment are always dangerous and require very careful handling. The outer margin of zone C shades off gradually.

Experiments were first carried out in water, the test animals being fish—perch. A depth charge of 6 oz. gelignite was used. Fish in zone A were rendered obviously hors de combat, showed gross lacerations, especially of the gills, and were sometimes torn in pieces. These findings might well have been anticipated, and may in any case be passed over as not germane to our subject. Fish, in zone B, after the detonation, floated in an almost vertical position, but slightly inclined with their ventral surfaces uppermost. At this early stage their mouths protruded just above the surface of the water, and the whole of their bodies were rigid; if the fish were pushed down to a depth of about 2 ft. they sank slowly to the bottom of the tank and there remained. The first reflex shown by fish in this zone was erection of the dorsal fin upon stimulation of the skin to either side of it. Next there occurred spasmodic movements of the opercula and gills: these were violent and irregular. In those fish which eventually recovered, swimming movements began to be possible about half an hour after the detonation. At first the movements were only carried out in response to stimulation of the abdomen—stroking with a piece of stick—and they carried the fish only forward in a direct line; no turns were observed at this stage even when an obstruction was encountered. These first swimming movements occurred with the ventral surface uppermost and lying almost flush with the surface or the water. Thus it can be said that up to this stage the fish had lost their power of equilibration and with this the whole of their orientation in life. The majority of the perch which attained the power to swim, as just described, gradually became more and more active, and after passing through a side-uppermost position, and remaining quiescent for long pauses, they righted themselves and began to swim about in the ordinary way. In about twelve hours after the detonation those fish which had not entirely succumbed were to all appearances completely normal. Dissection of the dead fish showed, as a rule, haemorrhages in the upper region of the spinal cord
and medulla, congestion of the foreparts of the brain, and a number
of haemorrhagic points in the mid-brain and basal ganglia region. The
gills and other viscera were congested and often showed areas of
haemorrhage. The extent of these lesions was naturally variable.
Dissection of fish taken at random immediately after the detonation
revealed either varying degrees of the above-mentioned lesions or,
by macroscopic examination, nothing abnormal was discovered. It
is regrettable that at the time these experiments were carried out
we had no facilities for preparing sections or making microscopical
examinations of our material.

The results of detonation towards the inner boundary of this zone
would seem to be due to sudden decompression and to the rude shaking
which the creatures received. Towards the outer margin of B zone and
the inner region of C zone the effects upon the fish could not be sharply
distinguished, the severity of the symptoms gradually diminishing and
the anatomical lesions becoming rarer with the radius of the circle
described from the centre of detonation. Fish well out in C zone swam
about vigorously in a disturbed and excited manner, but, though they
dashed about wildly and apparently at haphazard, it was generally
noticed that the movements which they made carried them away from
X, the centre of detonation. A few came to the surface and splashed
about; when netted no physical abnormality could be demonstrated,
and those returned to the water soon behaved in a completely normal
manner.

In considering the effect upon fish of vibrations thus set up in
water one must not be unmindful of the great development in these
creatures of sense organs which are directly susceptible to stimulation
of this type. We refer to the pre-oral mucous canals, which enable a
fish deprived of its eyes to guide itself along tortuous passages by
the delicate appreciation of the reflected pulses of water; also the
lateral line organ which subserves similar functions. It is, therefore,
conceivable that the effects of violent vibrations may be in part due to
excessive stimulation of these numerous special sense organs; the fish
being, as it were, swamped by the flood of incoming impressions. If
this be so, the shock, though physical, would be more accurately
described as of neurogenic than of commotional origin. Probably both
factors play a part, and in any case it is impossible to discriminate
between them in the experiments as carried out. It is well known
that in man unconsciousness can be produced by purely reflex stimu-
lation, of the skin, of the eyes, of the ears, &c.—e.g., by bastinado, by
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extremely bright lights as of furnaces, and by rotation in a chair. In such cases the whole ego is swamped by an excessive flood of afferent impulses which produces a condition of neurogenic shock, there being no "commotio." We have no evidence which leads us to think that neurogenic shock as above described plays any appreciable role in the causation of the war neuroses, and shall not refer to this aspect of the subject any further.

Experiments with mammals—rats and mice—on land were carried out in the following ways: Perforated zinc cages were used, partly to fix the position of the animals and partly to protect them from flying fragments. These cages were distributed at measured distances about the point X. The charge was standardized to 3 oz. of explosive. In one series of experiments the charge was buried in a hole 6 in. deep in order to imitate on a small scale a shell which penetrates the ground before detonating (delay action). In this case a major portion of the force seems to be directed conically in an upward direction, and there is less lateral spread along the surface of the ground. Using 3 oz. of gelignite in this way, a crater measuring about 18 in. across was formed; the B zone extended roughly 12 in. outside this, and the C zone another 20 in. or more. That is to say, that under these conditions demonstrable results were produced at a distance of from 4 ft. to 5 ft. around X. In a second series of experiments the charge was not earthed, but simply laid upon the surface of the ground in order to imitate on a small scale a shell exploding immediately upon light contact (non-delay action). In this case the crater formed was much smaller, but the force expended in a direction parallel to the surface of the ground more extensive, and the boundaries of the zone lay further out from X. With these exceptions the results in the two series of observations were essentially the same.

