**Reports and papers on epidemic poliomyelitis.**

FOR OFFICIAL USE.

# REPORTS

TO THE

# LOCAL GOVERNMENT BOARD

ON

# PUBLIC HEALTH AND MEDICAL SUBJECTS.

(NEW SERIES No. 61.)

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1. Dr. R. J. Reece's Report on the prevalence of Acute Poliomyelitis in Devonshire and Cornwall in 1911.
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# REPORTS AND PAPERS ON EPIDEMIC POLIOMYELITIS.

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## Introduction by the Medical Officer.

To the Right Honourable John Burns, M.P.,
President of the Local Government Board.

Sir,—I have the honour to submit to you reports and papers on the subject of Poliomyelitis.

Since the epidemic of this disease in Sweden in 1905 and 1906, it has been ascertained to occur in epidemic form in various parts of the world, notably in Northern Europe, and also in the United States of America, where outbreaks of a greater or less intensity have been reported from many different States since 1907.

In this country the occasional appearance of small groups of associated cases has been recognised for some years past, and it has frequently been suspected that these cases have formed part of a much more considerable and more widely-spread local prevalence. This belief has been strengthened by the fact that the disease manifests itself in very different forms, so that its true nature may easily pass unrecognised.

In these circumstances it has lately become important to watch for the occurrence of epidemic poliomyelitis in England, in order to take a suitable opportunity to make a comprehensive study of its clinical and epidemiological character. Two such investigations have now been made for the Board; one in Devon and Cornwall by Dr. Reece, the other in the Midlands and in Dorsetshire by Dr. Farrar.

Dr. Reece's inquiry (Report No. 1, below) developed from small beginnings. In August of this year it was considered advisable that he should visit Holsworthy, in Devon, to ascertain whether a few cases occurring there were cerebro-spinal ("spotted") fever, as was locally believed, and confer with the sanitary authorities concerned regarding any preventive measures which might be necessary. Soon after his arrival in the district, however, he was able to satisfy himself not only that the symptoms of the cases in question were those of poliomyelitis, but that similar cases were occurring here and there in different parts of Devon and Cornwall. By making systematic inquiries in both counties, and with the assistance of medical officers of health and local medical practitioners, Dr. Reece succeeded by the middle of September in tracing as many as 154 cases, most of which he personally visited. Of these, 34 were fatal. At the same time, as his report shows, he investigated minutely the various attendant conditions which seemed likely to bear upon the prevalence of the disease. Materials for pathological examination were obtained as far as possible, and

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Dr. Gordon, who examined them (Report No. 4, below), found that typical lesions of poliomyelitis were present in the spinal cord of fatal cases, and that the characteristics of the specimens of cerebro-spinal fluid obtained from numerous patients were consistent with a diagnosis of poliomyelitis and in no case corresponded with that of cerebro-spinal fever, which is associated with the presence of the diplococcus of Weichselbaum. Moreover, material from two of the cases, forwarded by Dr. Gordon to Professor Levaditi of the Pasteur Institute in Paris, was there found to produce typical acute poliomyelitis on inoculation into monkeys.

In 1910, Dr. R. Farrar had made inquiries for the Board, on somewhat similar lines, into an outbreak in which he collected 74 cases occurring in clusters in parts of Leicestershire and the counties adjoining it, and about a dozen cases in the city of Nottingham. In the majority of the latter, which Dr. Farrar classifies as a group apart, meningeal symptoms preponderated, and a "gram-negative" diplococcus was obtained from the cerebro-spinal fluid; they were in all probability cases of cerebro-spinal fever. All the rest Dr. Farrar attributes to poliomyelitis. Owing to his absence in China on official duties, his report (No. 2 below) was not completed until the present autumn, and he has consequently been able to review and strengthen this conclusion in the light of recent information. In this Midland outbreak, as also in a small epidemic of poliomyelitis in Cerne, Dorsetshire, which he investigated later in 1910, Dr. Farrar collected and recorded not only the clinical histories of each case, but also all the facts which could be obtained which bore on the infectivity of the disease.

For knowledge of the nature of the virus of poliomyelitis, its transmissibility by inoculation from man to monkeys, and from monkey to monkey, it is necessary to go to reports of work done in foreign laboratories, notably that of the Pasteur Institute. In view of the importance of the subject to readers to whom these publications may not be readily accessible, Dr. Gordon has contributed for the purpose of this volume (No. 3 below) a brief critical review of the experimental work on the virus of poliomyelitis, together with an account of the histology of the disease and observations bearing on immunity to it. With the same object, Dr. Macewen has prepared (No. 5, below) a review of the literature of the disease in regard to its clinical character and its epidemiology, drawn principally from the extensive observations which have been made in Sweden, the United States, and other countries in which conspicuous epidemics have occurred.

The whole of the facts ascertained by Dr. Reece and Dr. Farrar, in connection with the epidemics which they respectively investigated, deserve careful study. They will be found in many respects closely parallel to observations which have been made in course of some of the foreign epidemics which Dr. Macewen has noted. On the clinical side, stress should be laid upon the great diversity of the symptoms which were met with (dependent on the part of the nervous system attacked, and the intensity of the virus in the individual case) ; on the appearance of disease among adults as well as in children ; on its considerable fatality ; and on the permanent paralysis often left in those who recovered. On reviewing the circumstances associated with these outbreaks it must be admitted that, while accepting the facts that poliomyelitis has been proved transmissible by inoculation in the laboratory, and that in nature it can on occasion show a very high degree of infectivity, we still do not completely understand the epidemiology of the disease. In particular, explanation is required of the remarkable extent to which poliomyelitis has been found in remote country places and isolated dwellings, and of its intense ability to spread in one house or village while completely failing to infect in the majority of others under seemingly comparable circumstances of population and environment. In both of the epidemics reported on by Drs. Reece and Farrar "abortive" cases, in which little or no paralysis resulted, were detected in considerable numbers, and it may reasonably be conjectured that such cases play an important part in the dissemination of the virus. In this connection reference should be made to Dr. Reece's account of an outbreak of illness, associated with paralysis in only one or two instances, which affected nearly every house in the hamlet of Stoke Rivers during the present autumn. If the prevalent illness in this case was true poliomyelitis, it was at once extremely infectious and so slight in its manifestations as hardly to be recognizable.

It may be added that both reports contain instances where the circumstantial evidence suggested that the infection was conveyed by an apparently healthy "carrier." This has been suspected in some epidemics abroad. It is evident, however, that much further research work is needed, both in the laboratory and by exact and painstaking inquiry into the circumstances of local outbreaks, with a view to the more exact determination of means of communication of this disease and to the discovery of practically available methods of recognising the presence of infection in early cases of suspected disease and in contacts with known cases of the disease.

For convenience of reference, I have appended below a copy of a memorandum on poliomyelitis recently prepared by me and circulated by the Board to sanitary authorities. This memorandum is based principally on the facts contained in the reports now submitted, and draws attention to the precautionary measures against the disease which it is advisable for sanitary authorities to take. They include making poliomyelitis a notifiable disease under the Infectious Disease (Notification) Acts, and taking prompt measures to secure adequate isolation of cases on receipt of notifications. These were the principal measures which Dr. Reece urged, from the outset of his inquiries, on all the sanitary authorities concerned in Devon and Cornwall. It is to be regretted that in far too many cases those authorities possessed no isolation hospital accommodation, and that (to judge by the experience of the epidemic) they have frequently failed to realise the need for efficient methods of administration for the protection of their inhabitants and summer visitors against communicable diseases.

ARTHUR NEWSHOLME.

December, 1911.

# Copy of Memorandum on Acute Poliomyelitis.

*Circulated to Sanitary Authorities, December*, 1911.

This disease has long been known under the name "Infantile paralysis" as a form of paralysis of which sporadic cases occur, chiefly in children and less frequently in adults.

In recent years it has occurred in epidemic form in America, in Australia, and in some parts of Europe. A number of local outbreaks have also occurred in this country.

The virus of the disease has not been identified; but it passes through the finest filter; and the filtrate when inoculated into monkeys produces poliomyelitis in them.

The virus attacks the nervous system, causing inflammation of the grey matter of the spinal cord, especially of its anterior cornua. Microscopically the characteristic lesions found are hyper-aemia, a well-marked exudation of lymphocytes around the blood vessels and degeneration of the large motor cells in the anterior horns. Less often the whole cord, grey and white matter, may become involved and a transverse myelitis result. In some cases the brain may be primarily or secondarily affected. Hence the suggested name of Polio-encephalo-myelitis. The meninges also may become implicated in the morbid process.

The symptoms vary with the localization of the virus, and may be very diverse in character: in certain cases of the disease the difficulty of diagnosis is great.

There is no characteristic skin eruption. Usually an initial febrile attack occurs, the temperature seldom rising above 102°-103° Fah.: commonly there is malaise and drowsiness, accompanied by headache and occasionally by vomiting: there may be nasal and pharyngeal catarrh. The patient is fretful, and objects to being moved. Rigidity of the muscles of the neck and spine frequently occurs and the head may be retracted: there may be pain or tenderness over the spine. Kernig's sign is often present. Plantar, patellar or abdominal reflexes are commonly absent; the patellar reflex may, however, be exaggerated in the early stage. Paralysis, mainly of the flaccid type, supervenes shortly after the appearance of these primary symptoms. It affects one or more groups of muscles, especially of the limbs, but may involve any part of the muscular system.

This is the common form of the disease, but occasionally a child may go to bed apparently well and be found in the morning to be suffering from paralysis.

There is little difficulty in recognising the above forms of the disease. But apart from the above symptoms or in addition to them, there may be symptoms caused by implication of the medulla, brain, meninges or special nerves; and in such cases, especially when the meninges are implicated, confusion in the differential diagnosis between poliomyelitis and cerebro-spinal fever is apt to occur. The meningeal type may also be confused with tuberculous meningitis or septic meningitis. In such cases the previous medical history of the patient, and the

absence of other similar cases in the vicinity may aid a correct diagnosis.

The symptoms of Poliomyelitis, before the onset of paralysis, have frequently been attributed to Influenza. Cases at this stage have also been diagnosed as Sunstroke, Enteric Fever, Food Poisoning and a number of other diseases.

The occurrence of cases of Poliomyelitis in which no paralysis supervenes has been established, the symptoms of these mild or so-called abortive attacks being similar to those ushering in the graver forms. Such symptoms, if they occur in patients having association with paralytic cases, should arouse suspicion.

The fatality of Poliomyelitis has varied within wide limits in the epidemics of the last five years. Commonly 10 to 12 per cent. of the attacks prove fatal: but in such an estimate much depends on the proportion of mild or so-called abortive cases which have been recognised.

Over half the patients who survive an attack of Poliomyelitis are crippled for life.

The disease is most prevalent in the summer and least prevalent in the winter months. It appears to have no special relationship to social or sanitary conditions; and, so far, has been oftener recognised in sparsely populated districts than in large towns.

In monkeys experimentally inoculated with the disease the incubation period is stated to vary from 3 to 46 days, the general period being about 11 days. Clinical evidence points to an incubation period in the disease when naturally acquired by man of four or five days, though shorter and much longer incubation periods are recorded.

Experimental research on monkeys has shown that the virus is present in the naso-pharyngeal mucous membrane; and that it can be communicated experimentally through the respiratory and digestive tracts.

Careful observation of localised outbreaks furnishes circumstantial evidence of the transmission of the disease from person to person, including its transmission by a healthy "carrier."

The disease presents an erratic character as regards infectivity. In some instances it has developed after only slight or temporary contact with a patient suffering from Poliomyelitis, and multiple attacks in households are not uncommon. In other instances children have been known to sleep in the same bed with a patient and to escape infection.

The examination by competent bacteriologists of the spinal fluid obtained by lumbar puncture affords material aid in the differential diagnosis between Cerebro-Spinal Fever and Poliomyelitis. In the former the presence of the diplococcus intracellularis of Weichselbaum is characteristic of the disease, in the latter this organism is never present in the spinal fluid, but there is in it an excess of lymphocytes.

As the disease can be recognised with certainty by histological examination of the cord, this procedure should be adopted in fatal cases in which there is a suspicion of Poliomyelitis. Pieces of the cervical and lumbar enlargements—if not the whole cord—should be forwarded to a competent patholigist in 10 per cent. formalin.

Owing to possible confusion between Cerebro-Spinal Fever and Poliomyelitis, they should be considered together and both should be added to the schedule of diseases compulsorily notifiable under the Infectious Diseases (Notification) Act, as the first step towards administrative control. This is necessary in order to obtain early information of the presence of one or other of these diseases in a district.

The possibility of administrative control over Poliomyelitis is greatly increased by active co-operation between the medical officer of health and the medical practitioners in a district. In view of the difficulty in diagnosing certain forms of Poliomyelitis, medical practitioners should be invited to confer with the medical officer of health as to cases of anomalous illness; thus the medical officer of health may be enabled to assist the practitioner with information as to similar cases which may be in his possession.

The Sanitary Authority should be prepared to give facilities for the examination by competent bacteriologists of material derived from the sick.

It is important that precautionary measures should be taken in all suspected cases. As it has been shown experimentally that the virus is present in the nasal mucous membrane, an antiseptic solution should be applied by means of a spray to the throats and nasal passages, not only of the patients, but of all persons brought into contact with them. The virus can be killed experimentally by a one per cent. solution of peroxide of hydrogen, or by a solution of permanganate of potassium—1 : 500.

All discharges from the patient as well as all articles which may be soiled by such discharges should be immediately disinfected.

The sick should be isolated from the healthy, and if appropriate isolation and nursing cannot be obtained at home the patient should be treated in an isolation hospital. The sick room and its contents should be disinfected at the end of the illness.

No child should be allowed to attend school from an infected family.

ARTHUR NEWSHOLME,

Medical Officer.

Local Government Board,
November, 1911.

# No. 1.

# Dr. R. J. Reece's Report to the Local Government Board on the prevalence of Acute Poliomyelitis in the Counties of Devonshire and Cornwall in 1911.

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## Circumstances of the Inquiry.

Towards the end of July, 1911, a medical practitioner informed the Board that unusual cases of sickness in children were occurring in his neighbourhood at Holsworthy, North Devon; and he requested that a medical inspector of the Board should be sent to inquire as to the nature of the disease. At the same time Dr. W. G. Gray, Medical Officer of Health for the Holsworthy urban and rural districts, submitted a special report to the Board in which he stated that several cases considered to be cerebro-spinal meningitis had come under his notice, and that certain of these had had a fatal termination. He furnished detailed notes of some cases that had occurred in his private practice, and he sought the assistance of the Board's medical staff.

As it appeared that the cases were still occurring, and that more than one sanitary district was involved, the Board decided to have the outbreak investigated by a medical inspector.

On 27th July I received instructions to visit the affected districts, and I commenced my inquiry on the spot the following day. This inquiry lasted over a month, as it was found that the current illness was much wider spread than had been at first suspected, and it became necessary to collect information from the whole of Devonshire and Cornwall.

Opportunity of examining patients was freely afforded me by the medical practitioners, and after seeing several patients I formed the opinion that the symptoms exhibited by the cases pointed rather to epidemic poliomyelitis than to epidemic cerebro-spinal fever. This provisional diagnosis was subsequently supported by bacteriological and histological examination of material obtained from several of the patients in a plurality of districts; so that on review of the whole series of cases collected from the two counties (*see* Table of cases 1–154), I have no doubt that the prevalent disease was wholly poliomyelitis, not poliomyelitis occurring concurrently with cerebro-spinal fever. It was not possible for me to see every suspected case which occurred, and I was unable to obtain personal knowledge of certain fatal cases apparently connected with the outbreak under consideration in which death was

certified as due to cerebro-spinal fever, acute broncho-pneumonia, syncope, acute rickets or other cause. In some of these cases inquiry as to the cause of death was made by the local coroners. But it has seemed necessary to include some of these cases in my list, as the information available as to their symptoms, and the circumstances of their environment, made it, in my opinion, impossible to exclude poliomyelitis as the direct, or at least the primary, morbid condition.

It is very probable that certain mild cases, of the so-called "abortive" type, were not brought to my notice, particularly in the earlier months, some not even coming under medical observation. After publicity had been given in the newspaper press to this outbreak the public became aware of the importance of regarding even minor cases of illness with suspicion unless and until professional aid had been sought. In these circumstances many cases of the disease were detected which would not otherwise have been heard of. In some instances I was invited to see cases which were not of the nature of poliomyelitis, and which I have not included in my list.

In country districts, where the inhabitants are well known to each other and local events are freely commented on, it is much less likely that cases of illness would be overlooked than in large cities where people frequently do not know their next-door neighbour. Nevertheless I feel satisfied that many cases occurred, which owing to the absence of compulsory notification, or to inaccurate diagnosis, or to absence of medical attendance, never came to my notice.

After I left the district on 26th August I kept touch with the further local developments, being in this respect materially assisted by the Medical Officers of Health of the counties of Devonshire and Cornwall; and the list of cases I append, though I am of opinion it fails to include many cases, is as complete as I can make it for cases occurring on and before the 16th of September. It does not, however, include cases which have occurred in the county boroughs of Plymouth and Devonport, and the urban district of East Stonehouse, which adjoins these two boroughs; these three districts practically form one town.

I have laid stress on the difficulty of obtaining information, as it must be remembered that neither acute poliomyelitis nor cerebro-spinal fever are included among the notifiable diseases by the provisions of the Infectious Disease (Notification) Acts of 1889 and 1899. These diseases can, however, be made notifiable in districts by local councils with the consent of the Local Government Board. At the commencement of my inquiry acute poliomyelitis was not a notifiable disease in any district in Devonshire or Cornwall. Later the majority of the district councils in both counties applied for the sanction of the Board, which was at once granted, and by 16th September compulsory notification of both diseases had become general in both counties. In certain districts, however, compulsory notification was not adopted. This matter will be referred to later.

## Number and Distribution of Known Cases.

The number of known cases, together with the number of deaths which occurred in the several districts up to 16th September, is as follows :—*

| Devonshire— | | Cases. | Deaths |
|---|---|---|---|
| Ashburton ... ... | Urban ... | 1 | — |
| Barnstaple ... ... | Rural ... | 1 | 1 |
| Barnstaple ... ... | Urban ... | 1 | — |
| Bideford ... ... | Rural ... | 17 | 1 |
| Crediton ... ... | Rural ... | 1 | 1 |
| Crediton ... ... | Urban ... | 1 | — |
| Exmouth ... ... | Urban ... | 2 | — |
| Holsworthy ... ... | Rural ... | 42 | 10 |
| Holsworthy ... ... | Urban ... | 3 | — |
| Honiton ... ... | Rural ... | 1 | — |
| Newton Abbot ... | Rural ... | 4 | — |
| Newton Abbot .. | Urban ... | 4 | — |
| Northam ... ... | Urban ... | 2 | — |
| South Molton... ... | Rural ... | 8 | — |
| South Molton... ... | Urban ... | 7 | 4 |
| Tavistock ... ... | Rural ... | 3 | 1 |
| Tavistock ... ... | Urban ... | 1 | — |
| Teignmouth ... ... | Urban ... | 2 | 1 |
| Tiverton ... ... | Rural ... | 1 | — |
| Torquay ... ... | Urban ... | 2 | 2 |
| Torrington ... ... | Rural ... | 2 | — |
| Totnes... ... ... | Rural ... | 2 | — |
| | | 108 | 21 |

| Cornwall— | | Cases. | Deaths. |
|---|---|---|---|
| Bodmin ... ... | Rural ... | 1 | 1 |
| Camborne .. ... | Urban ... | 3 | 3 |
| East Kerrier ... ... | Rural .. | 7 | — |
| Falmouth ... ... | Urban ... | 1 | — |
| Launceston ... ... | Rural ... | 3 | 1 |
| Launceston ... ... | Urban ... | 1 | — |
| New Quay ... ... | Urban ... | 2 | 1 |
| Penryn ... ... | Urban ... | 7 | 1 |
| Redruth ... ... | Rural ... | 3 | — |
| St. Austell ... ... | Rural ... | 2 | — |
| Stratton ... ... | Rural ... | 5 | 1 |
| Stratton and Bude ... | Urban ... | 8 | 4 |
| Truro ... ... ... | Rural ... | 3 | 1 |
| | | 46 | 13 |
| Total for both counties | ... | 154 | 34 |

* Subject to the reservation on page 11. Additional cases have occurred in certain of these districts, and certain other districts have been invaded since 16th September.

From the map which accompanies this report it will be seen that the greater number of known cases occurred around Holsworthy, in Devon. There is, however, a batch of cases in the Penryn and East Kerrier districts in Cornwall. It is worthy of note that the first known case in Devon occurred on the 3rd day of May in the Holsworthy Urban District; the date of onset of the first known case in Cornwall, which occurred in the Penryn Urban District, was fixed as 27th May. The dates indicate that two foci of infection were present about the same time at widely separated points. Although careful and sustained inquiry was undertaken with regard to source of infection in these cases, no satisfactory explanation was obtained of their occurrence.

Reference to the map shows that there are large tracts in both counties in which no cases of the disease came under notice. The high moorland district of Devon and the hilly portion of Cornwall are but sparsely inhabited, and there is very little inter-communication between the residents owing to the long distances to be traversed and to the absence of railways. But it is also very probable that owing to the disease not being notifiable, such cases as may have occurred have not come under review.