In zone A cages in and immediately outside the crater formation were simply blown away and distorted; the contained animals were killed or severely wounded by direct violence. These, together with animals wounded by fragmentation or the shattering of the cages, will not be further considered. Those animals which escaped this direct trauma were invariably rendered unconscious, their eyes were blood-shot, and frequently there was external haemorrhage from the ears, nose, and mouth. On dissection varying amounts and degrees of internal haemorrhage in the viscera and central nervous system were revealed; the alveoli of the lungs were almost always found to be to a certain extent ruptured. These gross changes would seem in part
attributable to the direct blow of the displaced air, which strikes like a solid substance, and in part also to the effect of exceedingly rapid decompression immediately succeeding this. At the inner margin of zone B the findings, though in the main similar to those just described, were very much less in degree. When cages were shielded by a small mound of earth from the direct impact of displaced air, the animals were sometimes found to be dead, even though they exhibited no signs of external injury, except for injection of the conjunctive, and upon dissection the only abnormality disclosed by coarse examination was a general capillary engorgement, especially noticeable in the central nervous system and meninges. Further out, in zone C, a state of stupor or transitory loss of consciousness was an almost constant result. The duration of this state varied inversely with the distance from X. During the course of recovery twitching movements of the limbs were often present, and then the animal, if stimulated by appropriate physical agents, showed massive and exaggerated reflex reactions. For example the application of a pin-prick to one hind limb might at this stage be followed by a bilateral flexion of the hinder extremities, or even by a generalized convulsion. There was frequently considerable difference in the activity of the reflexes on the two sides of the body—a hemiplegic distribution—but as a rule the fore limbs were more severely affected than the hind limbs, and showed weakness, as evidenced by inco-ordination and dragging, after the latter had regained normal movement. Reflex responses, though vigorous, died away quickly, but they were often succeeded by a weaker repetition even when no fresh stimulus was given. Rapid fatigue of the response on repeated stimulation was a noticeable feature. By dissection no gross macroscopical evidence of internal injury to such animals was discovered, though in some cases there was capillary engorgement as in animals nearer X. Whether this should be taken as indicating that vasomotor changes play a part in the production of the associated phenomena we have no means of determining. Animals left alone during the stage of recovery gradually began to behave in a manner more normal to them, though for a considerable time their conduct, postures, and gaits were awkward and abnormal. The severity of these disorders varied greatly from animal to animal, and consequently it was impossible to delimitate precisely from biological observations the borders of the several zones. In the C zone, animals crouched down and huddled themselves together whenever a detonation took place; after this state of diminished activity, a state of apparent excitement with increased restlessness
was generally observed. If, however, animals were left exposed in this position during a series of experiments they invariably crouched down as flat as possible, and seemed to be in an absolutely stuporose condition. When liberated, they made no efforts to run away, but remained, for a long time, motionless in a huddled up posture. The interpretation of these latter abnormalities in behaviour is difficult. Should "fear" be allowed as the main cause, or are other more material factors also at work?

If instead of employing, in the experiments, a single explosive substance at the point X, one detonated a composite or mixed charge—standardized to 3 oz. in every instance—all the above mentioned effects became more pronounced. The increase of effect was particularly noticeable in the C zone and its immediate surroundings. In this connexion it is interesting to record some observations carried out upon physically and mentally fit men of an Ammunition Proof and Demolition Section during the course of their ordinary duties. The staff was drawn from infantry-men who had some technical knowledge. They were sheltered in a well-protected dug-out about 100 yds. distant from X, the centre of detonation. In this case X was a large crater blown in the centre of the demolition ground, and contained the official maximum weight for each single destruction—viz., 200 lb. Presumably the men were, for ordinary explosives, outside the zone which has been referred to in this paper as zone C. Although the men had some technical knowledge they did not know the nature of the various explosive fillings which were to be destroyed at any given time, and it was interesting to study the differing effects of these upon them. Single simple fillings such as T.N.T., picric acid, and tetryl, rarely caused any of the men discomfort under the conditions just described, yet when mixtures such as ammonal (British), amatol (British and German), "donarit" and "gluckauf" (German) were detonated many of the men complained of unpleasant physical sensations—e.g., "catching in the throat," "peculiar feelings in the chest," "creepy feelings down the spine," or "weakness of the legs"; others were seen to shake violently, and the rate of their pulse and respirations increased. When separated or layered fillings of different explosives, having velocities of detonation which differed considerably from one another, were detonated the effects were decidedly greater, and several of the men under these conditions passed into a state of stupor, which was followed in some instances by violent shakings and twitchings, and in one case by vomiting. Men thus affected were not fit for duty for the next two or
three days. Moreover it was noteworthy that any man who had once manifested the more intense symptoms was subsequently less resistant. And although he was fully aware that no real danger attended the demolitions, he seemed to be in a state of mental anxiety, and afterwards was more easily and readily affected by the detonations than he had been previously. To use the expression of his colleagues "he seemed to have got a sort of respect for H.E."

Now one word upon the nature of these mixed and layered fillings. Examination of certain German shells shows that layered fillings were deliberately employed although in manufacture they involve more trouble and labour. For example, the 15 cm. howitzer shell has the top half of the filling composed of a mixture of 60 parts trinitroanisol and 40 parts ammonium nitrate. This mixture yields a detonating wave with a velocity of 4,850 metres per second. The lower half of the shell is filled with dinitrobenzene, which yields a detonating wave with a velocity of 6,000 metres per second. The upper and lower charges are often separated by a diaphragm of wax or wood. This type of filling is also found in the 25 cm. "Minenwerfer" short shell. As the latter has not to take the grooving of a gun tube the container need not be strong. In practice it was made of a sort of Britannia-metal which pulverized on the detonation of its contents. The shell, therefore, cannot have been intended to act by "fragmentation." German documents reveal the fact that this type of shell was especially intended for use immediately prior to an attack, and in sectors where the opposing troops were well entrenched and possessed good dug-outs. Hence it is highly probable that the Germans were aware of the peculiar demoralizing effects of these mixed and layered fillings. Reference should also be made to the German "Disk-granaten," known to British troops as the "oyster-grenade," and to the "Stiel-handgranaten," known as the "potato-masher"; neither of these kill by fragmentation, but both are intended for killing by detonation and for demoralizing.

It would be interesting to know if the Germans' use of these fillings was based upon experimental findings, and if so what was the precise nature of their experiments.

Most of those who have been through a severe bombardment, either from shells similar to the above mentioned, or from aërial bombs, can bear testimony to the peculiar demoralizing effect it had upon them, and how this state of demoralization was succeeded, owing to summation of effect, as the bombardment continued, by a sort of fixed "glassy feeling," even when the shells did not explode
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particularly close to them. In order to eliminate as far as possible the psychic factor, which under such circumstances plays its part, it is instructive to study cases in which the effective detonation fell like a "bolt from the blue" upon individuals who were not in a state of tense expectation or fatigue at the moment of the incident. At the same time one cannot ignore the importance of the predisposing factors which are highly complex. There is, however, as regards the type of neurosis developed, nothing distinctive about them; so far as our observations go they all seem to operate in a general way by lowering the resistance of the individual to all forms of shock. Of all predisposing factors—fatigue, both physical and mental, is undoubtedly the most important. A type of history which has attracted our attention by its frequency is that of the soldier who had been moved rapidly from one part of the front to another, and on arrival at the new sector was sent direct into the firing line. He generally says that the trenches were in bad condition and that while exhausted on his first evening here his company were subjected to heavy bombardment. Under conditions of this sort the soldier seems particularly liable to break down either immediately or during the course of the day following. A rest of comparatively short duration exercises a truly wonderful influence in restoring the resistive powers.

In our experience the following points are helpful in determining the nature of the primary causal factor in cases where it is doubtful:—

First, to deal with cases in which "direct concussion" is the causal agent. The initial symptom is unconsciousness, often of several hours' duration, and possibly persisting for days. Consciousness when recovered does not remain clear, but the patient passes through a phase of variable length during which he loses and regains it; the so-called "dipping of consciousness." Aphasia and sphincter troubles are common at this stage, which is frequently associated with an active delirium in which the patient re-enacts episodes of his military service. The general condition is one of extreme exhaustion; all mental processes are sluggish, and there is great difficulty of concentration. Amnesia, both for the immediate and remote past is a common occurrence. The voice is often altered, becoming slow, monotonous, and higher pitched than is natural to the patient. In severe cases these symptoms tend to persist for many months, and relapses are easily induced by any fatigue: we have noticed them after long railway journeys. Clinically the importance of recognizing these symptoms is that the prognosis in cases of genuine "direct concussions" is much graver than in those giving a
spurious history of concussion after being thrown down or partially buried. Also psycho-therapeutic treatment is of no avail, except in so far as fixation of symptoms may have supervened in the later stages. In such cases experience shows that prolonged rest and freedom from strain of all sorts are essential.

The discrimination of cases of "indirect concussion" requires more careful study. Unconsciousness or stupor of relatively short duration is the rule, but it is often difficult to obtain an accurate history on this point as there is a tendency on the part of the patient to exaggerate, and corroborative evidence is rarely forthcoming. The phenomenon of "dipping of consciousness" is not observed, and active delirium of the occupational type is rare. Amnesia, though frequently present, is much less in extent, is only retrograde, and is more readily recovered from than in cases of direct concussion. There is a greater tendency to the superimposition of functional troubles such as paralyses and algesia. These may develop immediately, but more commonly they come on later as the symptoms of commotional origin are recovering. If they are removed by appropriate treatment, the patient after a few weeks' complete rest, generally "feels as well as ever." Easy fatigability and diminution of power to concentrate are, however, apt to be more persistent symptoms, and the patient in spite of his statement that "he feels quite all right" should be given a more prolonged period of freedom from strain. The apparently rapid recovery of objective symptoms in cases of indirect concussion would seem in part responsible for the tendency to regard them as of purely psychogenic origin.

Cases in which the neurosis has arisen as the result of exposure to detonation in what we have termed the C zone are naturally more difficult to differentiate from those of purely psychogenic origin. The description, already given, of men so exposed in the ammunition proof and demolition ground affords a better picture of the onset of the neuroses than can be got from the more complicated conditions obtaining in trench warfare. One must remember that in the latter the bombardment is more incessant; the soldier is in a state of expectancy, and possibly also of physical fatigue, all of which factors sensitize or predispose him to the effects of detonation. In a rather disparaging way any shell exploding outside the A zone is commonly referred to as merely the "last straw," its influence thus being attributed to purely emotional factors. It is, however, a "last straw" which cannot be lightly disregarded, and one is justified in maintaining that a sufficient number of such "straws" will cause the downfall even of the most
robust. And one should bear in mind the fact that the stability of an explosive is under such conditions diminished, and that it may eventually detonate. It is not difficult to conceive that physical processes which thus give rise to alterations in the direction of instability in a relatively simple chemical compound such as an explosive may in a similar way affect the far more delicate colloidal solutions contained in the central nervous system either directly or indirectly.