Cornwall, the most western county of England, adjoins no county but Devon, which forms its north-eastern boundary. Their combined length measured in a straight line from N.E. to S.W. is 100 miles, and the broadest part, from N.W. to S.E., is some 60 miles across.

*Climate.*—Under ordinary conditions of weather in this country the climatic conditions are mild and humid, with westerly and south-westerly winds blowing in from the Atlantic. But in the summer of 1911 the temperature was exceptionally high, the thermometer frequently reaching to 90° Fahrenheit in the shade, and the climatic conditions were of an unusual character.*

*Topography.*—The country is one of steep hills, intersected by valleys, with occasional low lying land towards the sea. The villages are scattered over the uplands or distributed along the sea-coast at such points where water supplies have been available from time immemorial. Few, if any, towns or villages are of modern growth, although some have expanded rapidly of late years as fashionable seaside resorts.

*Industries.*—The districts mainly involved are purely agricultural. There are some local potteries. In the south a considerable quantity of china clay and some granite is exported, and in the south-west of Cornwall there is mining for tin and copper, but the mining districts were but little affected. In the summer months both counties are largely frequented by visitors, and in many of the villages and hamlets along the coast and in some of the remote country districts inland, every available house, including farm houses far removed from railways, caters for summer visitors.

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* See meteorological data on p. 35.

*Age Incidence of Ascertained Cases.*

Poliomyelitis is ordinarily known in medical practice in this country as a disease of some rarity, occurring sporadically rather than in epidemics, and essentially as a disease of young children, especially infants. Hence the common term "infantile paralysis," which is applied to it. It is noteworthy therefore—and the fact explains no little of the want of recognition of the disease and failure in correct diagnosis—that in the Devon and Cornwall outbreaks adults were attacked, sometimes severely or fatally; although, as will be evident from the following table, the incidence was found to decrease markedly with age. The age distribution of 153 cases, of which there are records,* are as follows:—

| *Ages in years, under* | *1* | *2* | *3* | *4* | *5* | *6* | *7* | *8* | *9* | *10* |
|---|---|---|---|---|---|---|---|---|---|---|
| No. of cases, Male | 7 | 14 | 9 | 10 | 5 | 8 | 5 | 7 | 7 | 4 |
| Female | 4 | 2 | 7 | 8 | 6 | 4 | 6 | 4 | 3 | 1 |
| Total ... | 11 | 16 | 16 | 18 | 11 | 12 | 11 | 11 | 10 | 5 |

| *Ages in years, under* | *11* | *12* | *13* | *14* | *15* | *16* | *17* | *18* | *19* | *20* |
|---|---|---|---|---|---|---|---|---|---|---|
| No. of cases, Male | 1 | 1 | 2 | 3 | 1 | 1 | 1 | 1 | — | — |
| Female | 3 | — | — | 2 | 1 | — | 1 | — | — | 1 |
| Total ... | 4 | 1 | 2 | 5 | 2 | 1 | 2 | 1 | — | 1 |

| *Ages in years, under* | *21* | *24* | *25* | *26* | *29* | *30* | *31* | *36* | *37* | *43* |
|---|---|---|---|---|---|---|---|---|---|---|
| No. of cases, Male | — | 1 | 1 | 1 | — | 1 | 1 | — | 1 | 1 |
| Female | 1 | — | — | — | 2 | — | 1 | 1 | 1 | — |
| Total ... | 1 | 1 | 1 | 1 | 2 | 1 | 2 | 1 | 2 | 1 |

The age distribution of the fatal cases is as follows:—

| *Ages in years, under* | *1* | *2* | *3* | *4* | *5* | *6* | *7* | *8* | *9* | *10* |
|---|---|---|---|---|---|---|---|---|---|---|
| No. of cases, Male | 2 | 3 | 1 | 2 | — | 1 | 3 | 1 | 3 | 1 |
| Female | 2 | — | 1 | 1 | — | 1 | 2 | — | — | — |
| Total ... | 4 | 3 | 2 | 3 | — | 2 | 5 | 1 | 3 | 1 |

| *Ages in years, under* | *11* | *12* | *13* | *14* | *15* | *21* | *29* | *31* | *37* | |
|---|---|---|---|---|---|---|---|---|---|---|
| No. of cases, Male | — | 1 | 2 | — | 1 | — | 1 | 1 | 1 | |
| Female | 1 | — | — | — | — | 1 | — | 1 | — | |
| Total ... | 1 | 1 | 2 | — | 1 | 1 | 1 | 2 | 1 | =34 |

The case fatality is 22·1 per cent. This is higher than in most of the epidemics abroad, of which we have records; and it is consistent with the thesis that mild cases of the disease have escaped recognition.

* The age of one child could not be ascertained.


POLIOMYELITIS
DEVON AND CORNWALL IN 1911.
DISTRIBUTION OF 154 CASES.
MAY to 16th SEPTEMBER 1911.

*Time elapsing between date of onset and date of deaths in fatal cases.*

The ages of the fatal cases and the duration of the illness was as follows :—

| | | | | | |
|---|---|---|---|---|---|
| No. of cases ... | 2 | 1 | 1 | 2 | 1 |
| *Age ... ...* | *10 wks.* | *3 mths.* | *9 mths.* | *$1\frac{6}{12}$ yrs.* | *$1\frac{10}{12}$ yrs.* |
| Duration of illness in days | 1 and 4 | 5 | ?14 days | 5 and 7 | 3 |
| No. of cases ... | 1 | 1 | 3 | 2 | 4 |
| *Age in years...* | *2* | *$2\frac{11}{12}$* | *3* | *5* | *6* |
| Duration of illness in days | 5 | 9 | 1, 3 & 6 | 2 | 3,3,4 & 9 |
| No. of cases ... | 1 | 1 | 2 | 1 | 1 |
| *Age in years...* | *$6\frac{9}{12}$* | *7* | *8* | *$8\frac{6}{12}$* | *9* |
| Duration of illness in days | 4 | 8 | 2 and 3 | 3 | 5 |
| No. of cases ... | 1 | 1 | 2 | 1 | |
| *Age in years ...* | *10* | *11* | *12* | *14* | |
| Duration of illness in days | within 24 hours | 5 | 1 and 5 | 3 | |
| No. of cases ... | 1 | 1 | 2 | 1 | |
| *Age in years ...* | *20* | *29* | *30* | *36* | |
| Duration of illness in days | 38 | 3 | 5 and 21 | 3 | |

Therefore, irrespective of age,

1 case died within 24 hours of onset of the illness ;

3 cases died 1 day
3 cases died 2 days
9 cases died 3 days
3 cases died 4 days
7 cases died 5 days
1 case died 6 days
1 case died 7 days
1 case died 8 days
2 cases died 9 days
1 case died 14 days
1 case died 21 days
1 case died 38 days

} after onset of the illness.

## Clinical Characters.

In the tables at the end of this report will be found an analysis of the clinical facts which I have obtained from personal observation and through the assistance kindly given me by many medical practitioners, regarding each of the 154 cases referred to above.

An account of the general clinical characters of acute poliomyelitis, compiled from the literature of the subject by my colleague Dr. Macewen is published together with this report.

Here, therefore, it will suffice to refer to the salient points of the Devon and Cornwall cases. Examples of all the different types of the disease as described by Ivar Wickman were seen among the cases tabulated, with the exception of the "ataxic" type. It is, however, possible that the medical attendants may have observed such "ataxic" cases among their patients.

In the majority of cases the paralysis which occurred was of the *flaccid* type ; very few cases presented signs of *rigid* paralysis.

The paralytic cases concerning which definite information was obtainable could be classed as follows :—

*Paralysis of Limbs.*

| | | | |
|---|---|---|---|
| Both legs ... ... ... | 22 | Both arms and both legs ... | 9 |
| Left arm and both legs ... | 6 | Right arm and right leg ... | 1 |
| Right arm ... ... ... | 5 | Right leg ... ... ... | 2 |
| Left arm ... ... ... | 3 | Left leg ... ... ... | 4 |

*Other Paralysis.*

| | | | |
|---|---|---|---|
| Eye muscles ... ... | 2 | Legs, arms, trunk, larynx, diaphragm and intercostals | 1 |
| Eye and legs ... ... | 1 | Arms and intercostals ... | 1 |
| Eye and general paresis ? paralysis of intercostal muscles ... ... ... | 1 | Legs and intercostals ... | 1 |
| | | Legs, abdominal muscles and intercostals ... ... | 1 |
| Eye and right arm ... ... | 1 | Arms, legs and intercostals | 4 |
| Face ... ... ... ... | 2 | Arms and respiratory muscles | 1 |
| Legs, right arm, wry neck... | 1 | Legs, intercostals and diaphragm ... ... ... | 1 |
| Leg and jaw ... ... | 1 | Left arm, intercostals and neck ... ... ... | 1 |
| Left leg, spine and back ... | 1 | Right arm, intercostals (widely dilated pupils) ... | 1 |
| Right leg, back and neck ... | 1 | Legs, arms and sphincters ... | 1 |
| Arms, legs and back ... | 1 | Legs, arms, thorax and bladder ... ... ... | 1 |
| Leg and neck ... ... | 1 | All four limbs and squint ... | 1 |
| All four limbs and occipital muscles ... ... ... | 1 | Whole body ... ... | 1 |
| Face and right leg ... ... | 1 | Muscles of neck ... ... | 1 |
| Left arm, left leg, right side of face ... ... ... | 1 | | |
| Legs, 7th nerve and neck ... | 1 | | |
| Muscles of respiration ... | 2 | | |

*Other forms of Paralytic Symptoms.*

| | | | |
|---|---|---|---|
| Muscular rigidity ... ... | 3 | Muscular rigidity in right leg and back ... ... | 1 |
| Inability to stand ... ... | 1 | Stiff neck ... ... ... | 1 |
| Undefined ... ... ... | 4 | "Laboured breathing" ... | 1 |
| Rigidity in back ... ... | 1 | | |

*Paresis.*

| | | | |
|---|---|---|---|
| Both legs ... ... ... | 6 | Legs and back ... ... | 2 |
| Left arm and legs ... ... | 1 | Legs and arms ... ... | 1 |
| Right arm and legs... ... | 1 | Left arm ... ... ... | 1 |
| Left leg ... ... ... | 1 | | |

Of the remaining cases 27 are definitely stated to have had no paralysis and in 14 cases there is no record.

The cases under review furnish the following considerations :—

*Date of onset.*—The dates in the column in the table giving the date of the onset in each case records as nearly as possible the day on which each of the cases was found to be ill. It was difficult to determine in many cases whether acute attacks were preceded by prodromata or for how long the patient was manifestly ill before acute symptoms were noticed. In some few cases there appeared to have been no prodromata.

*The temperature* of the patients was not always taken ; the majority of the attacks were certainly accompanied by fever in the earlier stages. The temperature of the patients where the information is available seems seldom to have risen above 102·5 Fah. High fever was rarely observed.

*Headache.*—In 120 cases of which I have definite information, 114 suffered from headache ; and in 6 cases, it is said, there was no headache.

This symptom may exist in the case of infants who cannot talk, when it is difficult to make certain of its presence.

*Vomiting.*—Vomiting when it occurred was one of the first signs of the onset. It rarely persisted after the acute attack had culminated, generally subsiding with the initial symptoms. In 120 cases of which there is definite information, vomiting occurred in 79 cases ; it was absent in 41, but in a few of these cases "nausea" is recorded. In 34 cases there is no record.

*Twitching or convulsions.*—There were two cases in young children where convulsions occurred at the commencement of an attack, and in one of them "convulsions" occurred at intervals throughout the whole of one day.

Twitching of a muscle or group of muscles occurred in 34 cases, and is recorded as absent in 40. No mention is made of the condition in 80 cases.

Twitching of the muscles of the face, especially of the muscles round the mouth, was observed in several cases ; and in two cases there was persistent sniffing, independent of any catarrhal symptoms of the nasal passages.

*Delirium.*—Was comparatively rare. It only occurred in severe cases and in those which showed meningeal symptoms.

As a rule the sensorium remained quite clear, and even in fatal cases the patients were conscious to within a few minutes of death.

"Delirium" is recorded as being present in 21 cases, and as absent in 52. No mention is made of this condition in 81 cases.

*Hyperæsthesia.*—In 41 cases definite pains in groups of muscles or limbs, &c., are recorded ; in 14 cases hyperæsthesia is said to have been present, but no details are furnished. In 81 cases there is no record ; but it is possible that among these cases the condition may have been present, for in a large number of cases there was tenderness to the touch, and young children would scream on being moved.

Extra sensitiveness to touch or to movement was definitely stated to be absent in 16 cases ; and certain patients developed paralysis without apparently experiencing any pain.

*Anæsthesia.*—In only two cases is loss of sensation recorded ; in one case "almost complete anæsthesia" (Case 102), and in the other "complete loss of sensation lower third of flexor surface, and one-half extensor surface of the right arm" (Case 18).

*Irritability of temper* is recorded as being present in 47 cases and as absent in 17. There is no record in 90 cases. When present the children were fretful and peevish, and resented being disturbed in any way.

*Drowsiness.*—This sign was a most marked one in the cases I saw. The patients appeared to sleep, and if aroused were irritable and cross, and wishful to be allowed to sleep again, and if left alone would "instantly go to sleep."

This condition even in severe cases was markedly different from coma, the patients could always be roused and their minds were quite clear.

In case 21, a man aged 42, this sign lasted for three days ; at intervals he was awakened to take food, and he dropped off to sleep immediately after eating.

It was observed that in children one of the earliest signs was that the child "lay about and slept."

Sometimes the patient would twitch or start while asleep, but generally the sleep was undisturbed.

This condition of drowsiness was observed in 80, and was absent in 20 cases of which there are records.

*Kernig's Sign* was recorded as being present in 63 cases, though not always in both legs. It is said to have been absent in 41 cases ; in 50 cases there is no record.

In some cases the condition was manifest and unmistakable. As to whether it is elicited in certain cases must depend on the personal factor of the examiner ; many persons in health cannot completely extend the leg on the thigh when the latter is flexed and this fact leads to uncertainty with regard to Kernig's Sign when the examiner has never examined a case where the sign is undoubtedly present. In such cases there is not only inability to extend the leg but also the attempt causes pain in the spine, sometimes to an excruciating extent.

*Tache Cérébrale.*—In certain cases this sign was produced within a few seconds ; but unless the actual time of its manifestation is stated the record of cases in which it was observed is of very little value. However, it is said to have been present in 54 cases and absent in 22 ; in the other 78 cases it is not mentioned.

*Condition of reflexes.*—The reflex generally examined for was the patellar reflex, the plantar reflex was less often investigated. Very few examinations for other reflexes were made or recorded.

With regard to the patellar reflex, it appeared that if the patient was seen at quite an early stage the reflex might be found exaggerated, but in many cases it was soon lost. In some cases the reflex was only absent on one side. A record of the condition of the reflexes in 80 patients will be found in the table of cases. In 15 cases the reflexes were stated to be normal or present and in 59 cases there is no record.

*Bladder symptoms.*—In the notes on eleven cases there are references to retention of urine in five cases, to incontinence of urine in three cases, to paralysis of the bladder in one case, to incontinence of urine and fæces in one case, and in one case paralysis of the sphincters is mentioned.

*Epistaxis.*—In six cases there is a record of epistaxis. In one case, No. 67, the bleeding became itself a source of danger owing to its effect on the pulse. In 46 cases there was no epistaxis ; in the remaining cases there is no record.

*Discharge from throat or nose.*—This was only noted in four cases and was absent in 52 cases in which this sign is noted. In one case there was a discharge from the ears.

*Sore throat.*—In only 59 cases is distinct mention made of the condition of the throat. In 39 of these cases it is stated that there was no sore throat, in nine that there was "slight" sore throat, in seven that there was "sore throat." In one case the throat is said to be congested, in another that there was redness of the fauces. In the remaining two cases difficulty in swallowing is recorded but this may have been due to paralysis of certain of the muscles of deglutition. Case 68 was at first thought to be suffering from diphtheria and diphtheria anti-toxin was administered.

*Enlargement of Glands.*—Inquiry was made as to whether glandular enlargements were present especially in regard to throat glands. In only 40 cases was any record as to the presence or absence of enlarged glands received. In 35 the records stated that there were no enlarged glands, in one that an enlargement of the glands of old standing existed, in three cases the "throat glands" are reported to have been enlarged, and in one case that the glands of the neck were enlarged and that there were small ulcers on the tongue.

*Retraction of the Head and Neck.*—This sign varied from what amounted to mere rigidity of the neck muscles, making attempt at moving the head and neck painful, to such complete retraction of the head as bring the occiput almost back to the spine. In case 121 this retraction was so extreme as to cause surprise that it was anatomically possible.

In 57 cases there is a record of retraction of the head, and in 56 cases that no such retraction took place ; in 41 cases there is no record.

"*Rashes*" *and* "*Skin Eruptions.*"—These occurred in 23 cases in localised groups of cases and were variously described. It became a matter for consideration whether the presence of the "rash" was due to some predisposition of the patients, or of the circumstances of their surroundings, or to variation in the seat of attack by the virus; or whether indeed the record of its appearance was influenced by the personal factor of the medical attendant. Some observers recorded "rashes" in several of their cases, others stated that no rashes had occurred.

In addition several cases were described as "flea bitten" or "insect bitten." In 89 cases it is definitely stated that there was no "rash"; in 42 cases there is no record.

*Herpes.*—In seven cases herpes is recorded as having occurred. In two of these cases it is described as "herpes labialis"; in one on the right cheek; in another on the left cheek; and in one as "on side of mouth"; in two the position is not described.

*Constipation and Diarrhœa.*—Constipation is recorded as a condition present in 45 cases, diarrhœa in seven cases, diarrhœa followed by constipation in three cases, and as absent in 11 cases. In 88 cases there is no record.

In a few cases it is stated that constipation when relieved was associated with very large stools of peculiarly offensive odour. In one fatal case of a woman aged 30 (case 148) where this condition obtained the death certificate was signed "intestinal toxæmia." In many cases there is a record that the tongue was thickly coated or furred; and this was the condition in nearly every case which I had the opportunity of examining.

*Sweating* was profuse in certain cases, but I have no numerical record of its frequency.

The following are the clinical notes of a few cases, which occurred in Devon and Cornwall. As I am unaware that temperature charts of acute poliomyelitis have been published in England, the charts of three cases are given. In one of these cases the patient died; in the other two cases, one a severe case, the patients recovered though some paralysis remains in each instance.

*Case* 121.

*From Notes made by Dr. W. G. Gray.*

*Female child*, aged 3 years, previously healthy.—Was first noticed to be poorly on Friday, August 18th.

*August 19th*.—Slept well last night, was apparently in good health up to dinner time, and played about in the morning Then complained of feeling poorly just before 1 p.m. and was put to bed. Was listless, and vomited. That evening her mother found child could not walk or stand. There were no catarrhal symptoms of nose or throat at any time during the illness.

*Sunday, August 20th.*—Passed a restless night. Seen by medical attendant at 6 p.m. Child looks ill and has pain in the head. T. 101·4, P. 128. Is unable to stand owing to loss of power in both legs. Patellar reflexes absent. Kernig's sign on left side. Cannot bear to be moved; there is tenderness down the spine. Tongue coated. Bowels are constipated—no action since Friday, when some salts and senna were given to the child. No tache cérébrale. Is very drowsy. Had had a fair night; during the morning she ate an apple (raw) and three or four plums and two biscuits. This was the last solid food she ate. She drank milk freely. Milk obtained from their own cows.

*August 21st.* Passed a restless night. Condition much the same. Tâche cérébrale is now marked. Head retracted, very fretful but does not speak. No action of the bowels. Much tenderness all over the body. T. 100·6°, P. 112.

*August 22nd.*—Tache cérébrale very marked. Plantar reflex: left normal; right absent. Retraction of head and neck. Very drowsy. Has staring look, with eyes fixed on the ceiling. Marked red flush across forehead. T. 99·4, P. 98.

*August* 23*rd.*—Paralysed from waist downwards, practically unconscious; has extreme retraction of the head and neck; so much so that the back of the head almost touches the spine. Passed urine involuntarily. Is unable to swallow fluid. T. 98, P. 76.

*August* 24*th.*—Died.

Patient was given urotropin grs. 5. every four hours from August 20th.

A post-mortem examination was ordered by the coroner in this case.

Plate I, Figs. 1, 2 and 3, show micro-photographs of a section of the spinal cord of this case, prepared in Dr. Mervyn Gordon's laboratory, and prove conclusively that the cause of death was acute poliomyelitis.

*Case* 31.

*Clinical Note by Dr. W. G. Gray.*

*Married Woman*, aged 36, 4 para.

Was confined of a child at 8 p.m. July 22nd, after a normal labour. Was attended by the Parish Nurse (Nurse M.).

*July* 24*th.*—Milk began to come in breasts. Had slight headache. Temperature and pulse normal. Lochia normal.

*July* 25*th.*—Normal day.

*July* 26*th and July* 27*th.*—Complained of headache, otherwise condition normal.

*July* 28*th.*—Headache worse, vomited. At 4.15 p.m. had a convulsion followed by others at 6 p.m., 6.30 p.m., 9.15 p.m., 10.50 p.m. Seen by her doctor the same evening at 8 p.m. T. 99°. Pulse 78. She was very drowsy and stuporous, but could be roused and put out her tongue when told to do so, did not answer when spoken to. Urine ⅓rd albumen.