With cases of purely psychogenic origin, many of whom have never reached the danger zone at all, it is not our object to deal.

An interesting question which does arise at this point is as to whether the two aetiological factors, emotion and commotion, may not operate together and thus confront the observer with a third group of patients in whom the origin of the neurosis is mixed. Obviously, if once the possibility of a physical origin be conceded, this question must be answered in the affirmative. We suggest, further, that this is the most common occurrence, and that whichever be the primary cause of the instability, the patient, once sensitized remains for a long time, perhaps always, hypersensitive to both commotional and emotional stimulation. Under war conditions a vicious circle is thus established.

From a large number of instances of "real shell shock" as distinguished from those of "neurasthenia" the following is selected as worthy of report here, not because one example can be held as establishing any general principle but because it may be taken as a representative type of the group of patients in whom the primary causative factor is "commotional," but in whom this physical origin has become overlain by emotional elements during the progress of the case, as so commonly occurs.

A corporal, aged 22, went to France with the original Expeditionary Force, and served through the Mons retreat, the battle of the Marne and at Ypres with great coolness and courage. He liked military life and was particularly well adapted to fighting. In May, 1915, he was one day sitting with a companion in a trench preparing some food when a large high explosive shell detonated on the parapet almost immediately above him. His comrade was killed, but this he did not know until long afterwards. For a few moments he lost consciousness, but, from the account of others, this period of unconsciousness must have been brief. He then went over the top, exposed himself with apparent recklessness, and rushed about in a state of oneiric delirium shouting various orders to his men. After this incident he was sent down to the base for a rest, but in a month he was returned to his unit to all outward
appearances as fit as ever before. Inwardly, however, he noticed a great change in himself, and found to his great disgust that he no longer could remain calm when under shell-fire. His anger and shame at the idea of suffering from "shell shock," which he considered merely a polite phrase for "wind up," were so great that he used much effort and managed to control himself for the next two years during which he remained in France. From July, 1917, to March, 1918, he was in England training as a cadet, and during that time he ceased to think of all his unpleasant experiences, and when he returned to France he went with the same feeling of confidence as he did in 1914. But, to his intense disgust, he discovered that close proximity to shell-fire threw him into an uncontrollable state of excitement. He began to suffer from severe discomforts in the head, and felt so sleepy that he could hardly keep awake. It now became necessary for him to avoid, by every possible trick, the occasion of a public breakdown; this he managed to achieve. In October, 1918, he returned home on ordinary leave: on his second day in England—i.e., as soon as he relaxed control—he fell into a state of stupor and was admitted to hospital. Here, over a period of at least two months, states of stupor alternated with paroxysms of excitement and weeping. At the present time (January, 1919), he fatigues easily, is liable to headaches, has difficulty in concentrating his thoughts, and becomes confused in the neighbourhood of traffic.

A history of this type is by no means rare, but is met with fair frequency in the special neurological military hospitals. It is desirable from every point of view that cases of this type should be separated from those in which the underlying causal factor is purely psychogenic, no matter how the latter be further subdivided. One cannot remain content with any classification which is based upon an arbitrary grouping of superficially similar characteristics. Every effort should be made to discriminate between the several types upon the basis of their pathogenic origin, for both treatment and prognosis are largely determined by this. Had our patient been seen when at the base after the first detonation we should have been justified, surely, in describing him as an instance of "shock" of purely commotional origin, and in treating him accordingly. If, on the other hand, we had seen him for the first time in October, 1918, and had known nothing of his previous history, we might have regarded his manifestations as of purely psychogenic origin. Experience shows, however, that psycho-therapeutic treatment is of no use while acute symptoms of commotional origin are present, and even when, in mixed cases, these have subsided, the progress of the patient is as a rule distinctly slower than in purely psychogenic cases.

Although one not infrequently meets with cases in which, apparently, a single causal factor has been operative, we maintain that a careful and
unbiased study of histories in the special neurological military hospitals, leads to the conclusion that the combined action of the two aetiological factors is the more common event.

Conclusions.

The neuroses of war may be brought about:—

(1) By the action of "purely emotional shock," which at the present time is well recognized, but whose importance tends to be over-estimated.

(2) By the action of "purely physical shock," a study of the mechanism of which is the main object of this contribution to the subject.

(3) By a combination of the two above-mentioned aetiological factors; this is the most common occurrence, and, after the psycho-neurotic symptoms have been cleared up, the residual actual neurosis, which has formed a foundation for the whole disorder, is often exposed.

The object of this paper will be amply served if it help towards correcting the present tendency to regard the neuroses of war as of exclusively emotional origin, and towards gaining a more general recognition for the underlying physical basis demonstrable in a considerable proportion of them. At the same time it seems to us to open up the whole question of the interdependence of the physical and psychic basis of hysteria and its allied conditions.
Section of Neurology.

President—Dr. Henry Head, F.R.S.

Cases with Signs of Neurological Interest.

By Henry Head, M.D., F.R.S. (President).

The President (Dr. Head, F.R.S.) showed three officers who presented signs of neurological interest:

(1) A case of cervical rib, where there was some weakness and wasting in the right hand. He had been wounded on the left side of the head, and the condition in the hand was attributed, in a military hospital, to the injury to the brain, and not to the effect of the accessory rib.

(2) A case of injury to the parietal region, which had produced loss of sensation in the left hand. This showed all the characters described in Brain, 1918, xli, p. 59 (on "Sensation and the Cerebral Cortex").

(3) An officer from whom a pistol bullet had been successfully removed by Mr. Wilfred Trotter. It lay at the base of the brain, and had injured the anterior clinoid process. Mr. Trotter removed the bullet by the frontal route, and the osteoplastic flap healed perfectly. The patient had entirely lost his headache and other symptoms.

At a meeting of the Section, held May 8, 1919.
Case of Cervical Concussion.

By E. Miller, Lieutenant, R.A.M.C.

(Shown by Dr. J. Godwin Greenfield.)

Private T., admitted to hospital on March 3, 1919. Nine days previously he had been thrown from a motor wagon into a ditch. He was unconscious for about half an hour, and on recovering consciousness had complete paralysis of both arms and legs, with incontinence of urine and faeces. The power in the arms returned to some extent four days later. Tingling in the legs was present from the very outset, but no sensory loss was observed. On February 18 he could move his left arm and leg very slightly. On February 25 he could move his right leg and right arm, but the arm was still very weak. The control of the sphincters was recovered during the second week.

On admission to Tooting Military Hospital on March 3 he showed the following condition: He had voluntary power for all movements of the upper and lower limbs, stronger on the left side than on the right. No sensory loss was observed; his tendon reflexes were exaggerated more on the right side than on the left, with ankle clonus on the right side. The superficial reflexes were all absent, the plantar response being of the extensor type on both feet.

The power of the limbs improved daily, and on March 12 he could support himself in a walking chair. On the next day he was put into a swimming bath of warm water (temperature 90° to 95° F., depth 3 ft.). In this he could walk unsupported the whole length, but still showed some rigidity of the right arm and leg. On March 24 after daily exercises in the bath he could walk about the ward unsupported and without sticks.

Condition on May 8: He walks well with a slight drag of the right leg, and the right hand is rather stiff. The skiagram shows no fracture nor dislocation of cervical vertebrae, but atrophic changes in the body of the sixth cervical vertebra.

The case is of interest as showing the rapid increase of functional power in cases of spastic paraplegia which may be obtained by the use of a warm swimming bath.
Case of Pachymeningitis Cervicalis of Syphilitic Origin.

By E. Miller, Lieutenant, R.A.M.C.

(Shown by Dr. J. Godwin Greenfield.)

Private O., aged 38. History of venereal disease eighteen years previously. For six months he has had wasting of the muscles of the left arm, especially in the hand, and more recently has noticed some drawing up of the little finger in the right hand, so that he could not straighten it normally. For about four months he has had pain in the back of the neck.

Present neurological state: Except for slight wasting of the right side of the tongue, there is no affection of the cranial nerves. Motor power in the trunk and legs is good. In the right arm it is normal, except for difficulty in straightening the little finger. In the left arm there is weakness of extension of the elbow, with wasting of the left triceps, flattening of the ulnar surface of the left forearm, and wasting of all the intrinsic muscles of the hand, with clawing of the ring and little finger. There is some diminution of sensibility over the ulnar area of the left hand, otherwise sensation is everywhere normal. The deep reflexes are absent in the arms but normal in the legs, with brisk abdominal responses and flexor plantar reflexes.

The cerebro-spinal fluid gave a normal number of cells per cubic millimetre, but had a considerably raised albumin content (0.08 per cent.). The Wassermann reaction was strongly positive both in the cerebro-spinal fluid and in the blood.

Under anti-syphilitic treatment with novarsenobillon, mercury and potassium iodide there was general improvement in the condition, diminution in the pain in the neck, and an improvement in power in the left arm.
Section of Neurology.

President—Dr. Henry Head, F.R.S.

Encephalitis Lethargica.¹

By E. Farquhar Buzzard, M.D.

Very important observations have recently been published by Sir John Rose Bradford, Captain E. F. Bashford and Captain J. A. Wilson, on the bacteriology of several diseases which, according to these writers, are caused by filter-passing organisms. If the results obtained by their research are confirmed our knowledge of the pathogenesis of lethargic encephalitis has been materially advanced and incidentally we shall be in a position to regard this disease as belonging to a group, including influenza, rabies, toxic polyneuritis and nephritis, the members of which have been showing a regrettable degree of activity during the last year or two.

But this advance in our knowledge of the laboratory aspects of lethargic encephalitis must not be allowed to dull our interest in its clinical features and my object this evening is to call attention to a few points which have obtruded themselves on my notice during the epidemic which has prevailed with more or less severity from the spring of last year until the present time.

In a previous paper² I confined myself to the discussion of a group of cases in which the encephalitis had its chief incidence on the cerebral hemispheres, in which haemorrhage was a conspicuous feature and in which a fatal issue was the general rule. In these patients there was a

¹ At a meeting of the Section, held July 9, 1919.
² Lancet, 1918, ii, p. 835.
remarkable absence of severe constitutional disturbance and of high fever, both of which were prominent features in the case of a patient whom I saw with Dr. Wadd at Richmond.