*July* 29*th.*—Convulsions 12.35 a.m., 6 a.m. None afterwards. Vomited. Condition much the same; pointed to her head when asked if she was in pain. Was able to speak a few words, but with much difficulty. Still very drowsy. Lochia normal. T. 97·8°. P. 62. Urine drawn off by catheter. Bowels acted twice. Very restless. Said her left arm was numb.

*July* 30*th.*—No change in her condition, could talk better, but still with difficulty. Towards evening the Nurse noticed that the patient's left arm was useless.

*July* 31*st.*—Saw patient towards evening. Still in a drowsy condition, complains of great pain in forehead and back of neck but speaks only with much effort and requires some rousing. T. 97·2. P. 52.

Kernig's sign absent. Patellar reflexes, only feeble response. Plantar reflexes exaggerated. Tâche cérébrale. No rash. Lochia normal. Complete paralysis (motor) of left arm.

*August* 1*st.*—No change in her condition.

*August* 2*nd.*—Headache rather better. No sleep except of a restless character. Is very slow to understand when spoken to and still speaks with difficulty and then only using the shortest words. T. 98°, P. 62. No improvement in the left arm.

*August* 4*th.*—Much less headache, had two hours sound sleep last night. Head still retracted, she still "burrows" her head into the pillow.

*August* 6*th.*—Patient continues to improve though paralysis remains as before. Has begun to take interest in her surroundings. Patellar reflexes still very feeble. Headache is much better.

*August* 9*th.*—Decidedly better in every way. No headache but still complains of being sore and stiff about the neck. Can now flex her forearm a little and is able to hold a cup in her fingers.

From this date she made uninterrupted progress and was able to be moved out of bed on August 12th. Paralysed arm gradually improving.

*Present condition. October* 3*rd.*—Is able to perform most of her household duties. Left arm is still weak and there is some wasting of the muscles of shoulder (Deltoid), upper arm and forearm. Complains that

her sight has been impaired since her illness. Is unable to lift any heavy object. The finer movements of her wrist and fingers are impaired as she is only able to sew or knit for very short periods.

*Case* 80.

*From Notes by Dr. A. W. D. Hunt.*

Male, age 3.—Onset of the disease on 28th August; the child being very drowsy, and unable to stand. First seen by doctor 9.30 p.m. on 28th August. There was general muscular paresis, but no marked paralysis. Vomited on 29th and 30th, but this vomiting was not a marked symptom. On 30th a well marked squint developed the internal rectus and superior oblique muscles of the eye being affected; the temperature which had been normal rose to 102° F. The retraction of the head increased, and there was a certain amount of subsultus tendinum.

When seen on 31st at 11 a.m. the child was in extremis, showing symptoms of heart failure with possible paralysis of the intercostal muscles. Death took place at 5 p.m.

Micro-photographs of a section of the spinal cord of this case made in Dr. Mervyn Gordon's laboratory are reproduced in Plate II; they show without any doubt that the cause of death was acute poliomyelitis.

In the previous year there had been a fatal case of illness in this household. On 7th September, 1910, a female child, aged 5, was attacked by sudden illness; she died on 13th September, 1910. The medical attendant informs me that the head symptoms were marked, but there was no muscular paralysis. He signed the death certificate as "Tubercular meningitis, asthenia." Looking back he can see a marked resemblance of symptoms between the fatal attacks of these two children.

*Case* 114.

*From Notes by Dr. Leonard B. Betts.*

A farmer, aged 29. Taken ill on 15th September with pain in the loins and bad headache; said he ached all over. He did not vomit. He continued at work on the farm that day, and he passed a very restless night. On 16th September he remained in bed all day, took some salts in the morning, bowels acted twice during the day. He vomited once: this was the only occasion when he vomited. On 17th September he was seen by his medical attendant at 8 p.m. Complains of "soreness of neck" and "sore throat." Slips down into the middle of the bed and has to be lifted up on to the pillow. Quite conscious and replies readily to questions. Complains of difficulty in breathing. Had walked up stairs to bed in the morning, and the difficulty in breathing had came on in the afternoon. Temperature 101° Fah. Pulse 100 (regular). Respiration 36, gasping in character; alæ nasi working; patient says it was hard work to breathe. Tongue furred, breath very offensive, fauces red, no membrane in the throat, no nasal discharge, no herpes, no rash, no muscular twitching, no convulsions, no irritability of temper, no drowsiness, tache cérébrale marked, no epistaxis, no glandular enlargements. Patella reflex cannot be elicited, plantar reflex is increased, sphincters not affected. Kernig's sign very marked, no loss of sensation, can swallow liquids. The chest wall does not move on inspiration. Heart's apex in normal situation, no bruit, heart sounds somewhat forcible at apex. No impairment of resonance over the back and front of chest, breath sounds only slightly audible. Abdominal respiration marked, abdomen not distended. No enlargement of spleen. No rash. He can raise his legs from the bed, there is obvious loss of muscular power in left arm, the grasp of the right hand is good, he cannot sit up in bed, he has no strength in his neck or back, he cannot raise his head from the pillow. He appears to have retraction of the head and neck.

He became very restless during the night of the 17th, but passed into a state of insensibility an hour before death ; breathing became less and less and ceased at 4 a.m. on the 18th. No lumbar puncture and no post-mortem examination were made.

Patient lived in a large farmhouse of eleven rooms, of which six are bedrooms. There is no overcrowding, the house is clean. The other inmates of the house are the patient's mother, aged 56 ; his sister, aged 24 ; his brother, aged 14 ; and a man servant, aged 23. There had been no previous illness in the house, and the patient had never been ill before. His brother had slept with him up to the 15th September. There had been no illness or deaths among the animals on the farm.

In the literature of poliomyelitis mention is made that certain cases have developed a cutaneous eruption not unlike the rash of chicken-pox. In this connexion, and also in respect of the possibility of intra-uterine infection, the following case has points of interest.

A male child was born on 4th August, the labour was difficult and the child was delivered by forceps. On the 6th August, the nurse, Nurse W., informed the doctor in attendance that the child had been vomiting all night and had a rash on his chest. On examining the child the doctor found that there was an erythematous rash extending practically all over the body, and that scattered over this rash were punctate vesicles not unlike chicken-pox. There was no rise of temperature, tache cérébrale was very marked, retraction of the head doubtful, Kernig's sign (if reliable in an infant two days old) was present.

This child was seen in consultation by the medical officer of health, and the conclusion arrived at was— that as the child was only two days old, it could not be a case either of cerebro-spinal meningitis or of acute poliomyelitis. In three days the vomiting ceased and the rash disappeared.

On 6th August, the mother of the infant developed an acute headache with rise of temperature ; these symptoms disappeared on 9th August. She did not vomit ; Kernig's sign was absent.

On the 28th August, while attending another confinement case, Nurse W. (Case 97) was suddenly attacked by acute occipital headache, clonic contractions of the masseter muscles, nausea, retching and giddiness. Kernig's sign was present and reflexes exaggerated. Patient complained of pain in the back of neck and over spine, and frequency of micturition. The urine contained a trace of sugar but no albumen.

There was no history of any wound.

She was seen in consultation by the medical officer of health who agreed with the medical attendant that the case was one of acute poliomyelitis.

On 7th September the patient was evidently better, but she complained of subjective sensations—flashes of light and headache. Her reflexes were all increased and she had ankle clonus.

This case is referred to on p. 49.

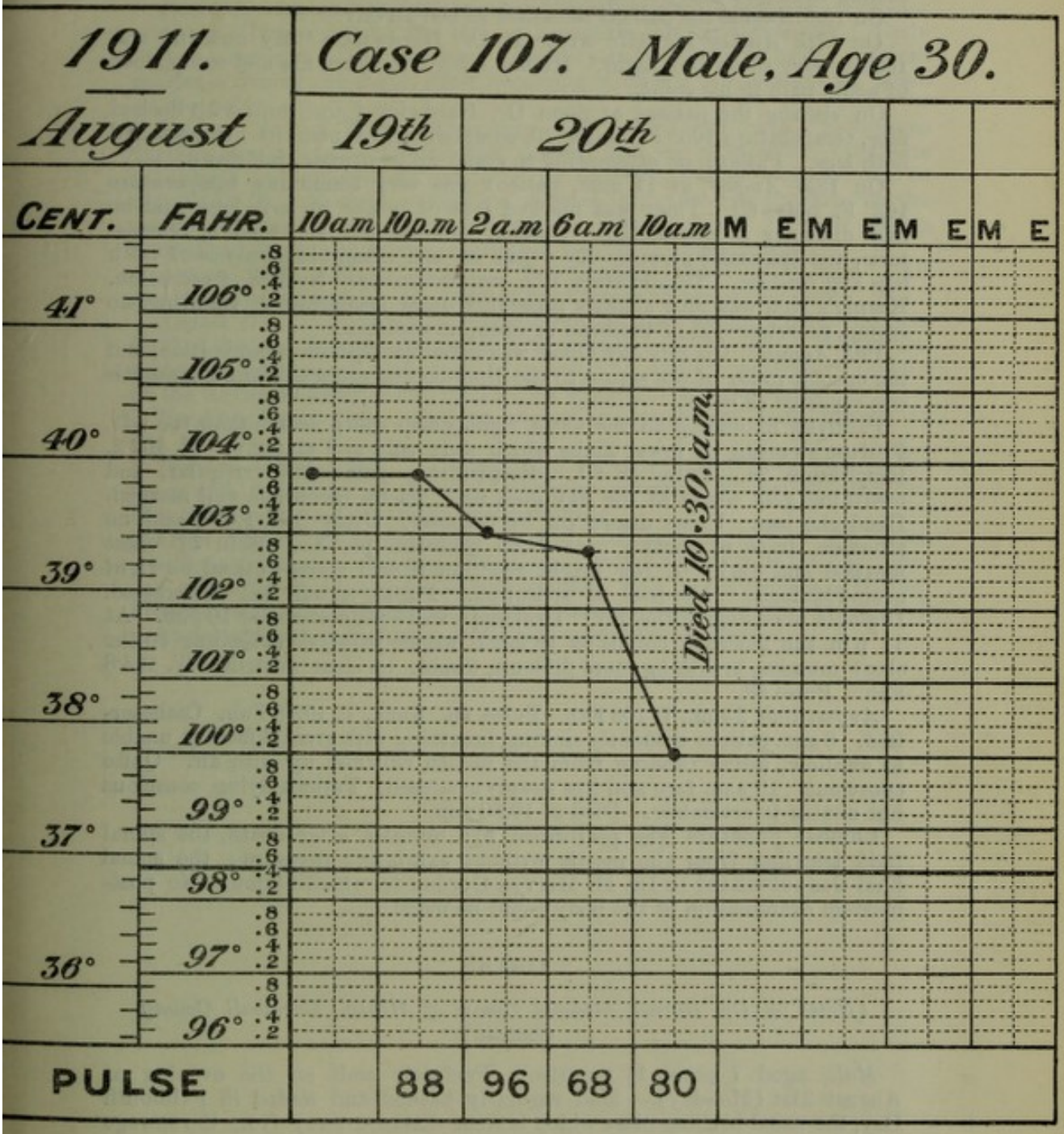
The following cases are instructive in their clinical aspects :—

*Case* 107.

*From Notes furnished by Dr. Arthur Hardwick.*

*Male*, aged 30, married, no family. Indian civil servant. No history of phthisis or of syphilis ; had had "Fever" in India some years ago, and a recent operation for hæmorrhoids. Came to New Quay towards the latter of July. Previous to this and subsequent to the operation he had been boating on the Thames during the intensely hot weather. On July 30th the skin of both legs, from the ankles to the middle of the thighs, was intensely red, blistered and inflamed from the effects of exposure to the sun. Otherwise the patient was in the best of health. Rectal bougies were passed on 30th July, 6th and 13th August. Up to this last date the patient was in good health, daily playing golf, cricket, bathing, &c.

On *12th August* patient played in a cricket match at Camborne. Heavy rain fell during the match, but play was continued ; patient got wet


1911.
August
Case 107. Male, Age 30.
19th
20th
CENT.
FAHR.
10a.m
10p.m
2a.m
6a.m
10a.m
M E M E M E M E
41°
106°
105°
40°
104°
103°
39°
102°
101°
38°
100°
99°
37°
98°
97°
36°
96°
Died 10·30, a.m.
PULSE
88
96
68
80

through and returned to New Quay in his damp clothes. When Dr. Hardwick visited him on 13th August to pass the rectal bougie, he appeared to be quite well, but complained of feeling tired.

On 14*th August* patient went by rail to Penzance, and thence by motor to the Land's End. The heat was intense ; patient spent the day bathing, paddling, and climbing the cliffs.

On 15*th August* he played two rounds of golf, although he complained of feeling tired and that his legs ached.

On 16*th August* the patient remained in bed all day.

On 17*th August* his wife wrote to Dr. Hardwick : "My husband was feeling very seedy last night. He had a high temperature and complained of acute pain in his spine."

On visiting the patient at noon Dr. Hardwick found him with flushed face, temperature 103° F., pulse 70, complaining of pain in his back and both legs. Patient, on attempting to stand up to urinate, fell down.

On 18*th August*, at 11 a.m., patient was very tremulous, temperature 102° F., pulse 60. There was marked loss of power in both legs, and he had difficulty in micturition. Patellar reflexes, plantar, tendon, cremasteric, and epigastric absent. By 4 p.m. he had complete paralysis of both legs and bladder. Temperature 102, respiration 24. No loss of sensation. Kernig's sign was only slightly present, frontal headache, no vomiting, no cramp, no retraction of the head or neck, no diplopia.

19*th August*. On this day blood was taken for opsonic examination, and the opsonic index of the serum to the diplo-coccus intracellularis meningitis was reported as =0·9.

Paralysis extending to the arms, right grasp more feeble than the left. Profuse sweating of lower limbs which were cold and clammy. T. 102·2. Respiration 24. Pulse 48–60. Respirations somewhat irregular, and breathing gets worse as the day goes on. Tache cerébrale well marked. Headache less. Urine drawn off by catheter. Urine S.G. 1,034, acid, no albumen, trace of sugar ; urea 0·035 grammes per c.c. Skin of chest mottled and dark. A few bright red punctiform spots noticed on right groin ?Miliaria. No loss of sensation. Pupils equal but contracted. Diplopia once during the day. Urotropin was administered at 10 p.m. At 11 p m. the patient's condition is much worse, paralysis extending to the chest muscles, breathing more difficult, unable to clear nostrils. T. 103·8 Fah. Pulse 88.

*August* 20*th*, 2 a.m., T. 103 Fah. Pulse, 96. 6 am., T. 102·8 Fah. Catheterised. 8 a.m. patient evidently sinking, breathing with great difficulty, unable to swallow, saliva running from the mouth churned up with air. Quite conscious. 10 a.m. face and lips deeply cyanosed. Patient dying, conscious but unable to articulate. Died at 10.30 a.m.

Lumbar puncture was performed five minutes after death, the spinal fluid trickling from the needle without exhibiting pressure ; the spinal fluid was submitted to Dr. H. Mervyn Gordon for examination. No post-mortem examination of the body could be made.

### *Case* 67.

(*Notes by Dr. Burnet, Medical Officer of Health, Cornwall County Council.*)

*Male*, aged 7 years 11 months. Perfectly well on the evening of August 21st (Monday). Had regularly bathed and waded in Falmouth Bay, the usual bathing place which is some distance away from the sewage outfall pipe.

*Tuesday, August* 22*nd*, said he was too tired to get up—complained of stuffiness at the back of nose and inability to breathe through it. Eventually brighter and journeyed to Truro by train with his mother and sister, where he drove for two hours in a cab during which time he looked rather heavy, his eyes becoming suffused. Could eat no lunch and so was taken home to bed complaining of headache. He had complained of slight pain in the side in the morning, but he was more cheerful returning from Truro being noticed to sing and laugh whilst in the railway carriage. The veins of his eyelids occasionally filled producing a purple colour which

soon vanished. 9 p.m., temperature 103°, both knee jerks present—the right one slightly deficient. No tâche cérébrale—suspicion of Kernig's sign.

*Wednesday, August 23rd*, 8 a.m. Temperature 101°—no vomiting since illness—refuses food. 2.30 p.m., temperature 102°. Rigidity on standing —Tache Cérébrale well marked, appearing in 5 seconds—right knee jerk absent, left present but feeble—Kernig's sign well marked. 6.30 p.m., Epistaxis. 7 p.m., temperature 102°, no pain—feels better, but speaks of "ceiling going round."

*Thursday, August 24th*, both legs paraplegic—perfectly helpless. Bladder symptoms (frequency). No loss of sensation but hyperæsthesia. Acute herpes labialis, rapidly increasing. Rigidity and pains in the neck. Violent epistaxis during the night, itself becoming a danger on account of effect on the pulse.

*Friday, August 25th*, temperature 103° max., 103° min. Symptoms further exaggerated—rigidity still present and no retraction. Complete paraplegia but alternating extension or flexion. Plantar reflexes can be obtained. Trunk rigid, complains of pains in the toes on pressure at the occiput. Ophthalmoscopic examination shows retinæ normal, vision normal, no ocular symptoms, no bulbar symptoms, feels better.

*August 26th*. Much worse. Temperature about 101° max. All symptoms exaggerated. No further implication of the spinal cord.

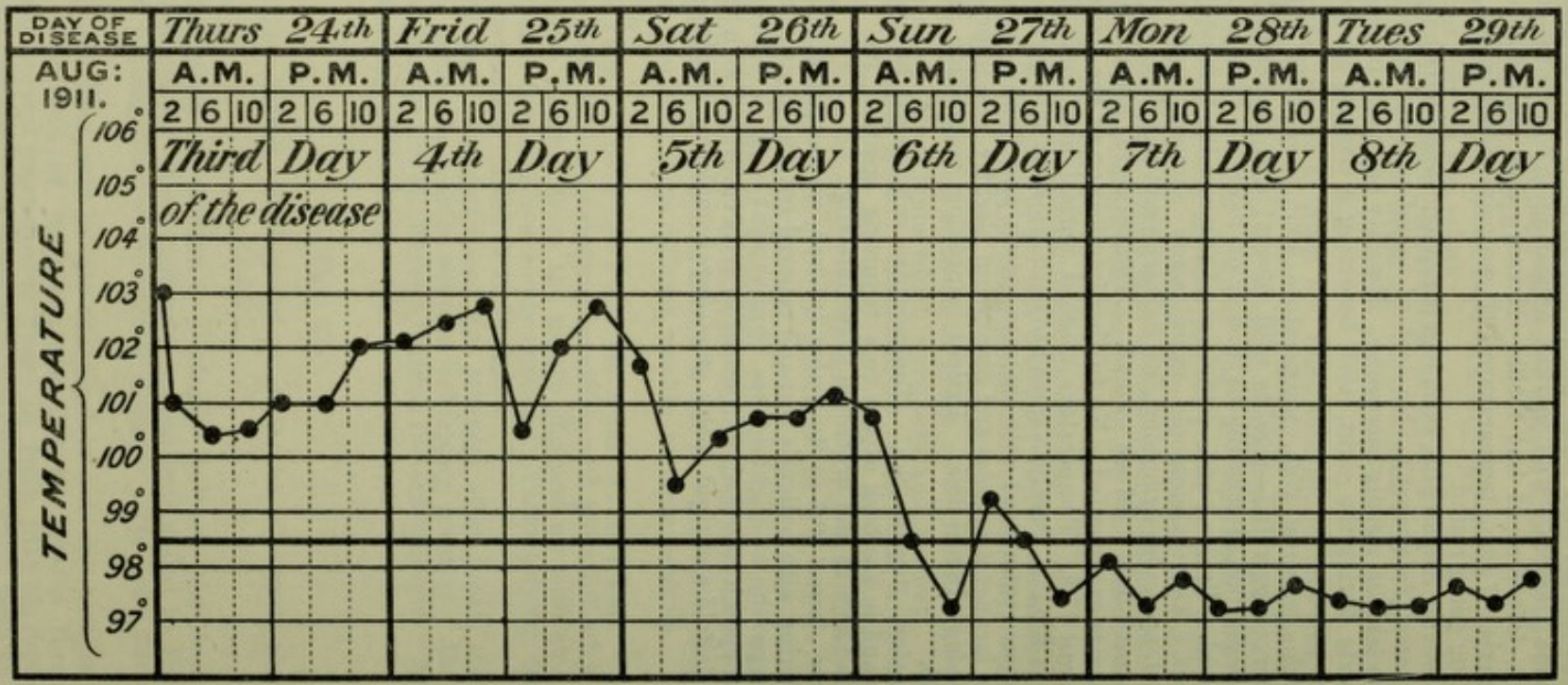
*August 27th*. Temperature reached normal line about 6 a.m. and was 99·2° at 2 p.m. Paralysis spread to both arms but chiefly right. Bulbar disturbances commenced with staccato speech, and finally paralysis of certain of the laryngeal muscles and diaphragm slightly, and intercostals.

*Monday, August 28th*. Patient improving, respiration still between 32 and 40.

*Tuesday, August 29th*. Temperature sub-normal but great pain in the knees and in the back of the neck requiring opiates. No urinary trouble. Has had aspirin and urotropin systematically.

From *August 29th* to *September 4th* had violent pains behind the knees interfering with patient's rest and relieved only by aspirin in the day time, and nepenthe at night—takes nourishment well, is extremely irritable. Proper speech returned, movement in arms recovered with the exception of right deltoid and right feeble grip—legs completely paralysed.

## Case 67. Male Aged 7 11/12 Years.



*Case* 116.

(*Clinical Note by Dr. E. George Hall.*)

Male, aged 6 years, returned to Bristol on Saturday, August 5th, after a fortnight's holiday at Bude. He was taken ill in the train, with severe headache and fever. On reaching home he was put to bed and given a purge, followed by a saline on the following morning, which acted effectually. He vomited slightly once, immediately after a dose of citrate of magnesia.

On August 6th patient was still feverish ; complained of severe frontal headache, and also pain at the back of the neck, and on that evening the temperature was 103°.

On August 7th seen by his medical attendant in the morning for the first time ; temp. 102·5° ; headache and pain in cervical region, radiating to each shoulder ; tongue furred. The head was retracted, and the muscles of the neck were tender to the touch. Symptoms similar to those of a rheumatic "stiff neck." Evening temperature 102° ; pulse 130.

On August 8th, at 10 a.m., temperature was 102·5°, headache, lassitude, loss of power in the occipital muscles, the head having to be entirely supported, and loss of power in both arms, so that they had to be lifted. Pulse 130. Curious form of breathing : the submaxillary muscles being drawn in at each inspiration. Increased weakness ; headache and cervical pains increased, giving rise to slight cry out occasionally ; mind clear, but apathetic. Bowels tympanitic ; relieved by an enema. Water passed naturally. Knee jerks now absent. Kernig's sign absent.

Lumbar puncture was performed. Spinal fluid was evidently at high pressure, but clear. Smear showed no sign of pus or of organisms. During the day all symptoms became aggravated, all limbs became helpless. Temperature gradually fell, pulse rate also fell, and became rapidly weaker. Extreme apathy, but could be roused. Pupils dilated, but equal. Occasional irritative spasm of the jaws, but no other convulsive symptom. Death took place quietly at about 10 p.m.

There was no vomiting save after the magnesia. There was no intercostal paralysis. There was some paralysis of the muscles of the floor of the mouth, which bulged on inspiration, in the manner of a frog's. There was no paralysis of the diaphragm.

In association with this case is one not included in the list of cases tabulated in this report, inasmuch as it occurred in Bristol.

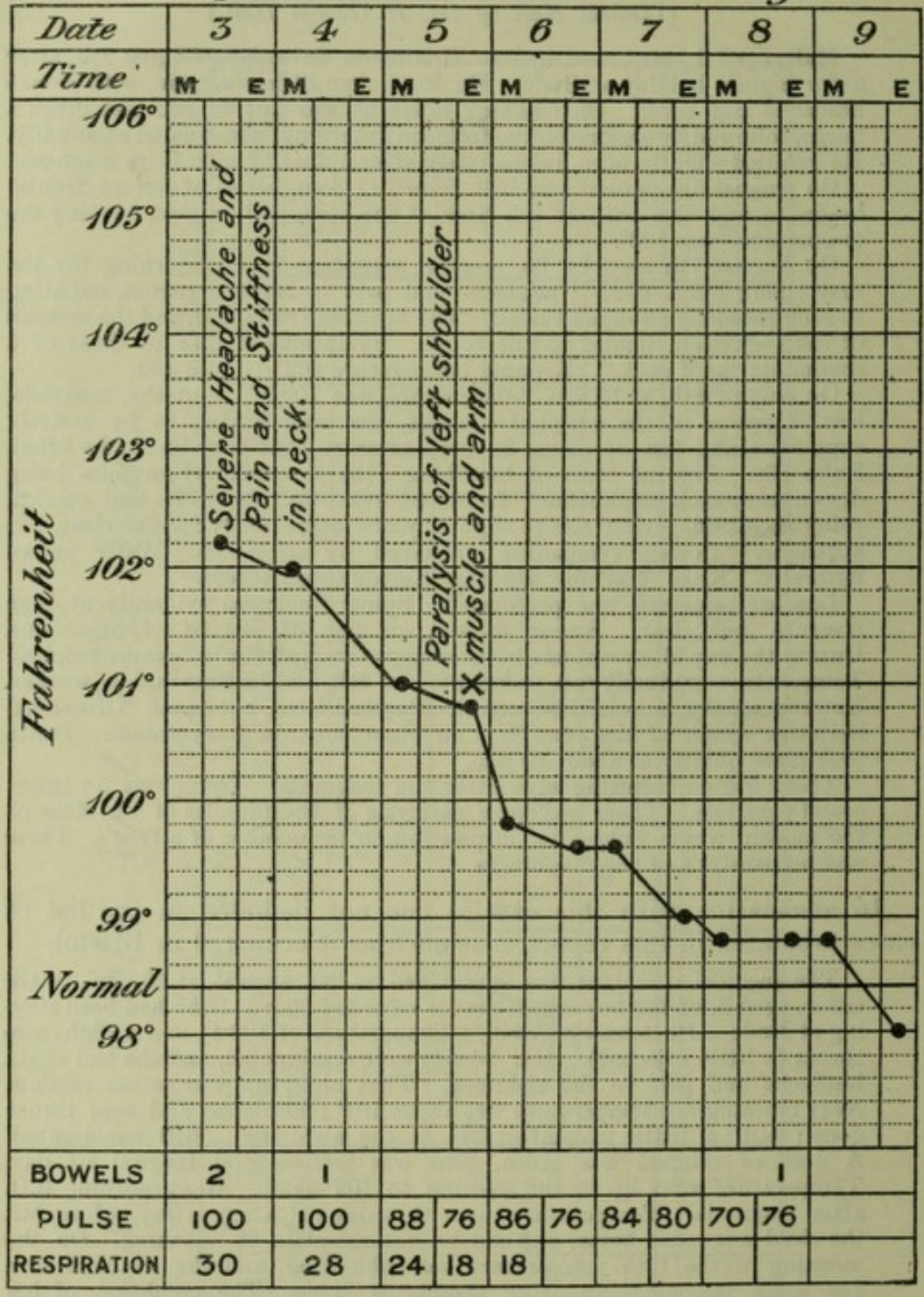
The medical attendant was summoned on 9th August, at 10 p.m., to the sister, aged 3, of the boy whose case is recorded above. She had been staying at Bude. He found her with a temperature of 102·4°, and a slight sore throat (a little injected). Her bowels were constipated, and she had slight headache and pain in the abdomen. That night she was rather restless. Next morning her temperature was about 101° ; headache and sore throat gone ; still a little uncomfortable in the abdomen. Still constipated. A dose of calomel was given. She was restless and languid all day. Temperature went up in the evening to 102° again. Next morning B.O. after magnesia. Temperature same that day (11 a.m.) as day before, but the child was much better and she did not complain of anything. On the morning of the 12th temperature was subnormal, and she was well save for some languor. No other symptoms since. The condition of her nervous system was carefully inquired into every visit, and nothing abnormal was discovered.

But for the circumstances of her brother's illness one would have called the case an ordinary derangement of stomach and bowels. The child did not really complain of anything all the time. On the first night her mother noticed that she looked flushed and was tossing in her sleep, took her temperature and found it 102·4°, and summoned medical aid.

*Case* 112.

The following temperature chart relates to Case 112 in the tabular statement. (Male, aged 8.) On September 21st the paralysis still persisted in the shoulder muscles.

*1911. September. Case 112. Male Aged 8.*




*After Effects.*

Although in a certain number of the patients who were afflicted with paralysis of one or another group of muscles, the paralysis was transitory and recovery more or less complete, there are still many cases in which this paralysis must needs remain permanent. Thus, when the epidemic is at an end it will leave behind victims who will remain crippled for life, some an affliction to their families, and others certain, sooner or later, to require to be maintained at the public cost. It is, indeed, one of the most serious aspects of poliomyelitis—an aspect which should be a special incentive to combating

the disease in all possible ways—that escape from death so often does not mean restoration to normal activity or active life.

### *Curative Treatment.*

There is no known specific remedy for this disease. The treatment usually applied was on general lines and directed to the relief of symptoms. Urotropin was employed in some cases, but the result was not encouraging. Opium administered primarily to relieve pain appeared to afford general assistance to the patient. Once the patient is attacked comparatively little can be done for him, and this constitutes one of the most serious features of an outbreak.

There is a prospect, however, that before long the experimental study of this disease may lead to the discovery of a specific curative agent. The recent work of Levaditi in Paris already inspires the hope that an anti-serum may be obtained from immunised apes.

## Evidence of the Nature of the Epidemic.

From the outset, in view of the need for better knowledge of the nature of the disease, and particularly whether it included cases of cerebro-spinal fever, I endeavoured to secure specimens of spinal fluid for examination by Dr. Mervyn Gordon, and also any material, such as the spinal cord, which was available on post-mortem examination. Examination of the spinal fluid obtained by lumbar puncture will, in practically every case, enable a positive diagnosis of cerebro-spinal fever (spotted fever) to be made if the diplococcus intracellularis of Weichselbaum is found to be present, and the identity of the diplococcus is satisfactorily established.* On the other hand, the absence of the diplococcus, together with the presence of lymphocytes in the fluid, are consistent with the disease being poliomyelitis.

I had to meet many difficulties in obtaining material for examination. The majority of the medical practitioners of Devon and Cornwall had never had occasion to perform lumbar puncture, and many were reluctant to attempt it; the folk of these counties are peculiarly adverse to anything in the nature of "surgical interference," and they strongly resent examination of a body after death.

The difficulties of making anything like a complete post-mortem examination in an isolated country cottage are such that few medical men care to undertake such a task, unless for some strong and urgent reason.

But lumbar puncture and post-mortem examinations were made in a sufficient number of cases to furnish material the examination of which placed the diagnosis of the nature of the outbreak beyond all doubt.

As positive evidence, the results of microscopic examination of the spinal cords in cases 80 and 121 (see Plates I and II) may be referred to; and with material derived from cases 80 and 121

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* See Dr. Mervyn Gordon's Table, p. 125.

typical poliomyelitis has been induced in the monkey. (See Dr. Gordon's Report, p. 128.)

On the negative side, Dr. Gordon found no sign of the diplococcus intracellularis in any of the spinal fluids submitted to him from the cases in Devon and Cornwall.

## Circumstances associated with the Spread of the Disease.

Consideration was given in my inquiries to the several agencies of the spread of infection which have been suggested in connexion with outbreaks of this disease abroad, notably in Sweden and in the United States, as well as to any local circumstances which seemed at all likely to be connected with the spread of the disease. The principal considerations which it seems desirable to put on record, for comparison with past and future epidemics, are set out below :—

### *Distribution.*

The bulk of the attacks occurred in the residents in the district; comparatively few visitors were affected. Although cases occurred in some of the larger villages or small towns, the greater number occurred in small villages or groups of houses, and often in isolated farm houses and cottages.

### *Sanitary conditions.*

Insanitary conditions of the dwelling or its neighbourhood appeared to have no special determining influence. Some of the houses invaded were kept scrupulously clean, and this remark applies to some of the smaller cottages. On the other hand certain of the infected houses were filthy in the extreme, and their occupiers were dirty in their persons and their habits. Several instances came under notice where there was overcrowding of persons in houses and particularly in bedrooms which were imperfectly ventilated, and in certain of these houses multiple attacks occurred.

When introduced into the small towns, as at Holsworthy and Bude where some of the earliest known cases occurred, the subsequent cases were few. On the other hand, after its introduction into certain rural areas, the disease manifested a much more definite tendency to spread. In certain of these cases there appeared possibility of infection having been acquired by personal contact, but there were other cases in which no contact could be traced. One invaded cottage for instance stood in a lonely position in the centre of a moor, approached by a simple cart track. The nearest cottages to this one were half a mile distant, and in these cottages no one had been ill.

### *Dust.*

It has been conjectured that the virus may in some way or other be associated with dust, and that the comparative freedom of urban

communities from epidemics is connected with paving and street-watering. It has also been suggested that the epidemic prevalence of the disease in late years is to be associated with the advent of the motor car, which has increased the exposure of the community to road dust, particularly in houses close to high roads. It cannot be denied that several of the cases coming under notice lived in houses which were situated by the side of a road, but there was no special exceptional spread of the disease when it was introduced into these houses. Only three cases occurred in houses abutting on the main road through Holsworthy, where there is a very large amount of agricultural and general traffic, also a considerable amount of motor traffic in the summer months. The dust nuisance is very pronounced, especially as the streets are never watered, and markedly so in the public square in the centre of the town. The railway station is in the town. On the other hand, several of the patients lived in isolated cottages far distant from any road likely to be traversed by any vehicle other than a farm cart, and is impracticable for motor cars : some of these cottages could only be reached on foot or on horseback owing to the absence of roads.

Dr. Gray furnishes the following particulars with regard to cases occurring in the Holsworthy Rural District :—

HOLSWORTHY RURAL DISTRICT.—Situation, &c., of houses in which cases of Poliomyelitis have occurred.

| No. of Case. | Address and Parish. | Situation. | Amount of traffic, Agricultural and Motor. | Distance from nearest Railway. |
|---|---|---|---|---|
| 1 | Clawton Lane, Clawton | Adjoining parish road ... ... ... | Some agricultural. No motor ... ... | 3½ miles. |
| 4,5, 6 & 7 | Windy Cross, Thornbury | Adjoining parish road and at junction of two cross roads. | Fair amount of agricultural. No motor ... | 4 miles. |
| 8 & 9 | Thornbury ... ... | Adjoining little used parish road ... ... | Very little agricultural. No motor ... | 5 miles. |
| 10 | Clawton Bridge... ... | Adjoining four cross roads, one of which is a main road. | A good deal of agricultural and some motor | 3 miles. |
| 11 | New England, Ashwater | In the middle of a moor of 150 acres, a quarter of a mile from any road. | None ... ... ... ... ... ... | Over a quarter of a mile. |
| 16–19 | Halsdon, Cookbury | In a farm, a quarter of a mile from any road | None ... ... ... ... ... ... | 2½ miles. |
| 20–23 | Halsdon, Cookbury | | | |
| 24 | Pyworthy ... ... | In a farm, 100 yards from parish road ... | Very little agricultural. No motor ... | 3 miles. |
| 25 | Pyworthy ... ... | Adjoining parish road ... ... ... | Some agricultural. No motor ... ... | 2 miles. |
| 26 | Pyworthy ... ... | In a farm, 300 yards from parish road ... | Practically none ... ... ... ... | 3½ miles. |
| 27 | Pyworthy ... ... | In a farm, 200 yards from parish road ... | Practically none ... ... ... ... | 3½ miles. |
| 28 | Pyworthy ... ... | Adjoining parish road ... ... ... | Some agricultural. No motor ... ... | 3 miles. |
| 31 | Thornbury ... ... | At junction of three parish roads ... ... | Fair amount of agricultural. No motor ... | 4 miles. |
| 32 | Thornbury ... ... | 50 yards from parish road ... ... ... | Some agricultural. No motor ... ... | 4 miles. |
| 38 | Milton Damerel ... | In a farm, 200 yards from parish road ... | Practically none ... ... ... ... | 5 miles. |
| 39 | Bradworthy ... ... | Adjoining parish road ... ... ... | Very little agricultural. No motor ... | 10 miles. |
| 44–46 | Putford ... ... ... | Adjoining parish road ... ... ... | Very little agricultural. No motor ... | 10½ miles. |
| 52 | Clawton ... ... ... | In a farm, 100 yards from parish road ... | Practically none ... ... ... ... | 4 miles. |
| 55 | Putford ... ... ... | In a farm, 50 yards from parish road ... | Practically none ... ... ... ... | 10½ miles. |
| 115 | Black Torrington ... | Adjoining main road ... ... ... | Some agricultural. No motor ... ... | 5 miles. |
| 121 | Cookbury ... ... | At junction of two parish roads ... ... | Very little agricultural. No motor ... | 4 miles. |
| 57 | Putford ... ... ... | 50 yards from parish road ... ... ... | Practically none ... ... ... ... | 10½ miles. |
| 58 | Milton Damerel ... | Adjoining parish road ... ... ... | Little agricultural. No motor ... ... | 5 miles. |
| 61–66 | Putford ... ... ... | In a farm adjoining parish road ... ... | Little agricultural. No motor ... ... | 10½ miles. |
| 101 | Halwill ... ... ... | In railway station yard, 20 yards from parish road. | Fair amount of agricultural and some motor | 50 yards. |
| * | Cookbury ... ... | 30 yards from parish road ... ... ... | Very little agricultural. No motor ... | 4 miles. |
| * | Putford ... ... ... | In a farm, 100 yards from parish road ... | Practically none ... ... .. ... | 10½ miles. |

* Not tabulated in the list of cases in this report.

*Insect bites.*

The one factor common to nearly all patients I had the opportunity of examining (and wherever possible my examination was extended to every person living in the infected house) was that the people were insect bitten. In one case that occurred in a farm house which was scrupulously clean, the mother of the patient plainly resented my questions as to possibility of the patient having been bitten by insects. On the other hand in a clean farm house where more than one case occurred, and where each of the inmates I examined was insect bitten, the farmer, a well-to-do man, ridiculed the idea that fleas were absent from any farm house in the countryside. I do not, however, suggest that all the insect bites I saw were due to fleas; in some houses there were other insects which bite man, in certain patients the bites were attributed to the "harvest bug," which was stated to be unduly numerous in this district during the present summer, or to biting flies. Manure nuisances were observed in the neighbourhood of several of the invaded houses, and here a history of prevalence of biting flies, not necessarily of the mosquito or midge type, was obtained. So far as my inquiries went, it cannot be said that there was evidence pointing to any special biting insect as the carrier of infection, but from the circumstances of the case little exact information was to be had. As regards fleas, it is to be borne in mind that recent researches have shown that fleas can live for many weeks without food, and that if fleas can in fact act as carriers of the infection of poliomyelitis, infection might thus be conveyed for considerable distances by means of clothing. I was informed that there is a regular trade in Holsworthy of old clothes which are sent there for sale from Houndsditch in London, and Dr. Gray, the medical officer of health for Holsworthy urban and rural districts, made special inquiry concerning this trade. He was, however, unable to elicit any fact showing that any of these clothes had been purchased by the inmates of the invaded households in his districts, with one exception which is referred to below.

*Transmission of Infection by Clothes.*

This possibility called for consideration in the following case:—

The mother of the patients Nos. 16–19 bought a second-hand skirt and blouse in Holsworthy market for No. 16, the fatal case, a few days before her illness commenced. The child who wore these clothes was not attacked by the disease until three days after her younger brother became ill. These cases, however, did not occur until July and there had been earlier cases in the district. Moreover, the first known case in North Devon occurred in the town of Holsworthy on 3rd May (Case No. 2), and in this case no association with second-hand clothes could be established.

*House Flies.*

Dr. Gray has furnished me with the following note on the prevalence of house flies during the past summer in the Holsworthy Union:—

In this district in common, probably, with other parts of England, there

was a veritable plague of flies (house) in June, July, August, and most of September.

On all hands it is acknowledged that they have never proved to be so numerous and troublesome as they were during this past summer.

Despite this fact cases of diarrhœa, enteritis, &c., have been far fewer than usual.

I have made inquiries of the two chemists who trade in this district as to the sale of fly-papers, &c.

One says that he sold immense quantities of fly-papers, bands, &c., and was not able to supply the public fast enough, and that the manufacturers were unable to cope with his orders. The other chemist says that he manufactures the fly-papers himself, and states that he made this summer fifteen times the quantity he made last year, and ten times more than the average of the last 16 years. He keeps a yearly record of the quantity made.—W. G. G.

### *Relation to Death and Sickness in Animals.*

Dr. Gray has furnished the following notes in connection with one of the two first known cases in Devon (Case No. 1) as regards the occurrence of deaths of animals about the time of the invasion of the household by poliomyelitis—

Two pigs belonging to the family of Case No. 1 died, quite suddenly, four days after the child's death. Could not attribute the deaths to any particular cause.

At the time the child was lying ill, a neighbour who resides in a cottage about 50 yards from the house of Case No. 1 lost four fowls and nine chickens (about two months old) in two days. She states "that they were "all taken in their heads producing a peculiar nodding motion, they would "die within 1–2 hours after being taken off their legs; they would lie on "their backs with their heads thrown back; she had never known chickens "die like this before; she buried them all."

Another neighbour who lives next door to Case No. 1 also lost one fowl and 15 chickens (three weeks old) at the same time, she also states "that "they were taken in their heads with the same curious motion of the head, "they would run to drink and then suddenly fall on their backs with their "heads bent back and would soon be dead."

Both women say that the average course of the complaint was from 6–10 hours in duration and that they considered that the cause of death was the heat, but that "they had never known anything like it (*i.e.*, mortality amongst chickens) before."—W. G. G.