Case I.—A married lady, aged 27, in the last few weeks of her second pregnancy, complained on January 13, 1919, of diplopia and a strange feeling in her head. During the next two days she went about as usual but appeared at times to be confused, drowsy, and unable to remember the object of her shopping errands. On January 16 she had an easy and uncomplicated confinement. On January 17 her temperature gradually rose to 104° F., headache was severe and there was increasing drowsiness and disorientation. On examination she presented partial bilateral ptosis, weakness of the left inferior rectus muscle, an expressionless face, slight tremor of the tongue and hands, with normal tendon-jerks and doubtful plantar responses. The next day she was more drowsy and asleep all the time unless she was roused. The ptosis was more marked and the temperature remained between 103° and 104° F. The optic disks were slightly congested. A papular rash had appeared over a large part of the surface particularly over the forearm, buttocks and anterior aspects of the legs. A lumbar puncture obtained cerebro-spinal fluid in which no abnormal cells could be detected. On January 20 the patient was much worse. She lay in a muttering delirium, cyanosed, picking at the bed-clothes, with a pulse of 140, mucus râles in the chest, rapid respiration and an intense general morbilliform rash. There was now complete ptosis and palsy of all ocular movements. Swallowing had become impossible and the patient resented light thrown into her eyes. Death seemed imminent, but during the next forty-eight hours there was a remarkable rally. The temperature fell, the rash faded, pulse and respiration improved, but the ocular palsies and the dysphagia remained. At this time involuntary local spasmodic movements were very noticeable in the limbs. On January 23 the temperature was below normal and the patient had spoken a few words quite rationally and asked after her baby. Swallowing was again possible but the eye movements were no better. The same night, however, the temperature shot up to 104° F., the rash reappeared in a still more intense form, and the patient went rapidly downhill, dying on January 25.

This case was remarkable for the pyrexia, for the rash which challenged comparison with a very severe measles eruption, and for the occurrence of a fatal relapse, falsifying the apparently justifiable hopes of recovery which were entertained two days before death.

My next story concerns a mother and her son whom I saw at Weybridge with Dr. Barton and Dr. Rob.

Case II.—The son, aged 19, came home from a theatre on March 28, 1919, with a headache and feeling shivery. The next day the temperature rose and on March 30 a rash appeared, giving rise to the suspicion of scarlet fever.
The following day headache was severe and the patient had a sudden generalized convulsive attack which left him in a semicomatose condition. When I saw him on the following day he lay half unconscious with closed eyes and a marked resentment to all examination or disturbance. The right arm was obviously paralysed, the abdominal reflexes were sluggish, and the knee-jerks difficult to obtain. A lumbar puncture released fluid at increased pressure and this was carefully examined by Captain Greenfield, who reported that the only abnormality was an increase in the glucose content. The patient recovered consciousness a few hours later and was found to have difficulty in speaking and to have weakness of the right side of the face and of the right arm and hand. From that time recovery was progressive and uninterrupted, but at the end of a fortnight the weakness of the face and grasp was still perceptible.

Case III.—On April 14 the mother, aged 54, who had been much with her son, began to feel as if her tongue and throat were swollen and complained of headache. The next morning headache was severe, the temperature was 101° F., and the patient displayed an intense morbilliform rash all over the chest, abdomen and arm. Photophobia and vomiting added to her discomfort. The cerebro-spinal fluid was at high pressure and Captain Greenfield reported that it resembled the specimen obtained from the patient’s son except for a slight increase in the percentage of chlorides. The patient suffered from the headache and vomiting for two days and during the same period exhibited weakness of the left side of the face and of the right arm, but her recovery was more rapid than that of her son.

I believe that the occurrence of two cases of this disease in the same household is very rare, and this is the only instance which has come under my own observation.

During the last eighteen months I have seen a number of cases of encephalitis which form a marked contrast to the severe type I have just described to you, and many of which have never been considered or have never considered themselves as seriously ill. The group is characterized by the insidious onset of slight lethargy, an inclination to fall asleep whenever they sit down to rest or read, or a tendency to forget the small things of everyday life and sometimes by an alteration in temperament. The patient may complain of diplopia which lasts for a few days to a few weeks, or of a general stiffness or slowness in his movements. He may complain that although he is always dropping off to sleep in the daytime his night’s sleep is disturbed by restlessness, by mild cramps, or pains in his limbs, so that he wakes every hour or two. Headache may never be complained of and fever may be conspicuous by its absence.
Case IV.—A recently married lady, aged 27, was brought to me by Dr. Bevan because her husband said she was always dropping off to sleep and she complained of double vision. She had a slight feeling of nausea and said her head felt “buzzy.” She told me that she had gone to the butcher’s and left her coupon as well as her meat. On examination the left palpebral fissure was wider than the right and diplopia was obtained on directing the patient’s eyes to the left. She had no other physical signs and she made a complete recovery in the course of a fortnight or three weeks, but we took the precaution of sending her to bed for a rest. Her symptoms were almost identical with those of Case I in the first few days of her illness and it was impossible to say whether the more serious and fatal sequelæ might not supervene.