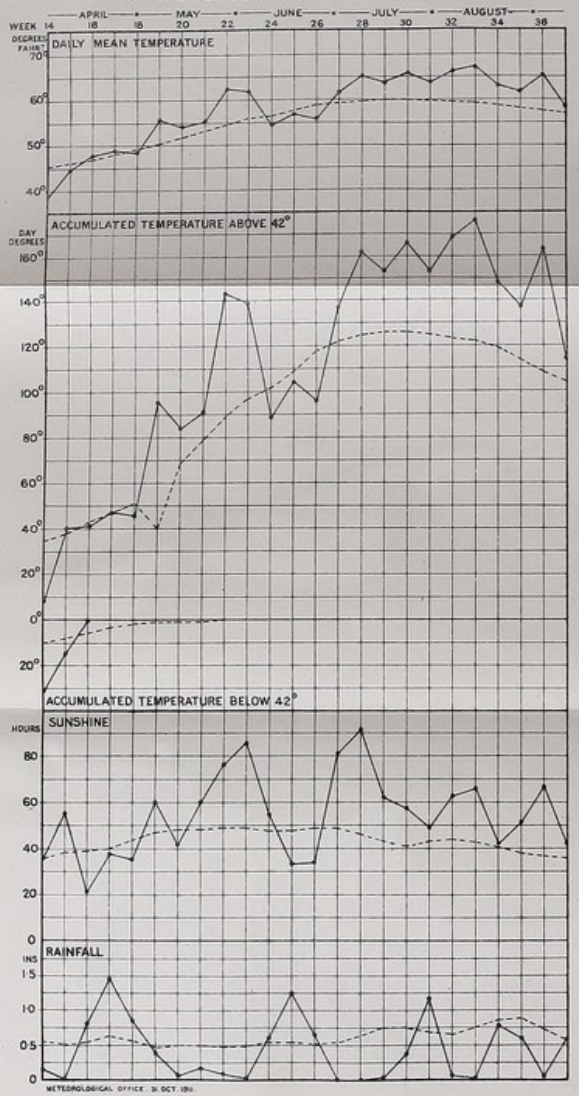
In two other instances young fowls were reported to have died a few days antecedent to the onset of disease in a household, and at one small farm, where three cases occurred, one proving fatal, the fowls were noticed to be deficient in plumage at a time when they would not be moulting; none of the fowls here had died. But fowls are commonly kept at the small farms and cottages in these counties; and it was easy to find instances where there had been disease and death among fowls and no known case of poliomyelitis in the human subject in the district. Again, at many of the invaded houses no fowls were kept.

No history of illness among dogs, cats, rats or mice could be ascertained at any of the invaded houses. There has been no swine fever in this district for over two years.

A young bull 16–17 months old, and a calf died on the farm where the farmer (No. 21) and three of his children (Nos. 20, 22 and 23) suffered attack (see pp. 38–9) and where also one of his farm labourers had four children ill (Nos. 16–19) and another of his farm labourers

## WEEKLY VALUES OF CLIMATOLOGICAL ELEMENTS FOR ENGLAND S.W. INCLUDING SOUTH WALES. [DISTRICT 8].

FOR THE PERIOD FROM APRIL 2 TO SEPT. 16, 1911.

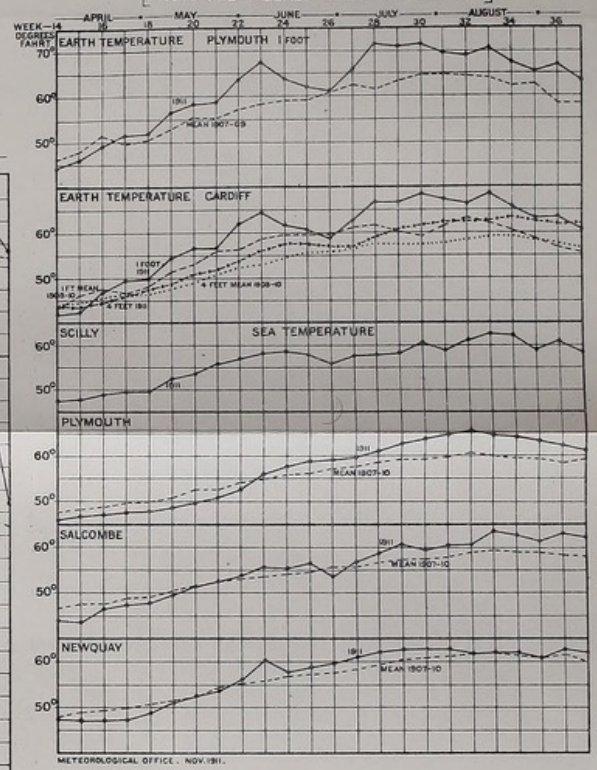



THE FULL LINES SHOW THE VALUES FOR THE CURRENT YEAR, 1911. THE DOTTED LINES SHOW THE AVERAGES FOR THE 25 YEARS 1881 – 1905.

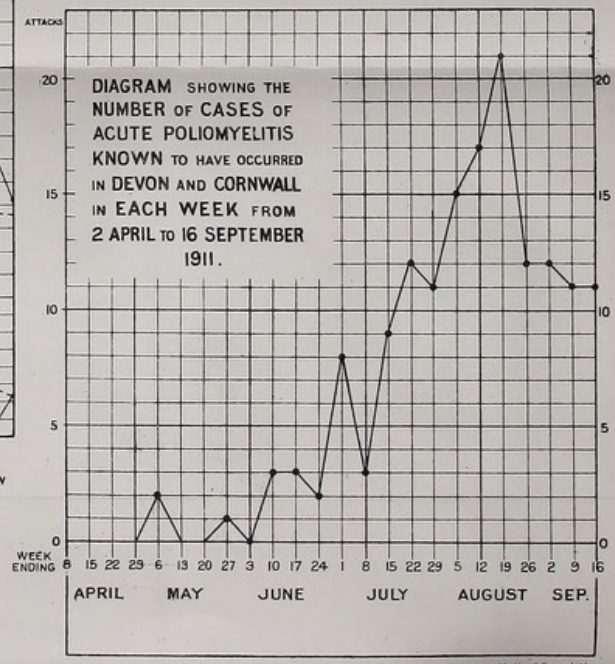
## EARTH TEMPERATURE AND SEA TEMPERATURE – ENGLAND SOUTH WEST

APRIL 2 TO SEPTEMBER 16, 1911.

[FROM THE WEEKLY WEATHER REPORT]




## DIAGRAM SHOWING THE NUMBER OF CASES OF ACUTE POLIOMYELITIS KNOWN TO HAVE OCCURRED IN DEVON AND CORNWALL IN EACH WEEK FROM 2 APRIL TO 16 SEPTEMBER 1911.




Malby & Sons Lith.

had a child (No. 24) which suffered from paresis of both legs. The bull was found ill one morning "as if it had sunstroke," and it died in two hours. This was a week before the first case of illness occurred among the people living on the farm. About five days after the bull died a calf 8–9 months old was found dead one morning with its head drawn back. No further particulars could however be obtained.

In Case 144, the medical attendant, Dr. Hopper, informs me that a horse belonging to the patient's father suffered from what is locally termed "poke neck." It is said to have been paralysed in the neck and forequarters. It fell down in the stable and was unable to rise; it was taken with assistance to a field, where it again fell down. It was shot without having been seen by a veterinary surgeon. This happened a week before the child was taken ill. It is of interest to note that this child's illness is the earliest of those of which I have records in Cornwall.

### *Influence of Season.*

Owing to the configuration of the counties of Devon and Cornwall the meteorological conditions vary within considerable limits at the same time in different parts of the counties.*

Dr. W. N. Shaw, F.R.S., Director of H.M. Meteorological Office, has kindly prepared the accompanying diagram in which the meteorological data are equated for the South-Western division of England. The facts recorded must only be regarded as showing in a general fashion the climatic conditions which obtained. To this diagram I have added the seasonal curve of the known cases of the disease.

The seasonal curve of the incidence of disease shows week by week the number of known cases from the period in May when the first cases came under notice until the week ending 16th September. It will be seen that the greatest number of known cases in any one week in the period under review occurred in mid-August. Speaking generally it can be said that the curve of known cases was rising while the temperatures of air, earth and water were in the ascendant, and that the decline in poliomyelitis coincided with, or closely followed, the declensions of the earth and sea temperatures at the places given. The diagrams also show that during the present year the temperature curves are all higher than the mean of past years.

In the northern hemisphere poliomyelitis has shown its greatest prevalence in the warm season of the year, and this distribution of season prevalence was observed in the outbreak under consideration.

## Incidence of Cases in Houses and Attack-rates at Ages.

Of the 154 cases occurring in 129 houses of which I have records there are 33 concerning which I have no detailed information of the number of inmates in the various households; the remaining

* Detailed information is furnished in the published reports of H.M. Meteorological Office.

121 cases occurred in 97 households. From these cases the following figures are taken :—

| No. of invaded households. | No. of persons living per invaded house. | No. of persons living in these invaded houses. | No. of multiple attacks in these houses. |
|---|---|---|---|
| 9 | 3 | 27 | |
| 14 | 4 | 56 | 2 cases in one house. |
| 22 | 5 | 110 | 4 cases in one house. |
| 15 | 6 | 90 | |
| 18 | 7 | 126 | 4 cases in one house, 3 cases each in two houses and 2 cases in another. |
| 7 | 8 | 56 | 2 cases in one house. |
| 3 | 9 | 27 | |
| 3 | 10 | 30 | 4 cases in one house.<br>6 cases in one house. |
| 2 | 11 | 22 | 4 cases in one house. |
| 3 | 12 | 36 | |
| 1 | 13 | 13 | |
| 97 | | 593 | |

The percentage of attack on the population of the invaded houses is therefore 20·4 per cent.

It has not been possible to get the exact ages of each of the inmates of the invaded houses in some forty-five cases ; but for the remaining 84 households the following information has been obtained :—

| Ages. | Number of persons at that age. | Number of persons at that age who suffered attack. | Percentage attacked. | |
|---|---|---|---|---|
| Under 1 | 25 | 10 | 40·0 | |
| 2 | 15 | 7 | 46·7 | |
| 3 | 32 | 12 | 37·5 | 44·0 |
| 4 | 26 | 14 | 53·8 | |
| 5 | 18 | 8 | 44·4 | |
| 6 | 26 | 7 | 26·9 | |
| 7 | 17 | 7 | 41·2 | |
| 8 | 21 | 7 | 33 3 | 36·8 |
| 9 | 15 | 9 | 60·0 | |
| 10 | 16 | 5 | 31·2 | |
| 11 | 8 | 4 | 50·0 | |
| 12 | 14 | 1 | 7·1 | |
| 13 | 11 | 2 | 18·2 | 19·2 |
| 14 | 7 | 2 | 28·1 | |
| 15 | 12 | 1 | 8·3 | |
| 15–20 | 22 | 1 | 4·5 | 4·8 |
| 20–25 | 41 | 2 | 4·9 | |
| 25–30 | 41 | 1 | 2·4 | |
| 30–50 | 114 | 3 | 2·6 | |
| 50 and over | 24 | 0 | 0·0 | |
| | 505 | 103 | 20·4 | |

The figures are few, and it would be unsafe to draw any but the broadest inferences from them. But so far as they go they tend to show that the liability to attack steadily decreases with advancing age; there being a progressive decrease in the percentage attack at age groups 0–5, 5–10 and 10–15 years. The incidence on the age group 10–15 years is four times greater than that on the 15–25 years group, which in turn is twice as great as that in the 25–30 and the 30–50 years groups.

In fourteen instances the child attacked was the only child in the house.

*Groups of Cases in Houses.*

It will have been noted that although by far the greater number of cases occurred as single cases in a household, multiple cases occurring in families were met with. Thus in four instances there were two cases*, in two there were three cases, in four there were four cases and in one instance there were six cases in a house.†

*Two cases in one house.*—Nos. 8 and 9. In this instance the two patients were a male aged 2½ and a female aged 5, the elder child being attacked some ten days after the younger. Each child suffered from headache and vomiting, with loss of patellar reflex, but only the younger showed any signs of paralysis—a partial loss of power in the legs. He also had retraction of the head. The only other inmates of the house were the father and mother aged 32 and 26 respectively. They lived in a small cottage and all slept in the same bedroom.

In another instance, Nos. 12 and 13, the first attacked was a female child aged two years, followed five days later by a male infant aged nine months. The elder child's illness was marked by high temperature, 104° F., headache, vomiting, irritability of temper, drowsiness, exaggerated patellar reflex and loss of power in both legs. The baby's attack was indicated by a temperature of 102° and extreme drowsiness; no paralysis of any muscles were noticed. There were indications to show that this infant had headache.

In the third, in Nos. 78 and 79, the patients were brothers aged two and a half and three and a half years respectively. They were both attacked on the same day, the onset of illness being sudden in character, marked by headache, vomiting, Kernig's sign, tache cérébrale, and drowsiness. The latter two signs were most marked in the younger child. This child had paralysis of the muscles of the neck and could not hold his head up. The elder child had at first muscular rigidity of both legs, which passed to paralysis nine days after the commencement of the illness, and in this case there was no marked retraction of the head. No other inmates of the house were affected; the house was clean. There was no known case of similar illness in the neighbourhood. The father, however, in connexion with his business had been visiting houses in Bude and Holsworthy district.

In the remaining instance the patients were sisters aged 5 years and 3 years and 8 months. The younger was attacked two days after the elder.

*Three cases in one house.*—Nos. 48, 49, and 50. In this instance a boy aged 12 was attacked by headache and vomiting. He suffered from pain in all his limbs, and was so drowsy that he could not be kept awake, and he had general muscular weakness. Three days after his attack, a brother aged 9 and a sister aged 6 became ill. They had headache; the boy vomited, the girl did not. The boy had no loss of reflex a month after attack, the

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* One of these instances of two cases in a house could not be included in the Table on the previous page owing to want of detailed information.

† The notes have reference only to the 154 cases which appear in the Table at the end of this report. The school outbreak, p. 42, is quite distinct and none of the cases dealt with in connexion with it are included in the 154 mentioned above: in it multiple cases also occurred in several houses.

girl had loss of patellar reflex on the left side. The adult members of this family were not attacked.

In the other instance, Nos. 44, 45 and 46, a baby aged 1½ years was first attacked. The child was drowsy, cried out at night while asleep. This illness lasted three days, when the child recovered. The day after the baby became ill, its brother, aged 3½ years, was attacked by acute headache and vomiting with irritability of temper. He seemed very ill for some 36 hours, when he recovered.

Neither of these children were attended by a medical man.

Six days after the baby was attacked its sister, aged 6 years, was attacked by headache, vomiting, twitching of the muscles, particularly of the muscles of the jaw. Her temperature when taken on the day after the onset was 101·6° F., pulse 150, respiration 28. There was marked irritability of temper and the child screamed when moved. Kernig's sign was present. Tache cérébrale was absent, and there was no retraction of the head and no rash. Paralysis of both arms occurred, followed by paralysis of the intercostal muscles and laryngeal muscles, and the child died the third day after its attack.

In this case lumbar puncture was performed shortly before death.

*Four cases in one house.*—Nos. 4–7. In this instance two children (twins) aged 10 months, were first attacked, and at the same time. Both seemed acutely ill for three days, and one suffered loss of power in the right arm, which persisted for at least a fortnight, when the child recovered the use of the limb. A week later a sister, aged 4 years, was attacked by headache and vomiting. There was no loss of muscular power, but the right patellar reflex was absent. Two weeks later a brother, aged 7, was attacked by acute headache, and his right patellar reflex was absent. There was no paralysis in this case.

The family consisted of a father and mother and nine children, eldest aged 13, seven of whom remained in good health. They lived in a four-roomed cottage, using three rooms as bedrooms. The house was dirty and its surroundings insanitary.

Nos. 16–19 live in a farm labourer's cottage. The first attack occurred on 13th July in a boy aged 9 (No. 17). He suffered from headache and vomiting. Kernig's sign was only slightly in evidence ; patellar reflex was absent. He had paresis of both lower limbs and walked with difficulty. The day after the boy was attacked (14th July) a sister, aged 7 (No. 19), suffered from headache, vomiting, and pain in the neck. The patellar reflex on the left side was diminished. There was no marked paresis, only general weakness. There were enlarged posterior cervical glands, but the child's hair was verminous. Neither of these children was attended by a medical man. Three days after the first case was attacked, *i.e.*, on 16th July, a sister, aged 5 (No. 16), suffered from headache, vomiting, and twitching of the muscles ; she had retraction of the head and neck, and died on the 18th July, the second day of the illness. This case was seen by a medical practitioner before death, and the death certificate is signed "Pneumonia." Nurse M. came to nurse this case and found the child had just died.

Ten days after this child died, *i.e.*, on 28th July, her sister, aged 13 (No. 18), suffered from headache and lost power in her right deltoid muscle. On the following day the patellar reflexes were absent, and the child felt very ill and faint. A fortnight after the beginning of her illness, while walking along a road, she put her right hand into her pocket and suddenly felt a sharp pain. She withdrew her hand with difficulty, and found the fingers drawn up and a feeling as if she had got several stones in her hand. She can half flex her forearm, cannot raise her upper arm to a right angle with the body, the fingers are flexed and contracted, she is unable to extend them, she can extend the thumb but not flex it. There is complete loss of sensation over the lower third of the flexor surface of the forearm and also over one-half of the extensor surface.

The whole of this child's body, but the right arm and trunk in particular, was covered to an extraordinary degree with a petechial rash evidently the result of insect bites.

The house and the people were filthily dirty.

Nos. 20–23. Six days after case 17, mentioned above, had occurred a labourer's cottage on the farm, *i.e.*, on 19th July, the farmer's son, aged 2

years and 4 months (No. 22), was feverish, very drowsy, and apparently suffered from headache: he recovered in two days. On the 21st, the farmer himself (No. 21), aged 42, suffered from headache, vomiting and constipation. Drowsiness was a marked symptom; he went to bed and practically slept for three days. He felt ill and weak for several days after. On the day of the farmer's attack, a daughter aged 8 (No. 20), complained of headache; her temperature was 101° Fah. She did not vomit, but was extremely drowsy. When seen a week later Kernig's sign was present, patellar reflexes absent. On 25th July, she had loss of power in the left leg below the knee, and suffered from retention of the urine for 24 hours; she suffered from constipation. She had no retraction of the head and no rash. On the 26th July, another daughter, aged 10 (No. 23), suffered from pain in the neck. She had no headache, nor did she vomit. She was very drowsy and could not keep awake. The patellar reflex was diminished but Kernig's sign was absent; there was no retraction of the head. When awake the child had attacks of weeping.

This household consisted of the farmer, his wife, seven children, eldest aged 10, and a governess. The governess had recently had "a bad cold," and the eldest child had no patellar reflex. Otherwise there had been no illness in the house. The farmhouse was very clean, but all the inmates were insect bitten. With regard to this matter the farmer said it would be impossible to find a farmhouse in Devonshire which did not contain fleas.

Associated with these cases is No. 24, which occurred on 22nd July, in another farm labourer's cottage on this farm. A child, aged 3, was attached by headache and vomiting, loss of patellar reflex, and paresis of both lower limbs. There was no retraction of the head and no rash. The other inmates of this cottage, the father, mother, and infant 7 weeks old and a female aged 29, were not attacked.

The fourth instance of four cases in one house in given on p. 40.

*Six cases in one house.* Nos. 61–66.—These six cases occurred in a family of ten persons living in a cottage situated near to the farmhouse where cases Nos. 44–46 occurred which have been previously mentioned (see p. 38).

On 14th August (the day after the death of No. 44), two children, a boy aged 5 (No. 63), and a girl aged 3 (No. 64), were "feverish" and suffered from severe headaches and drowsiness. Kernig's sign was absent. The boy had a slight sore throat; both children had loss of patellar reflex.

On 16th August, two more children, a girl aged 7 (No. 61) and a boy aged 10 (No. 66), were attacked by similar symptoms, but their drowsiness was less marked. On 17th August, a boy aged 13 (No. 65) was similarly affected. On 18th, a girl aged 14 (No. 62) was attacked in like fashion; she also vomited, had a sore throat, very furred tongue, and discharge from the nose. On 23rd August, she was so weak that she could scarcely stand and sweated profusely over the face and head on the slightest movement.

Kernig's sign was not present in any of these cases. All were insect bitten. Two other children, aged respectively 12 years and one year, and their parents, were not attacked. They live in a three-roomed cottage, which is grossly overcrowded, and the house is dirty.

These instances of multiple cases in houses, some occurring in series, suggest (though of course they do not prove) that person to person infection was operative, sometimes with much intensity.* They may be contrasted in this respect with the failure to induce natural infection from animal to animal in experimentally infected monkeys in the laboratories of Levaditi in Paris, and the Rockfeller Institution in America.

Further suggestion of communicability, whether directly from person to person by means of infectious discharges, or intermediately through biting insects were afforded in several of the instances which follow.

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* *See also* the account of the illness of the scholars, &c., at Stoke Rivers, p. 42.

*Possible transportation of infection by an apparently healthy carrier.*

*Case No. 33.* A boy aged 6, the only child in the family, suffered an unquestionable attack of poliomyelitis. The onset of his illness occurred on 24th July. He lived at Taylor's Cross in the Stratton rural district (*see* map). His father, "B.," a small farmer, sold a horse* to "W.," and according to the custom of the country he had to deliver the horse at the place of the purchaser. This he himself did, and he stayed for an afternoon meal with the "W." family. The place to which this horse was delivered is an isolated small farm house to the north of Hartland. It is only approached by narrow lanes, and finally reached by crossing two fields; it is distant in a direct line some 10 miles from Taylor's Cross. There had been previously no known cases of poliomyelitis in this particular neighbourhood, and the "W." family had had no illness, nor had any persons (other than "B.") visited them.

At meal time "B." sat between cases No. 40 and No. 41, two boys aged respectively 5½ and 2½ years. His visit was made on the afternoon of 2nd August while his son was still ill at home.

During the night of 8th August both No. 40 and No. 41 suffered an acute attack of illness and medical assistance was summoned on 9th August. No. 40 is said to have been ailing, with headache for a few days previously, and No. 41 was "lying about" for a day before the onset of acute symptoms. The symptoms were acute headache and vomiting; there was no marked retraction of the head, Kernig's sign was present, tache cérébrale well marked, patellar reflexes were absent; neither child was able to stand through loss of power in the legs. Their illness was marked by irritability of temper, drowsiness and constipation. Case No. 40 had some muscular twitchings and a severe attack of epistaxis on the night of 9th August.

On 15th August lumbar puncture was performed on case 40, and the fluid forwarded to Dr. Gordon for examination. At this date there was marked retraction of the head and of the spine, rendering the operation of lumbar puncture a difficult one. The younger boy was much better and able to sit up in bed.

On 11th of August their mother (case 123) suffered a mild attack, her symptoms being headache, pain in back of head, drowsiness, sore throat, constipation; tache cérébrale was well marked; she recovered in three days. On 12th August an infant aged eight months (case 124) was attacked. Temp. 101° Fah., vomiting, irritability of temper, tache cérébrale and Kernig's sign present, slight retraction of head and constipation; the illness only lasted 36 hours. The other two inmates of the household, the father aged 27 and the grandmother aged 73, were not attacked.

The following case is also possibly one in which the infection was transported by a healthy person. There had been previous cases not very far removed from the house in question, but inquiry failed to elicit that there had been communication between these previous cases and the household in question.

On July 18th Nurse M. went to attend case No. 16; on arrival she found the child dead; she washed the body and laid it out. On

* No evidence was obtained to indicate that this horse had suffered from any illness.

her return home she took a lysol bath and changed all her clothes. She did this on her own initiative; though she was unaware of the cause of death of No. 16 (it was one of the early cases), she thought the circumstances unusual. She did not spray her nose and throat with a disinfectant. Subsequently she attended case 31 in her confinement, which took place on 22nd July (see p. 20).

In the following instance there was communication with an infected house by two persons who remained healthy, though an infant in the house in which they resided subsequently became ill and died.

A farm labourer aged 36, died on 10th August — the particulars of this case are given under case 38.* This man's daughter, aged 11, lived at the house of the local blacksmith, and she saw her father the day before his death. Difficulty was experienced with regard to the burial of the dead man; the villagers fearing to assist. The blacksmith however rendered such help as he could and he also carried the coffin to the grave. On 17th August the blacksmith's only child was attacked by acute poliomyelitis, and died on 26th August. (Case 58.)

The possibility of infection having been acquired by attendance at large public gatherings was kept in view. In the summer season many local fairs are held in this part of the country, and although their possible influence in furthering the spread of infection could not be excluded, nevertheless no definite information was obtained tending to show that they had played any important part in the dissemination of the disease, with the possible exception of St. Peter's Fair. The evidence implicatory of this fair is not conclusive. I am indebted to Dr. Gray for the following particulars:—

> St. Peter's Fair is a very ancient Chartered Fair, and it is held annually in the second week in July at Holsworthy and lasts for five days: this year, 1911, the date of the fair was from July 11th–15th (both inclusive). The first day of the fair is devoted to business, and very large numbers of horses, cattle, and sheep are brought into the town for sale. The other days are given up to pleasure. The fair comprises a large number of "shows," "roundabouts," &c., including a cinematograph show. All are held under canvas, and many thousands of people flock into the town from many miles round. In every instance, save one, where a case of poliomyelitis occurred in Holsworthy and in the rural districts immediately round the town, one or more of the inmates of the invaded house visited the fair, and nearly all visited the cinematograph "show," which was at all hours of the day filled to overflowing. The atmosphere of the tent in which this "show" was held became very impure. I have made personal enquiries in each case as to the incidence on persons visiting the fair, and I find on examining the list of poliomyelitis cases in this district, that out of 21 cases reported during the month of July, 16 of them were taken ill during or soon after the fair.—W. G. G.

In connexion with the possible association of the disease with infection in lower animals, regard was had to the occupation of the householders of the invaded families. In 82 in which the occupation is stated 34 were farmers or farm labourers, while more were daily brought into relation with animals. The occupations of certain

* His death was certified by the medical attendant as due to "cerebro-spinal meningitis."

others would not necessarily bring them into association with animals.*

## Illness at Stoke Rivers in relation to School attendance.

Dr. Ivar Wickman has directed special attention to certain cases in which he considered that school attendance had favoured the spread of poliomyelitis in Sweden.

This outbreak in Devon and Cornwall first came under review at the end of the school summer term, and although inquiry was made as to school attendance having played a part in the dissemination of the disease no facts were elicited to bring the schools under suspicion. The greater number of the cases reported on occurred while the schools were closed for the summer holidays.

The matter however was kept in view, and particular inquiry made in regard of each case coming to notice after the schools were re-opened.

In Devonshire, in all cases where a child attending school had been attacked during the holidays, the school was not re-opened until the County Medical Officer of Health had examined the children, and had satisfied himself that cases of the disease had not been overlooked. This was not attempted in Cornwall.

On 27th September, Dr. Harper, the Medical Officer of Health for the Barnstaple Rural District sent me a short report on an outbreak of illness which he considered due to a mild form of poliomyelitis among the children attending the Stoke Rivers School in his district. The cases had not, owing he states to their indefinite symptoms, been notified to him as Medical Officer of Health. The outbreak was exhaustively investigated by Dr. Adkins, the County Medical Officer of Health, and to these two officers I am indebted for the following particulars.

Stoke Rivers is a small village about seven miles distant from the town of Barnstaple. The school is but a small one, there being only 41 children on the register. Not all these children were attending school, one or two being absent from the village.

There are 18 houses having a population of 119 persons. Of these persons, 61 are over the school attendance age and were not attending the school; 43 were attending school, although 2 were not attending the Stoke Rivers School; 15 were below the school attendance age. In this community there were 36 attacks which were more or less definite, and some 9 others may possibly have suffered in minor degree. Of these 36 cases, 4 were over, and 6 under the school attendance age, and 26 were attending school. Neither of the 2 scholars mentioned above as attending some other school suffered attack.

A diagram illustrating the facts elicited as to residence, age, school attendance, attacks, and dates of onset, has been prepared.

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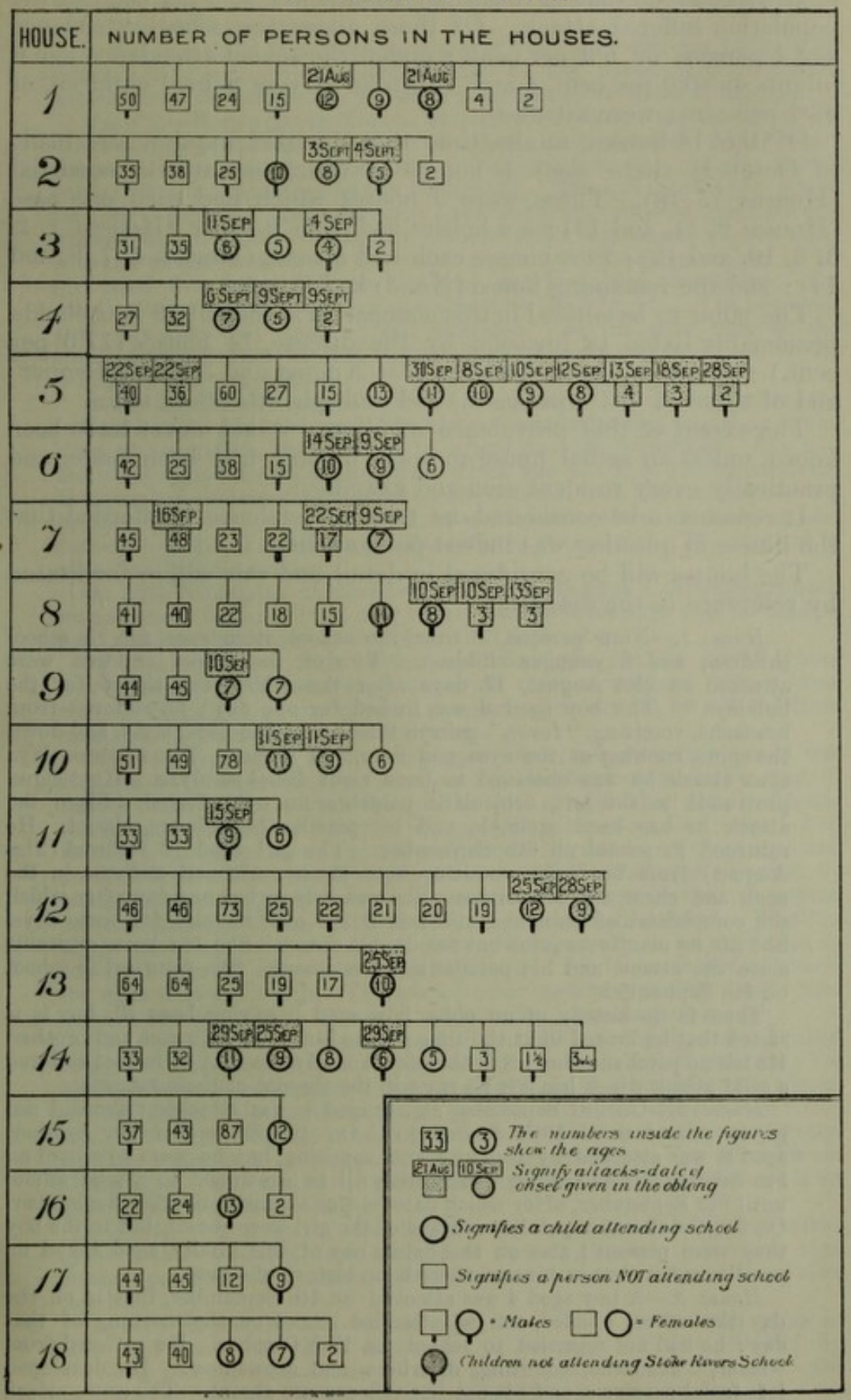
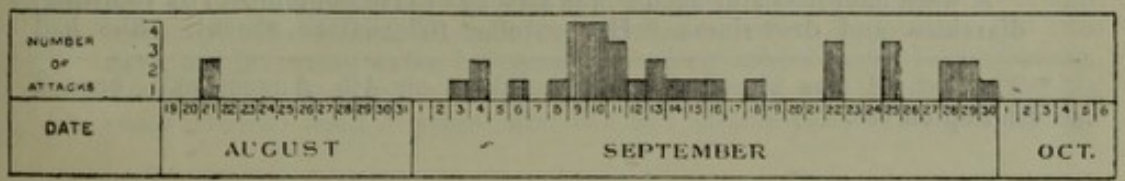
* *Occupations.*—Grocer (3), labourer (3); mariner, mason, blacksmith, roadman (2 each); and "No occupation" in 4 cases. The following had one each:—ironmonger, brushmaker, "armorer," shop assistant, photographer, plumber, baker, publican, milk dealer, oil merchant, optician, carter, rabbit trapper, game dealer, gardener, gamekeeper, police constable, scavenger, schoolmaster, yachtsman, postman, railway ganger, doctor, Indian civil servant, colt breaker, nurse, farm steward, club steward, laundry worker.

# STOKE RIVERS




## DIAGRAM SHOWING NUMBER OF ATTACKS DAY BY DAY.



It will be seen that in this small community 30·3 per cent. of the population suffered attack. Of those above the school attendance age 4 persons or 6·6 per cent.; under the school attendance age 6 infants or 40·0 per cent.; and 26 Stoke Rivers School children, or 63·4 per cent., were attacked.

Of the 18 houses, at the time that detailed inquiry was made (6 October), there were 4 houses in which no attacks occurred (Houses 15–18). There were 3 houses which had each one case (Houses 9, 11, and 13); six houses had each 2 cases (Houses 1, 2, 3, 6, 10, and 12); four houses each had 3 cases (Houses 4, 7, 8, and 14); and the remaining house (No. 5) had 9 cases.

The point to be noticed in this connection is that in 18 households presumably liable to invasion by the disease, 14 houses (77·0 per cent.) suffered invasion between 21st August and 30th September; and of these 11 (61·1 per cent.) had multiple attacks in them.

The extent of this prevalence of illness would never have been known unless an actual house to house inquiry had been made, and practically every resident seen and examined.

It remains to be considered as to what evidence is afforded that the illness in question was indeed poliomyelitis.

The houses will be considered in detail, and this will be facilitated by reference to the diagram.

*House 1.*—Nine persons, 4 over the school attendance age, 3 school children, and 2 younger children. Two of the school children were attacked on 21st August, 17 days after the school was closed for the holidays.* The boy aged 8 was in bed for one day; he suffered from headache, vomiting, "fever," pain in the back of the neck, arms, and down the spine, running at the eyes and nose, and herpes. Two or three days after attack he was observed to have right facial paralysis. His tongue protruded to the left, orbicularis palpebrarum not affected. Since the attack he has been excitable and his patellar reflexes are absent. He returned to school on 6th September. The girl aged 12 suffered (21st August) from headache, vomiting, sore throat, pain and stiffness in the neck and chest, and drowsiness. She was only in bed one day after which she complained of pain and stiffness in the calf muscles for some days. She has no manifest squint but has double vision. She has been excitable since the attack, and her patellar reflex is absent. She returned to school on 4th September.

There is no history of an elder boy aged 9 having been ill, but it is stated that he looked ill at the time of the illness of his sister and brother. He has no patellar reflex: it is therefore conceivable that he in fact suffered a mild attack which has left its trace in the absence of these reflexes.

*House 2.*—On 3rd September a girl, aged 8, was attacked; she did not go to school until 18th September. On 4th September her brother, aged 5, was attacked by headache and vomiting, but he went to school on 5th September, and though manifestly ill he continued to attend school until 8th September, after which date he was absent until 26th September. On 6th October the patellar reflexes of the girl were absent, but in the boy they were present; also on this date a boy of this family, aged 2, had no patellar reflex, but in his case there is no history of illness.

*House 3.*—A boy aged 4 was attacked on 4th September, that is on the day the school opened, and he attended school on the morning of that day: he had not returned to school on 17th October. He suffered from "fever," headache, vomiting, diarrhœa, and drowsiness. He developed left facial paralysis, and his patellar reflexes are absent. The acute attack passed off in two days, but he remained weak and ill.

A week later his sister aged 6 was attacked (11th September) by vomiting, diarrhœa and drowsiness. Her patellar reflexes are absent. She had

* The School was closed for the holidays on 4th August and opened on 4th September.

attended school from its re-opening; after her attack she returned to school on 18th September.

In this household is a younger boy aged two years who has no patellar reflex. It is conceivable therefore that he has passed through a mild attack which escaped notice, but there is no history of illness in his case.

*House 4.*—Here a girl aged seven who attended school was attacked on the 6th September; she returned to the school on 11th September. A sister aged five who was attending school and a boy aged two who did not attend school were both attacked on 9th September. This girl returned to school on 13th September. The girl aged 7 suffered from vomiting, giddiness and drowsiness. On 6th October her patellar reflex was absent on the right side and exaggerated on the left; she complained of her legs aching. The girl aged five suffered from vomiting, diarrhœa and drowsiness. On 6th October the patellar reflex was absent on the left side. The boy aged two only exhibited vomiting and diarrhœa. At the same time as the children were ill their mother had an attack of acute diarrhœa; she is not subject to this complaint.

*House 5.*—The first attack in this house occurred on 8th September, the patient being a girl aged ten. She had not been attending school up to the time of her attack, but returned to school on 11th September. She had the usual symptoms of a mild attack which was not followed by paralysis. Her patellar reflexes were present on 6th October. On 10th September her brother aged nine who had been attending school was attacked in like fashion; on 6th October his patellar reflexes were absent. He returned to school on 18th September.

On 12th September another boy of this family aged 8, attending school, was attacked but his illness only lasted two or three days and he made a good recovery, returning to school on 18th September.

On 13th September their brother aged four who did not attend school was attacked, his illness being somewhat more severe and lasting three weeks. Kernig's sign was present, patellar reflex absent, he suffered from retention of urine and paresis of the legs.

On 18th September another boy aged three, not attending school was attacked by sickness and diarrhœa; on 22nd September the father aged 40 had an attack of acute headache and the mother aged 36 an attack of acute vomiting; on 28th September a boy aged two, not attending school, had an attack of convulsions and suffered from diarrhœa and vomiting: and on 30th September a boy aged 11, who attended school was attacked by headache, vomiting, drowsiness and sore throat. Kernig's sign was present on 6th October, but the patellar reflexes were not diminished. Whether these cases were mild attacks of poliomyelitis, or are to be attributed to some other cause cannot be definitely stated: the facts are put on record for what they are worth. There were other members of this family who had not had any similar illness by 6th October. See diagram.

*House 6.*—Two boys, aged 9 and 10, attending school, were attacked respectively on 9th and 14th September by vomiting and drowsiness; the former also suffered from diarrhœa, and his right patellar reflex was absent on 6th October. On this date the left patellar reflex was absent in the latter case. The boys returned to school on 13th and 18th September respectively. In this house is a girl, aged 5, who was operated on for adenoids on 31st August. She had no patellar reflex on 8th October, but there is no history of illness in her case.

*House 7.*—A girl, aged 7, attending school, was attacked on 9th September by headache, vomiting, drowsiness, sore throat, diarrhœa. Kernig's sign present, patellar reflex absent. She had not returned to school on 17th October.

On 16th September her mother aged 48 suffered from acute pains in neck and back. On 22nd September a lad aged 17 was attacked by vomiting and diarrhœa; Kernig's sign present, patellar reflex absent. On 6th October he was still in bed and had difficulty in standing.

*House 8.*—On September 10th a boy, aged 8, who was attending school, was attacked by stiffness in the neck and legs, diarrhœa, drowsiness, and loss of patellar reflex. He returned to school on 18th September for two days, but becoming worse he ceased attending school until 16th October. Also, on 10th September his younger brother, and on 13th September his sister (twins) aged 3 years, who did not attend school, suffered similar

attacks in a modified degree. An elder brother, who attended another school, was not attacked.

*House 9.*—On Sunday, 10th September, a boy aged seven, attending school, was attacked by vomiting and drowsiness. His patellar reflexes were nearly absent on 6th October. He did not cease to attend school.

*House 10.*—On 11th September, two girls aged 11 and nine, attending school, were attacked by headache, vomiting, diarrhœa, sore throat and drowsiness. On 6th October the patellar reflexes were present in both children. They were not absent from school.

*House 11.*—On 15th September, a boy aged nine, attending school, was attacked by headache and giddiness. On 6th October his patellar reflexes were present. He continued to attend school.

*House 12.*—On 25th September, a boy aged 12, attending school, was attacked by headache and sore throat. On 6th October, Kernig's sign was present and the patellar reflexes absent. He had not returned to school on 17th October. On 28th September his brother aged nine, attending school, had an acute attack of vomiting, and recovered. On 6th October his patellar reflexes were present.

*House 13.*—On 25th September, a boy aged 10, attending school, was attacked by headache, vomiting, sore throat and drowsiness, On 6th October Kernig's sign was present and the patellar reflexes absent. He had not returned to school on 17th October.

*House 14.*—On 25th September, a girl aged nine, attending school, was attacked by headache, vomiting, sore throat and drowsiness, In this case the patellar reflexes were present on 6th October. On the 29th two boys aged 11 and six, attending school, were taken ill with headache, vomiting, sore throat and drowsiness, and on 6th October the patellar reflexes were absent in both children.

On October 6th three other children, a girl aged 5 attending school and two boys aged 3 and 1½ years not attending school, having no history of illness, had loss of patellar reflex.

There were no attacks (by 6 October) in the remaining 4 houses although from each of these houses children attended the school.

It is difficult to resist the conclusion that certain of the cases above detailed were attacks of poliomyelitis, and there are substantial grounds for suspecting that most if not all the other cases of minor illness, only brought to light by detailed investigation, represented a mild type of the same disease. If this was the case it points to the existence of additional difficulties in dealing with the prevention of the disease.

Unless cases of poliomyelitis had been known to be present in the county the illness associated with this school would probably never have been the subject of close inquiry. As it is, the school was not closed for this outbreak. In some of the cases no medical practitioner was in attendance, and when the acute symptoms subsided in 2 or 3 days the children returned to school. In only two of the first series of cases were medical practitioners called in, and at the time both of them considered the illness to be due to the results of some common food infection—the chief symptoms being fever, headache, diarrhœa and vomiting.

On 13th September, one of these medical men informed the Medical Officer of Health that one of the children, who was absent from schools, had facial paralysis. The Medical Officer of Health then went to the school and found ten children absent through sickness, and he visited these children at their homes.