Case V.—In February of this year Dr. Redfern sent to me a man, aged 59, who told me that in December last, as he was walking to catch his morning train to the City, he noticed “everything looking double” and that in the course of a week he became stiff all over, “making me feel like an automatic figure in my movements.” He had no headache but felt very sleepy and could not keep awake when reading. As he walked into my consulting room I made a mental diagnosis of paralysis agitans judging by his mask-like face, the gait, and the way in which he held his arms and hands. He complained of slight twitches at times in his hands and particularly of the slowness with which he dressed and undressed. The diplopia had cleared up and he was improving in every way. When I saw him again in April he was much better and could then write freely and dress himself in normal time.

Case VI.—On February 11 of this year I saw with Dr. Glover a lady, aged 53, who had complained for a fortnight of slight headache and sleepiness. On January 31 she developed diplopia, increased drowsiness and mental disorientation with a slight degree of ptosis which had disappeared when I saw her. She was restless and unable to concentrate her attention. She presented slight weakness of the right side of the face and the right hand tended to assume the paralysis agitans position with occasional twitching of the fingers. There were no other physical signs, but the temperature was apt to reach 100°F. on some days. This lady’s progress towards recovery has been very slow, especially in regard to her mental condition. She is still depressed, easily exhausted, and unable to keep her interest or attention fixed for any length of time.

Case VII.—In April of this year I saw with Dr. Mennell a lady aged 46 who, after feeling indisposed for two or three weeks, was seized on March 30 with some headache and vomiting and pyrexia which lasted for several days. Photophobia and bouts of drowsiness, with a milder degree of headache, had persisted after the fall of the temperature. On examination she presented a slight degree of ptosis on the right side, absence of all abdominal reflexes and sluggish tendon-jerks. She made a good but slow recovery.
Case VIII.—A gentleman, aged 63, seen with Sir Robert Fox-Symons in March of this year, complained that a week before our consultation he was seized with diplopia while playing billiards. This was followed by headache and general restlessness, a little cramp in the legs and paresthesia in both hands. He presented partial ptosis, more marked on the right side, and weakness of all ocular muscles, especially in regard to the internal and vertical movements. The right pupil reacted sluggishly to light. The knee- and ankle-jerks were difficult to obtain and the right plantar reflex was indefinite in type. In spite of the comparative severity of his symptoms this patient made a rapid recovery in the course of a fortnight during which time he was kept in bed.

Case IX.—On June 6, 1919, I was consulted by a lady, aged 42, who told me that two months previously she had been suddenly seized with diplopia, drowsiness, general loss of power, loss of memory and loss of control over her emotions. She stated that she felt as if she was in a dream, and that, whereas she had always been lively and bright, she was now quite altered in tempera- ment, and only wished to go to sleep. She had not suffered from headache or vomiting, and as far as she knew had no rise of temperature. On examination her pupils were found to be sluggish, there was left-sided ptosis and paresis of the left internal rectus muscle. Her face was expressionless and her movements generally feeble, but the reflexes were normal. It may be noted that she had never been in bed, and that she had been medically advised to go about as much as possible, and to seek lively company.

These cases of mild encephalitis could be multiplied, but I have described a sufficient number to indicate their general symptomatology and physical signs and to support the view I hold that they must have been very numerous during the last eighteen months. You will have noticed that the physical signs have been generally trivial, and would easily have escaped noticed with any but careful examination. The histories obtained from the patients and the account of their symptoms have been important factors in the diagnosis which has been confirmed by the general tendency towards recovery in the course of a few weeks or a few months. One can hardly doubt that early diagnosis is a matter of great importance in these cases, and that patients who are suffering from an inflammation of the brain should be prevented from carrying on their normal activities even though they are not troubled with headache, vomiting or pyrexia.

Let me now pass on to the consideration of another very interesting and serious aspect of the more severe cases of encephalitis in which the patients recover from the acute stage of the disease, but are overtaken by more or less disabling sequelæ at varying intervals of time. I refer to
the late development of involuntary movements affecting the limbs, jaw or tongue. Some of these patients have passed through an acute illness associated with severe headache, vomiting, fever and other cerebral symptoms which have given rise to the diagnosis of influenza, meningitis, &c. Some of them have displayed involuntary movements during the acute disease, the abnormal activities disappearing in the course of a long convalescence. It would be difficult to describe accurately the character of these movements; some have been irregular and choreiform, others more rhythmical and limited in their distribution. One of my patients had movements, confined to the left foot, which she aptly nicknamed her "joggles." Speaking generally, the movements are in abeyance while the parts concerned are in voluntary use, and are most obvious when the patient is resting. On the other hand, all these movements cease during sleep, and gradually subside in the process of going to sleep. The movements do not interfere with voluntary action and are only a source of complaint in that they interfere with the patient's peacefulness while at rest.

Case X.—W. H., aged 28, a fireman on the Great Eastern Railway. Family and previous history good in all respects. He was taken ill on June 4, 1918, and went to bed on June 6 with headache, general malaise and sleeplessness. The right side of the face became paralysed, there was difficulty in mastication and fluids regurgitated through his nose. The latter symptom only lasted about ten days, but by June 27 he displayed tremor of his right hand in shaving and writing, and his legs appeared to be weak. His reflexes at that time were normal. During the next six months he made little or no progress towards recovery, and by December his legs were showing involuntary movements, which spread to the left arm early in 1919. When admitted into the National Hospital in April of this year he displayed constant movements of both shoulders, more marked on the left side. The movement of the left arm may be best described as "trouser hitching," and included contractions of the deltoid, latissimus dorsi, trapezius and biceps muscles. There were also forced movements of the head, chiefly carried out by the deep muscles of the neck. In the legs contraction of several groups of muscles was obvious while he lay in bed, but the spasms were suspended during walking and other voluntary movements, and during sleep. The pupils were dilated and unequal, the left being larger and less active to light than the right. The right side of the face was weak in voluntary action, but at rest showed an increase of tone and clonic spasms. The strength of the limbs appeared to be about normal, but when the arms were outstretched there appeared coarse movements at the shoulders, which were increased by fatigue. The arm movements showed some dysmetria and dysdiadochokinesia. The reflexes were normal in all respects. During the last three months the patient has shown marked
improvement. His blood and cerebro-spinal fluid have been examined with negative results.