It will have been seen that the first two cases of the illness occurred on 21st August, at a time when the school had been closed 17 days, the patients being two children living in House 1. The next case occurred on 3rd September, and this child's brother did not go to school on the opening day, 4th September, as he was attacked by headache and vomiting, but he went to school on the 5th September

to 8th September while still ill. Also on the 4th September a child attended school in the morning, but was too ill to go in the afternoon, and he had not returned to school on 17th October. Thus on the day of the opening of the school there were children in attendance who were actually suffering from the malady, and the school cannot be held responsible for the initial spread of the disease. The subsequent spread of the disease, however, may well have been connected with school infection.

Given the fact of the existence of cases of poliomyelitis in a district, it seems important that minor cases of illness occurring among the school children and in their houses should receive the close scrutiny of the Medical Officer of Health.

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## Sanitary Administration in Devon and Cornwall, as Illustrated by the Outbreak.

When I first visited Bude, Stratton, Holsworthy, &c., and had satisfied myself that an epidemic disease, in all probability poliomyelitis, was prevalent, I endeavoured to impress on the district councils concerned that they should use all available means to ascertain the number of cases which had occurred and were arising, and should get ready suitable isolation hospital provision in view of the probable infectiousness of the malady, the large number of summer visitors in the neighbourhood, and the difficulty of giving satisfactory treatment and isolation at the homes of those attacked. The Stratton and Bude Urban and the Stratton Rural District Councils conjointly had acquired a site and were at that date erecting an isolation hospital; but as the building was not far advanced they promptly met their difficulty by the provision of hospital tents which they erected on their hospital site.

In the matters referred to above I found the Holsworthy Urban and Rural District Councils had already been well advised by Dr. Gray, the Medical Officer of Health for these districts; and it will suffice to refer to such cases as those noted on p. 37 above to realise that the prompt isolation of individual cases which he and I had in view might have done much to limit the spread of the disease. I found, however, in these districts (and later on I had the same experience elsewhere in both counties) great reluctance on the part of the local authorities to do anything which would either involve expenditure of money or in any way advertise the fact that the disease, then generally understood to be "spotted fever," existed in their districts, as by so doing their holiday traffic might be prejudicially affected. This reluctance, as it proved, was unfortunate; it created a feeling of local insecurity which satisfactory measures at the outset would have prevented. At Holsworthy and elsewhere the Medical Officer of Health was placed in the false position of having to allay needless alarm and to answer countless inquiries from residents and intending visitors without being in a position to reply that all cases of the disease were well known to him and isolated; and that the whole outbreak was strictly limited and under control. He might easily have been placed in this position by timely action on the part of his authorities. On 8th August the Stratton Rural and the Stratton and Bude Urban Councils passed

the necessary resolutions to make the diseases cerebro-spinal fever and acute poliomyelitis compulsorily notifiable under the Infectious Disease (Notification) Acts.

On 8th and 9th August similar resolutions were passed by the Holsworthy Urban (carried only by the casting vote of the chairman) and Rural (after an adjournment of the matter from a previous meeting) District Councils. But while this was valuable, so far as the local medical practitioners recognised their responsibility, the latter two district councils took no steps to provide hospital treatment or even home isolation with suitable nursing. In one district, Holsworthy Urban, the clerk of the authority went out of his way, in my opinion without any justification, to allay the public anxiety by claiming that hospital provision was available. On 5th August a letter signed by him as clerk to the urban district council was published in the local press; it concluded with the following paragraph :—

"Your information as to a hospital is also at fault. There is "an isolation hospital at Holsworthy."

Four photographs of this "isolation hospital," taken a few days after the clerk's letter appeared in the newspapers, are reproduced in this report. They show without need of written description the character of the hospital accommodation provided.

The pressing need for isolation accommodation in the Holsworthy Rural District, within which at that time some 30 cases had occurred, led Dr. Gray to order hospital "shelters" without waiting for the next meeting of his council. The circumstances were as follows :— When the child No. 58 was attacked (*see* p. 41) his mother was hourly expecting her confinement. She was forced to attend to her sick child, as no one could be found who would nurse either the child or the mother; there was no isolation hospital to which the child could be removed, and no one would receive the mother from the infected house. Before the "shelter" was delivered the child had died.

As more information came to hand showing the wide area over which cases of the disease were occurring, I visited first the districts nearest to Holsworthy, and subsequently others further afield, and was fortunate in receiving the advice and cordial co-operation in these matters of Dr. Adkins, the County Medical Officer of Health for Devon, and of Dr. Burnet, the County Medical Officer of Health for Cornwall. In the end, as a result of representations made by us, the two diseases, acute poliomyelitis and cerebro-spinal fever, were ultimately made notifiable under the Infectious Disease (Notification) Acts in 41 out of the 52 sanitary districts in Devon and in 24 out of the 41 sanitary districts in Cornwall. This number does not include the County Boroughs of Devonport, Exeter, and Plymouth, which are outside the County Administration, but where, however, the two diseases were made compulsorily notifiable.

The South Molton Urban District Council was equally divided on the matter of compulsory notification, and the mayor refrained from voting. Later, when several fatal cases had occurred in their borough, the council applied to the Local Government Board for sanction to make the diseases notifiable.

THE ISOLATION HOSPITAL OF THE URBAN AND RURAL DISTRICT COUNCILS, HOLSWORTHY, DEVON.

No. 1. Shows the front elevation of the "Hospital."

No. 2. Shows the back elevation of the "Hospital."

*Photographs taken in August*, 1911.

No. 3. Shows one side view of the "Hospital."

No. 4. Shows a view of the interior of the "Hospital." The construction of the floor of the front room is clearly shown. Although at the time the photograph was taken there had been no rain for a month, water stands several inches deep on the floor of the back room seen through the doorway. There is no furniture.

*Photographs taken in August*, 1911.

The Totnes Rural District Council at their meeting on 15th September, at a time when the disease had appeared in their district, decided not to adopt compulsory notification of the disease, apparently because they considered they would have to arrange for the isolation of those attacked.

The Urban District Council of Ilfracombe, fearing to frighten visitors from their town, agreed to a system of voluntary notification by arrangement with the local medical practitioners. The wisdom of this course is open to question ; it is conceivable that the visitors to the town would have had greater confidence in the local sanitary administration if the council had taken the more effective precaution of making the disease compulsorily notifiable. Medical practitioners in the absence of statutory duty are loth to notify such cases, partly on account of objections made by friends of the patient, and partly because they fear that by so doing they exceed their professional duty to the patient, and may even render themselves liable to a prosecution for damages. The local newspaper press, by directing attention of the public to the presence of the disease in the counties, thus affording all classes of the community a knowledge of facts as to the sickness, in all probability materially contributed to lessen spread of infection by sick persons the nature of whose illness would, but for information derived from the press, have been unrecognized.

As regards hospital treatment of known cases, most of the districts were in like position ; no isolation hospital provision was available. Several cases of consequent hardship to individual sufferers and needless danger to contacts came under notice. Such a case has been incidentally mentioned in connexion with cases 38 and 58 (*see* pp. 41 and 48). The isolation of the first case in this instance might have prevented what was little less than a tragedy in this small village.

The case of the nurse, W. (97), referred to on page 22, occurred in the Bideford Rural District, in which there is no isolation hospital provision. When attacked she was nursing a confinement case in a three roomed cottage. The medical attendant wanted to remove the mother and her family, but they refused to leave the house. The owners of empty houses refused to let them for the isolation of the nurse, and the landlady of the cottage where she lodged declined to have her back. Under these circumstances the medical attendant, in the face of adverse criticism, and to his great credit, took the nurse into his own house, isolated her as far as possible and engaged a nurse to attend her.

Local authorities generally were advised that the invaded houses should be disinfected, and that the throat and nose passages of the patient and of all contacts should be sprayed with a solution of permanganate of potash.

Thorough disinfection of "fomites" by steam was, however, seldom undertaken ; very few of the district councils are provided with a steam disinfecting apparatus.

In the present knowledge of poliomyelitis search for and prompt isolation with proper treatment of infected cases are practically the only preventive measures which can be applied.

The epidemic under review has, in my opinion, important lessons which sanitary authorities in Devon and Cornwall would do well to take into account. As regards hospital accommodation the present position is as follows :—

## DEVONSHIRE.

There are 55 sanitary districts, including the 3 county boroughs in Devonshire, 37 urban, and 18 rural districts. Of these the following sanitary districts have some isolation hospital provision :—

| District. | Number of beds. | No. of different diseases accommodated. | Remarks. |
|---|---|---|---|
| Devonport Co. Boro' ... | 65 | 3 | Not built on loan, and 15 beds for small-pox. |
| Exeter „ ... | 80 | 4 | Built on loan. |
| Plymouth „ ... | 96 | 3 | Built on loan, and 44 beds for small-pox. |
| Barnstaple Urban District | 14 | 3 | Not built on loan. No accommodation for small-pox. |
| Bideford „ „ | 9 | 1 | Not built on loan. No accommodation for small-pox. |
| Tiverton Urban and Tiverton Rural Districts. | 23 | 2 | Not built on loan. No accommodation for small-pox. |
| Torquay Urban District ... | 17 | 2 | Built on loan. 6 beds for small-pox. |
| Heavitree „ „ ... | 10 | ? | Not built on loan. |
| Ilfracombe „ „ ... | 25 | 3 | Built on loan. ? 3 beds for small-pox not provided by loan. |
| Lynton „ „ ... | 2 | 1 | ... ... ... |
| Newton Abbot U. & R. Ds. | 26 | 3 | Not built on loan. No accommodation for small-pox. |
| Paignton U. & R. Ds. ... | 6 | 1 | Not built on loan. No accommodation for small-pox. |
| Plympton St. Mary R. D. | 12 | For small-pox only. | Not built on loan. |
| Teignmouth Urban District | 8 | 2 | Built on loan. No accommodation for small-pox. |

Budleigh Salterton, Crediton, Dawlish, Exmouth, and Sidmouth Urban Districts and Crediton and St. Thomas Rural Districts send cases to the Exeter Isolation Hospital.

East Stonehouse Urban District sends cases to Devonport, and Plympton St. Mary sends cases to Plymouth.

The isolation hospital provision of the Urban and Rural Districts of Holsworthy has been referred to in this report.

## CORNWALL.

There are 41 sanitary districts in the county, 27 urban and 14 rural districts. Of these the following have some isolation hospital provision :—

| District. | Number of beds. | No. of different diseases accommodated. | Remarks. |
|---|---|---|---|
| Falmouth Urban ... ... | 8 | 2 | |
| Penzance „ ... ... | 14 | 2 | Temporary building, used also by Penzance Port Sanitary Authority and Paul U.D.C. |
| Truro „ ... ... | 15 | 2 | No administration block, discharge block, or disinfector. No accommodation for small-pox. |
| Camborne „ ... ... | 8 | 1 | No administration block, discharge block, or laundry. |
| Newquay „ ... ... | 8 | 2 | Used also for small-pox. |
| Redruth Urban and Rural | 12 | For small-pox. | Conjointly for the two Districts. |
| Truro Rural ... ... | 6 | 2 | |
| Stratton and Bude Urban, conjointly with Stratton Rural. | ? | ? | In course of erection. |

It will thus be seen that practically no isolation hospital provision for effectively dealing with cases of infectious disease exists in the greater part of either of these counties, a matter of much importance not only to the inhabitants but to the large migratory population which comes—often with children—to Devonshire and Cornwall for holiday and health.

An isolation hospital to be of real assistance should be ready to receive the first known cases of infectious disease, as by so doing epidemic prevalence of infectious disease may be prevented. Makeshift hospitals are rarely of effective use when they are improvised in emergency under the influence of panic. Usually their administration is unsatisfactory, and the details of water supply, sewage disposal, cooking, and the housing of the nurses employed seldom receive adequate consideration. Rapid destruction by fire of hospitals built of unsatisfactory "temporary" materials is not unknown.

Great care should be exercised in regard to design and to construction of isolation hospitals, whether these buildings are provided out of loans sanctioned by the Board after submission of the plans to their expert advisers, or are erected out of current rates. Outside the county boroughs only three isolation hospitals in Devonshire have been built on loan sanctioned by the Board, and no hospital has been so erected in Cornwall.

As I pointed out in 1909, in a previous report which dealt with a

sanitary district in Devonshire,* a county council may, under the Isolation Hospitals Acts of 1893 and 1901, direct their Medical Officer of Health to report on the hospital requirement of any part of their county, and if this report shows that isolation hospital provision is required, they may hold a local inquiry, after which, if they see fit, they may make an order constituting a hospital district and defining its area. The order constitutes a hospital committee consisting of local representatives, but if a grant be made out of the county fund the committee may consist wholly or in part of county councillors. The order further gives the committee power to provide and maintain a hospital. The county council have power of raising money by loan for the purposes of the hospital.

In 1909 the Devonshire County Council had only recently appointed a County Medical Officer of Health, and a County Medical Officer of Health for Cornwall was only appointed during the present year.

A second consideration arising out of the outbreak, with which I could not fail to be impressed, was the unsatisfactory character of the organisation of sanitary authorities in both counties for the prevention of infectious disease generally, especially in regard to the sanitary staff available for the purpose. In these two counties outside the county boroughs, there are but two local Medical Officers of Health who are not engaged in general practice; one acts for several districts and the other holds other public offices. All but 14 of the Inspectors of Nuisances hold other appointments.† Of 77 Medical Officers of Health only twenty-two hold a diploma in public health or state medicine.‡

Medical Officers of Health in country districts receive comparatively small remuneration for the work now required of them by statute; and this work if efficiently performed frequently brings them into conflict with vested interests. Moreover they do not always receive that support which they should be entitled to receive from their councils. Again when epidemic disease shows itself in a district, a Medical Officer of Health who is engaged in private practice and particularly in a country practice can ill afford the necessary time to effectively deal with the outbreak. Several examples of this were met with during this recent epidemic, and the time which the several local Medical Officers of Health were prepared to give to their public health work and the amount of interest they took in it varied within very considerable limits.

A Medical Officer of Health may with the sanction of the Local Government Board be appointed for two or more districts (section 191, Public Health Act, 1875), and the Local Government Board may by order unite two or more districts for the purpose of appointing a Medical Officer of Health (section 286, Public Health Act, 1875). In the past there has been considerable difficulty in bringing

* Reports to the Local Government Board on Public Health and Medical Subjects (new series, No. 6). Dr. R. J. Reece's Report to the Local Government Board on a recent epidemic of scarlatina in the Sidmouth Urban District and on the General Sanitary Circumstances and Administration of that district, 1909.

† The inspectors of nuisances of the County Boroughs are not included in these 14. When this enumeration was made there were one or two vacancies.

‡ In certain instances the same gentleman acts for more than one district.

about combination of districts for the appointment of whole time Medical Officers of Health. This has arisen partly from the hostility of districts to combination, and partly because in sparsely populated districts it has been considered impracticable to combine sufficiently large areas to support a salary requisite for a whole time officer. The latter difficulty has been modified in recent years by the advent of motor transit.

The conditions under which combination of offices with a view to secure the service of a Medical Officer of Health not engaged in general practice are given in a memorandum of the Board,* and I am satisfied that in these counties great improvement would result if the policy indicated by the Board in this memorandum were carried out.

I am of opinion that a revision of the previous policy of sanitary administration, the provision of suitable isolation hospital accommodation, and the appointment of expert Medical Officers of Health, are matters of imperative importance if the counties of Devon and Cornwall are to maintain their reputation as health resorts.

In closing this report I desire to put on record my appreciation of the assistance afforded me by the officers of many district councils and by the members of the medical profession; without such assistance this report would be lacking in many essential details. Especially I would thank Drs. Adkins and Burnet, Medical Officers of Health of the administrative counties of Devon and of Cornwall; their assistance extended far beyond the amenities of official relationship. Of the District Medical Officers of Health, to all of whom I am indebted for much courtesy and on whose time I have trespassed considerably, I would specially thank Dr. Gray, of Holsworthy, for his cordial co-operation. It is painful to set on record that his unflinching devotion to the health interests of his districts, and the unswerving manner in which he carried out his duties as a Medical Officer of Health, failed to meet with the approbation or even the approval of many of those immediately concerned.

RICHARD J. REECE.

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* Memorandum of the Local Government Board in regard to appointments of Medical Officers of Health and Inspectors of Nuisances, 1910.

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 9 ms., F. | 6th May. | | | + | | | | | | | + |
| 2 | 5, M. | 3rd May | 103·2°. P. 136. | + | No | + | | + | + | + | + | + |
| 3 | 4, M. | 15th June | 102°. P. 120. | + | No | + | Slight | General | + | + | + | + |
| 4 | 10 ms., M. | 29th June | | + | No | | | | | + | No | |
| 5 | 10 ms., F. | 29th June | | + | No | | | | | + | No | |
| 6 | 7, M. | 22nd July | | + | No | | | | | + | No | |
| 7 | 4, F. | 1st week in July. | | + | + | | | | | + | | |
| 8 | 2½, M. | 9th July | | + | + | | | | | | No | |
| 9 | 5, F. | About 19th July. | | + | + | | | | | | No | |
| 10 | 2½ ms., F. | 17th July | | + | | | | | | | | |
| 11 | 3 ms., M. | 21st July | 100·8—102 F., Pulse 122-138. | + | + | | | | + | + | + | |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| | 11th May—Paresis, right arm, passing to complete paralysis on 13th May. 14th May—left arm affected, complete paralysis on 15th. 19th May—both legs completely paralysed. Passed urine involuntarily. | | | | + | Blush resembling erysipelas left side of head and Forehead. | | | Duration of illness 14 days. **Died 23rd May.** |
| Plantar and patellar absent. | Complete motor paralysis of both legs. | No | No | No | + | Half-a-dozen parpuric spots on arm and leg. | | C. | All family attended Holsworthy Fair. |
| | Slight loss of power lower limbs and right arm. Aphasia for 4 days. | No | No | No | + | No | | C. | Father, mother and three children all sleep in one bedroom—12′×18′×6′6″. |
| | Loss of power right arm for 14 days. | | | | + | No | | | Very ill for 3 or 4 days. Four of eleven inmates attacked. Dirty house with insanitary surroundings. |
| | No | | | | + | No | | | |
| Patellar—absent right, diminished left. | No | | | | No | No | | | |
| Patellar—absent right, present left. | No | | | | No | No | | | |
| Patellar diminished. | Partial loss of power, both legs. | | | | + | No | | C. | Father, mother and three children all sleep in one bedroom—12′×10′6″×6′6″. Two cases in one house. Farm labourer. |
| Patellar absent. | No | | | | No | No | | C. | |
| | 18th July — Paralysis both legs. 19th July — Paralysis of intercostal muscles. | | | | + | Several petechial spots on both legs and lower abdomen. | | | Clean house. **Died 21st July.** |
| Patellar and plantar absent. | 24th July — Paralysis of both legs and some paresis of both arms. 25th July — Both arms paralysed. 26th July — Intercostal muscles paralysed. | | | | + | 4 circular spots right arm, 6 spots right leg. 2 spots left leg, 3 spots right cheek. | | | Frontanelles shrunken, but spinal fluid under pressure after death (lumbar puncture). Taken to Fair 12th-13th July. None of the family attend school. **Died 26th July.** |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 12 | 2, F. | 1st July | 104° F. for about 4 days. | + | + | No | No | | + | + | | |
| 13 | 9 ms., M. | 6th July | 102° F. | + | | No | No | | | + | No | |
| 14 | 4, F. | 16th June | 101° F. | + | + | + | No | + | + | + | | + |
| 15 | 6, F. | 29th June | Feverish | + | + | | | | + | + | | |
| 16 | 5, F. | 16th July | Feverish | + | + | + | | | | + | | |
| 17 | 9, M. | 13th July | Feverish | + | + | | | | | + | Slight | |
| 18 | 13, F. | 28th July | Feverish | + | No | | | 12th Aug. Complete loss of sensation lower third of flexor surface and one half external surface, right arm. | | + | No | |
| 19 | 7, F. | 14th July | Feverish | + | + | | | Pain in neck. | | + | + | |
| 20 | 8, F. | 21st July | 101° | + | No | | No | No | | + | + | |
| 21 | 42, M. | 21st July | | + | + | | No | Pain back of head. | | Slept for 3 days. | | |
| 22 | 2½, M. | 19th July | Feverish, very hot. | + | | | No | | | + | No | |
| 23 | 10, F. | 26th July | | No | No | | No | Pain in neck. | | + | No | |
| 24 | 3, M. | 22nd July | | + | + | | No | Pain in legs. | | + | Right leg doubtful. | |
| 25 | 4, M. | 4th July | "Feverish" | + | + | + | | | + | | + | |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Patellar + | Loss of power both legs. | | | | + | Purpuric rash over each tibia. | Right cheek. | | Deaf right side, discharge from right ear. Semi-detached cottage. Fleas. A second case in this house. |
| Patellar present. | No | | | | | Spots on boby, 6th July. | | | |
| Patellar—left absent, right present. | Paresis both legs | | | | | No | No | | Grocer's shop. |
| | Loss of power both legs; one partial recovery, one paralysed. | | | | + | No | | | Five inmates—all sleep in one room. |
| | | Bled from nose after death. | | | + | | | | Fatal case—died 18th July. All family attended Holsworthy Fair on 11th and 12th July. All the family insect-bitten. Four inmates attacked. Farm labourer. A bull found ill, died in 2 hours—"like sun-stroke." Calf also found dead with retraction of the head. Reported on in text. |
| Patellar absent. | Walks with difficulty. | | | | | | | | |
| Patellar absent. | Paralysis of deltoid, right arm. 11th August—Paralysis of right arm. | | | | | Covered with insect bites to an extraordinary degree. | | | |
| Patellar absent. | No | | | | No | | | | Nits on hair. |
| Patellar absent. | Loss of power left leg below knee, 25th July, and retention of urine 24 hours. | | | | No | No | | C. | All family insect-bitten. Large farmhouse. No. 21.—Recovered in 3 days, but felt weak for several days. No. 22.—Recovered in 2 days. Four attacks in this house. No school attended. Governess in house. |
| | Felt very weak | | | | | | | C. | |
| Patellar present. | No | | | | | | | | |
| Patellar diminished. | No | | | | | | | No | |
| Patellar absent. | Paresis both lower limbs. | | | | No | No | | | Blepharitis. None of the family attend school. |
| Patellar absent both sides. | Lost use of all four limbs; arms recovered; cannot stand. | | | | + | No | | | Father, mother, girl 14, and patient, occupy one bedroom; boys, 16 and 7, sleep in another. Clean house. Earth floor. |