Case XI.—J. W., aged 44, a packer. Seen for the first time on June 1, 1918, at St. Thomas's Hospital, when he told me that six weeks previously he had noticed diplopia, mental confusion, and tremor of his right arm and right leg. He presented a typical paralysis agitans appearance in posture and facies. The reflexes were normal. A month later he complained of increased tremor in the right hand and dragging of the right foot, and constant twitching of the muscles of the right thigh. In July and August he was improving. In November he was walking more freely, but I noted coarse involuntary movements of the right hand. In January, 1919, he displayed continuous involuntary movements of the jaw and tongue, which were suspended during talking and eating. During the last six months these have gradually decreased, and are now hardly noticeable. He expresses himself as very much better in all respects, but his simulation of Parkinson's disease is still obvious.

Case XII.—A. M. P., a married woman, aged 35. Sent to me at the National Hospital by Dr. Davidson, who kindly supplied the following note, dated May 20, 1919: “Illness started in February, 1919 (when she was seven months pregnant), with acute pain down the left arm. Two or three days later I was sent for and found her sitting in a chair, twitching all over, muttering to herself, and only semiconscious. The movements were choreic, and I thought at first it was a bad attack of chorea gravidarum. I got her into the local infirmary, where she lay unconscious for several days and miscarried. She was discharged from the infirmary about a fortnight ago, but although her mind is clear, she is scarcely able to walk, and is on the twitch all day long.” On examination I found constant involuntary movement of internal rotation of the left shoulder, with flexion of the left elbow. The trapezius and pectoral muscles also took part in this movement. The right arm was similarly affected, but to a less degree. Tendon-jerks exaggerated. Left plantar response was extensor, right flexor. No sensory disturbance. Slight diplopia has been present, but has now cleared up.

Case XIII.—L. F., a single woman, aged 28, suffered from chorea as a child. Seen on April 4, 1919, at the National Hospital, when she gave the following history: A year ago went to the doctor with a sore throat and headache, and felt faint on her return home, when she lost power in her left arm and leg. This gradually recovered, but left her with involuntary movements in her left leg, and some tremor and unsteadiness in her left arm and hand. On examination the right pupil was larger than the left. The left abdominal reflex was absent, the left knee-jerk more brisk than the right, and the left plantar reflex indefinitely extensor in type. The left foot was in a state of constant movement while sitting, but quite steady when she was walking or standing. She has an old mitral lesion, and it is possible that her condition is due to an embolic lesion.
Some of these patients have been before you this evening, and I have little doubt that you will agree with me in regarding their symptoms as the sequelæ of encephalitis. You will also sympathize with me in the difficulty which I find in correlating their clinical phenomena with what is known of the morbid anatomy of the disease. The involuntary movements which have presented themselves during the acute stage, and which have shown a general tendency to disappear in the course of time, may be attributed to the effects of inflammation on some of the efferent systems originating in the mid-brain or brain stem. Whether the inflammation excites these movements or whether it allows their occurrence by interfering with their normal control is a question I find too difficult to answer. But still more interesting and obscure is the appearance of fresh movements in different parts of the body weeks or months after all inflammation must have subsided.

In studying the morbid anatomical features of this disease in several fatal cases which I have examined, I have been struck by the fact that the brunt of the inflammation falls upon the vessels, with the result that arterial and capillary hæmorrhages and venous thromboses are of very frequent occurrence. On the other hand, if the brain of a patient who has died months after the acute stage is examined, our attention is at once directed to the condition of the vessels and their walls. I can show you some photomicrographs prepared by Captain Greenfield from sections taken from the brain of one of my patients, in which you see that the vessels are filled with organized clot, and the vessel walls have become so thickened and calcareous that considerable difficulty has been met with in cutting sections. For this reason I am inclined to attribute the late clinical complications in lethargic encephalitis to secondary vascular disturbances, although I should feel baffled, to put it mildly, if I were asked to explain the precise mode of their origin.

I should like to say one word with regard to prognosis. Many of the palsies arising from this disease are undoubtedly permanent, and some of the patients who have survived severe attacks are hopelessly and pitiably disabled. On the other hand my experience leads me to think that the involuntary movements, although they may last for months, show a disposition to clear up, and I am hopeful that they may be regarded as temporary, though distressing, complications.

The question of diagnosis is one which might be discussed at great length, and I could cite some cases from which I have learned useful
lessons arising from the mistakes I have made. Let me mention in this connexion a complication of the acute stage which I have met with in two patients—namely, haemorrhage into the vitreous of one eye, producing almost complete blindness.

Finally, I should like to acknowledge my indebtedness to Dr. Blandy, the Resident Medical Officer at the National Hospital, for her careful and accurate reports on the cases which have been under my care in that institution.