| No | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 26 | 7, M. | 24th July. Said to have been ailing for fortnight previously. | "Feverish" | + | + | + right side of face. | | | + | + | + | + |
| 27 | 10, F. | 17th June | "Feverish" | + | + | No | | Violent pain legs and back. | | | | |
| 28 | 1 10/12 M. | 27th June | | | | | | | | | | |
| 29 | 2½, M. | 26th July | 100·0 (27th July). | + | + | + | No | Pain on movement. | + | + | + | |
| 30 | 8. M. | 31st July | 100·0 | + | + | + constant sniffing. | | | | | Right slight: Left absent. | Slt. |
| 31 | 36, F. | 26th July | 97·2. Pulse 52. | + | + | + face muscles. | | | + | + | No. | + |
| 32 | 4, F | 9th July | Feverish | + | No. | | | Pain back of head and neck. | | | + | |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Patellar absent both sides. | Aphasia, recovered; some facial paralysis right side. drooping upper eyelid; tongue protrude to *LEFT* side. | | | | | No | | | |
| | Total loss power both legs, 23rd June; paralysis right arm and right leg. | | | | No | | | C. | Removed to private general hospital at Halwell, 20th July. Not clean. Farm house. |
| | Right arm and right leg. | | | | | | | | Well-to-do farmer. No school attended. **Died 30th June.** |
| Patellar absent, plantar diminished. | Loss power both legs. | | | | Slight | Transient back and flank, ? character. | | | Large well appointed house. Clean. No school attended. |
| Patellar right diminished. | Loss power both legs. | | | | No | No; fleabitten. | | | Child attacked while in Bude with mother (a charwoman) who was cleaning a house. During convalescence—when an attempt was made to get him up—he collapsed (? cardiac failure)—and was extremely ill. Mother takes in washing. |
| Patellar feeble, plantar +. | Loss power left arm; speaks slowly and with great difficulty; retention of urine. | | | | No | No | | | Confined of a child on 22nd July. Attended in confinement by the nurse who laid out fatal case No. 16. Nurse went to nurse child but finding it dead laid it out, and, thinking circumstances rather suspicious, changed her clothes and had a bath of lysol, but did not disinfect her throat. |
| Patellar—right absent, left diminished. | Right arm; unable to walk for a fortnight. | | | | | No | | | On 7th July mother visited house of cases 4-7 but did not take the child with her. No school attended. |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 33 | 6, M. | 24th July. Failing for a week before attack. | | + | + | + | | Over lower dorsal region. | | + | Right + Left slight. | Slt. |
| 34 | 8½, M. | 27th July | 102·2 | + | + | + | | | + | | | |
| 35 | 7, M. | 28th July | 103°, 99 on admission to General Infirmary. | | + | No | | | | | + both sides. | |
| 36 | 5, M. | 14th June. Ill a week before. | | + | + | + | + | | + | | No K. on 10th Aug. | |
| 37 | 20, F. | 1st Aug. | 101·4 | + | + | + | | Whole body; extremely sensitive: Pain in the back and limbs 6th Aug. | | | + | + |
| 38 | 36, M. | 7th Aug. | "Fever," 102·0. | + | No | No | | No | No | | Right side. | |
| 39 | 7, M. | 28th or 30th July. Acute onset on 5th Aug. | | + | + | + | + | No | + | | | Slt. |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Patellar—right diminished, left slight. Plantar—right absent, left present. | Loss power lower limbs. | | | | + | No | | | Father of patient sold horse to father of case 40. See text. |
| | Right arm .. .. | Bloody discharge from nostrils after death. | | | | ? a sweat rash. | | | Stable adjoins house. Taken ill at school. Isolated house. All small rooms. Died 30th July. |
| Patellar—left exaggerated, right diminished. | Partial paralysis lower extremities; walks with difficulty. | | | | No | Not observed, covered with fleabites. | | C. | First symptom pain in legs. Admitted General Infirmary on 31st July. |
| Patellar present both sides on 10th August. | 10th Aug. Weakness in lower limbs: drags right leg; could not walk after attack; crawled on hands and knees. | | | | + | "Spots on back of neck." | + On side of mouth. | | Irritability passing into drowsiness. |
| Not examined. | None on 6th August, afterwards developed paralysis both legs. | | | | None on 6th August. | No | | C. | Lumbar puncture. Had been at home since March. Domestic servant. Menorrhagia, and has had hœmoptysis tuberculous. Died 8th September. None of the family attend school. |
| Patellar—right absent, left present. | Great weakness of legs; can stand upright, but if attempts to move knees give way and "down he goes." Can move left leg but not right. | | | | No | | | D. | Ascending paralysis. Died 10th August. Farm labourer. |
| Patellar present. | | | | | + | "Old rash about it," ? fleabites. | | C. | Had been ill a week. Going to school complained of headache. Had been kept at school by school attendance officer 8 days, although the mother had asked that the child should be allowed to remain at home for a day or two as he was ill. Father a farmer. |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 40 | 5½ M. | Acute onset on 9th Aug. | 103° | + | + | + | | | + | + | + | + |
| 41 | 2½, M. | 8th Aug. "Lying about one day" before acute onset. | 101° | + | + | | No | | + | + | Slight | + |
| 42 | 6¾ M. | 4th Aug. | Irregular, never higher than 102·0. P. 120, R. 40. | + | + | + | | | + | | | |
| 43 | 1⅔ M. | 7th Aug. | | + | + | + | | No | + | | + right, left slight. | + |
| 44 | 6, F. | 10th Aug. | 101·6. Pulse, 150 cir., Respiration, 28. | + | + | + | + | Screams when moved. | + | | + | No |
| 45 | 1½, M. | 4th Aug. | | | No | | | | + | + | Absent 13th Aug. | |
| 46 | 3½, M. | 5th Aug. | | + | + | | | | + | | Absent 13th Aug. | |
| 47 | 8, F. | ? 21st July. Acute attack on 5th Aug. | 101·6 | + | + | | + | | + | + Slept for 4 days. | Absent on 15th Aug. | |
| 48 | 12, M. | 14th July | | + | + | | | Limbs attacked, much pain. | | + Could not be kept awake. | | |
| 49 | 6, F. | 17th July | | + | No | | | | | | Absent 14th Aug. | |
| 50 | 9, M. | 17th July | | + | + | | | | | | Absent 14th Aug. | |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Patellar absent. | Paralysis both legs | + | | | + Not marked, but distinct opisthotonos making lumbar puncture difficult. | No | | C. | Brother of following case. Mother and infant attacked later. Nos. 123 and 124. The father of case 33 delivered a horse to this farm on 2nd August and stayed for a meal, sitting between their boys. No. 33 was ill at that time, 10 miles distant. |
| Patellar absent. | Paralysis both legs | | | | No | No | | C. | No known cases in this neighbourhood. Lumbar puncture done in case 40. |
| Patellar, Plantar – at first exaggerated. Before death – patellar absent, plantar present. | Descending paralysis; arms first and legs before death; paralysis of intercostal muscles; incontinence of urine. | | | Slight | Obvious but not extreme. | | | C. | Conscious until a few minutes before death. Lumbar puncture after death. Died 8th August. |
| Patellar absent, plantar present. | Both legs. Abdominal muscles. Later paralysis of intercostals. | | | Congested. | + | No | | | Furred tongue. Died 8th Aug. |
| | Paralysis both arms. Intercostal muscles. | | | | No | No | | | Lumbar puncture. Died 13th August. Father small farmer. |
| | No | | | | | | | | Ill, drowsy, crying out at night. Not attended by medical practitioner. Recovered by 13th August. Ill for 3 days. |
| | No | | | | | | | | Ill for 1 day. Recovered by 13th August. Three attacks in this house. |
| Patellar present both legs on 15th Aug. | Paralysis right external rectus muscle, right eye. When first attacked paresis of legs, inability to stand. | | | | No | No | | | |
| | Only weakness noticed. | | | | | | | | Father a blacksmith. Three attacks in this house. |
| Patellar – right present, left absent. 14th Aug. | Only weakness noticed, | | | | | | | | |
| Patellar present 14th Aug. | No | | | | | | | | |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 51 | 2, F. | 29th July. Acute 4th Aug. | Feverish | + | + | | | | | + | Absent 14th Aug. | |
| 52 | 3½, F. | 12th Aug. | 13th Aug.—101·6. Pulse 128. 14th Aug.—100·2. Pulse 122. | + | + | + | | Pains in head and neck, spine tender. | | | Present right side. | No |
| 53 | 1 11/12, M. | 13th Aug. | 14th Aug.—102·5. 15th Aug.—100·0. 16th Aug.—97·0. | + | + | + | + | 18th Aug.—upper part of trunk, but not lower. | | + | + | |
| 54 | | | | | | | | | | | | |
| 55 | 3, F. | 9th Aug. | 98·4, 99·6. Respiration 36. Pulse 120, irregular. | + | + | + | + | + | + | ++ | + | + |
| 56 | 7, F. | 17th Aug. | 102·6 (17th), 102·0 (18th), 103·2 (18th). Pulse 120. See temperature chart. | + | + | No | No | Pain and tenderness back of neck. | No | + | + | + |
| 57 | 1½, F. | 21st Aug. | | | | | | | | | | |
| 58 | 2 11/12, M. | 17th Aug. Poorly for a week before. | 25th Aug.—99·4. Pulse 104. | | + | | | Cannot bear to be moved, cries if touched. | + | | + on left side. | + |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Patellar present on 14th Aug. | Inability to stand for two days. Lost power left arm on 4th Aug. | | | | No | No | | | Father a sailor, away at sea. On 14th Aug., some power left arm; can flex but cannot extend it. |
| Patellar doubtful or absent Plantar exaggerated. | Some weakness left arm. Followed by plastic paralysis both legs and right arm. | | | | No, but cannot bear to have head moved. | Vivid flush across forehead. | | C. | Semi-detached cottage. 20th August — Lies in bed, dreads to be moved. Great tenderness down spine. Cannot extend legs, particularly right. Unable to raise right arm. Left arm raised with difficulty. Takes little notice of anything. |
| Patellar at first—left +, right diminished. 18th Aug.—leftabsent, right present. | General muscular reflex. | | | | + | Eruption upper part of legs, at first mistaken for scabies, not present on 18th Aug. but some discolouration remained. | | C. | Father a farmer. |
| | | | | | | | | | |
| Patellar and plantar present. | No marked paralysis. Unable to stand at first. | No | No | No | + | No | | C. | Father a farmer. Clean family. Tongue dirty. |
| Patellar absent, plantar right +, left normal. | No | No | No | Slight | Not observed. | No | No | C. | |
| | | | | | | | | | |
| Patellar absent. | Pupils widely dilated. Paralysis of right arm followed by paralysis of the intercostal muscles. | | | | + | | | C. | Tongue thickly coated. Child aged 11, daughter of case No. 38, lives in this house. She saw her father day before his death. Father of patient assisted at burial of No. 38. Father a blacksmith (see Text). **Died 26th August.** |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 59 | 5, M. | 15th Aug. | | + | No | No | No | 20th Aug.—Along spine. | No | | + | + |
| 60 | 9, M. | 30th June | 102 to 104 | + | No | No | No | No | No | No | No | No |
| 61 | 7, F. | 16th Aug. | Feverish | + | | | | | | | No | |
| 62 | 14, F. | 18th Aug. | „ | + | + | | | | | | No | |
| 63 | 5, M. | 14th Aug. | „ | + | | | | | | + | No | |
| 64 | 3, F | 14th Aug. | „ | + | | | | | | + | No | |
| 65 | 13, M. | 17th Aug. | „ | + | | | | | | | | |
| 66 | 10, M. | 16th Aug. | „ | + | | | | | | | | |
| 67 | 7½, M. | 22nd Aug. | 103° | + | No | + | | + | + | | + | + |
| 68 | 3, M. | 26th Aug. | Normal | + | + | No | No | No | + | No | No | No |
| 69 | 12, M. | 24th Aug. | | + | + | | No | | | + | + | No |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Patellar absent. | 19th, complained of pain right arm. Followed by loss of power in limb. 20th, cannot move either leg off bed. 27th, paralysed all four limbs. | | | + | No | Flea-bitten | | | Previous fatal case in adjoining cottage (see Case 120). Many flies. Father a farm steward. Some chickens had died at this farm 2 or 3 days before child became ill. |
| Normal | No | No | Mucous discharge. | | No | No | No | C. | Had not been out of Bude for months. Father a club steward. Clean house. Died 5th July. Certificate—acute bronchitis, cardiac failure. |
| Patellar absent. | None on 25th Aug. | | | | No | Flea bitten | | | Cottage quite close to No. 44. fatal on 13th August. Illness in this family began on 14th August when two children were attacked, followed two days later by two more children and two others sickened on the following two days. An infant 1 year and a child of 12 escaped attack. 65 and 66 not seen nor examined. *Overcrowded.* Dirty house. |
| " | " | | From nose. | + | No | " | | | |
| " | " | | | Slight | No | " | | | |
| " | " | | | | No | " | | | |
| | " | | | | No | " | | | |
| | " | | | | No | " | | | |
| Patellar, right absent. Left feeble | 25th, Paralysis both legs: 26th, difficulty in micturition. 27th, both arms and muscles of trunk; staccato speech; certain of laryngeal muscles, diaphragm and inter costals. | Violent | | | + | No | Herpes labialis 24th August. | C. | Removed to Hospital. This case is reported on the text of the report. |
| Patellar absent. | Paralysis of muscles of respiration. | No | | + | No | No | No | No | Mistaken at first for diphtheria, and D. antitoxin administered. Died 28th August. Same road as 76. |
| | | | | | Slight | | | | Was at South Molton on 18th August. Attacked while in Exeter. Died 29th August. |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 70 | 1 5/12, M. | 10th Aug. | | + | No | | No | | | + | No | + |
| 71 | 5/12, F. | ? 1st July | Feverish | + | + | + One convulsion. | No | | + | + | | |
| 72 | 2½, F. | ? 14th July | Feverish | + | + | + | No | + | + | + | | |
| 73 | 1 9/12, M. | 8th Aug. | | | + | No | No | + | No | + | | |
| 74 | 17. M. | | Feverish | + | No | No | No | + | + | + | Right leg. | |
| 75 | 1 7/12, M. | 4th June | Feverish | | + | + | | "Sore to be touched." | + | + | Not present on 16th June. | No |
| 76 | 7, M. | 26th Aug. | 101° Pulse, 120. | + | + | | | | | | + | + |
| 77 | 2, F. | 4th Sept. | 102° | + | + | No | No | + | + | No | + | + |
| 78 | 2 6/12, M | 7th Aug. | | + | + | | No | | | + | + | + |
| 79 | 3 6/12, M. | 7th Aug. | | + | + | | | | | + | + | + |
| 80 | 3, M. | 23th A g. | 103° | | + | + | | | | ++ | | |
| 81 | 4 | | | | | | | | | | | |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Patellar absent. | | | | | No | No | | | |
| Patellar present. | Complete paralysis right arm; contraction of flexors and fingers; dilatation of both pupils. | No | No | No | + | No | + | D. followed by C. | 14th September, complete loss power right deltoid. |
| | Left leg completely: right leg slight | No | No | No | + | No | No | C. | |
| | At first "weak in back," both legs then became paralysed, followed by right arm, wry neck on right side. | No | No | No | + | No | No | D. | Recovery — first the neck and then the back, and, lastly, the legs recovered. On 23rd September right arm paralysed with exception of slight movement of flexors of fingers. |
| Patellar absent. | Both legs and left deltoid. | Yes | No | No | No | No | No | No | |
| Patellar and plantar absent. | Both legs; 13th June, retention of urine 36 hours. | No | From ears. | No | No | No | No | D. | 6th September—no recovery of left leg; slight powers of extension right thigh. Fleas—patient bitten. Dirty house. |
| Present all. | Squint-paralysis of ext. rectus; fundi normal; no other paralysis. | | | | | | | | Lumbar puncture. Pain in stomach. Same road as 68. **Died 3rd September.** |
| Patellar, first + absent later. | One arm followed by the other on next day; paralysis of respiratory muscles. | No | No | No | No | No | No | No | **Died 9th September.** Very dirty. Many fleas. |
| | Paralysis of muscles of neck. Could not hold head up. | | | | | No | | | Brother of No. 79. Removed to Newton Abbot Isolation Hospital on 28th August. |
| | Muscular rigidity passing to paralysis of both legs on 16th August. | | | | Not marked. | No | | | Brother of No. 78. |
| | Marked squint. left eye; supr. oblique and internal rectus muscles: general paresis, ? Paralysis of intercostal muscles. | | | | Very marked. | No | | | **Died 31st August.** See text. |
| | | | | | | | | | |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 82 | 8, M. | 4th Aug. | 102° | + | + | | Slight | | | No | | |
| 83 | 1½, M. | 5th Sept. | | | + | | | | | + | No | |
| 84 | 6, F. | 20th Aug. | | + | | | | Pain right leg and back. | | Slight | + | Slt. |
| 85 | 11, M. | 14th Aug. | Fever | + | + | | + | | | + | + | + |
| 86 | 5½, F. | 25th Aug. | | + | | | + | | | | + | + |
| 87 | 3, M. | 10th Aug. | Fever, with pain in chest. | + | | | | | | + | + | + |
| 88 | 3½, M. | 29th July | Fever | + | + | | | | | | + | + |
| 89 | 3, M. | 23rd Aug. | 102° | + | | Slight twitching about mouth. | | Pain in back of neck. | | + | | |
| 90 | 3, F. | 10th Aug. | Fever, with pain in legs and arms. | + | No. | | | Pain in legs and arms. | | + no actual coma. | + | + |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| | Paralysis—first in legs, spread upwards, involving intercostals and diaphragm and muscles of arm; incontinence of urine before death. | | | | Slight at first, increased later. | No | | | Died 7th August. |
| | Paralysed .. .. | | | | No | No | | D. | Died 17th September. Clean house. Father an optician. |
| Patellar absent. | Considerable muscular rigidity, right leg and back. | | | | No | | | | |
| | | | No discharges. | No | + | No | | | Sanitary condition of house good. Father a game and produce dealer. Had been dabbling about the harbour at Ilfracombe. Had been in Ilfracombe one week, returned day he "took ill." Died on 19th August. |
| | Left leg and spinal muscles of back paralysed. | | | No | + | No | | | Child recovering. At Ilfracombe on 18th August had food there. Quite by herself all August, not associated with other children. Father a gardener. |
| | Evidence of muscular rigidity. | | No | | + | No | | | Father a mason. Not slept away from home. House in good condition. |
| Patellar absent. | Certain muscles of left leg only. | | No | | | No | | | Boy is recovering. Father a plumber. House in good condition. |
| Patellar absent on left side. | Muscles of back and neck appear to be paralysed, right leg weaker than left. | | No | Redness of fauces. | Slight rigidity of neck and retraction of head. | No | | | Not away from district during past month. (Date of Report, 2nd September, 1911.) Father a farmer. |
| | Slight rigidity in back of neck. About 3rd day considerable paralysis of both arms and legs and muscles of the back. | | Mucopurulent discharge from nose for first 2 days at height of illness. | Slight redness of fauces for 2 or 3 days. | | No | | | Well dry at time of illness—water obtained from river. Closet not very satisfactory. Father a farm labourer. Clean house. |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 91 | 8, M. | 2nd Sept. | 101·6. Subnormal on 5th, 6th and 7th. | | Once on 1st Sept. | | | | | + no coma. | + | + |
| 92 | $\frac{3}{12}$, M. | 26th Aug. | Slight feverish attack on Aug. 26th. Mother attributed it to teething. | | | | | | | | | |
| 93 | 9, M. | 2nd Sept. | Rigors | Pain in back of head. | + | | | Pain in legs. | | + no coma. | | + |
| 94 | 15, M. | About 15th Aug. Outset rather slow. | | | | | | | | | Marked both legs. | |
| 95 | $1\frac{2}{12}$, M. | ? 13th–19th August. Onset sudden. | | | Once | | | | | | | |
| 96 | 3, M. | ? 30th July–5th Aug. Onset sudden. | | + | | | | | | Marked; and still somewhat drowsy. [9th Sept.] | | |
| 97 | 35, F. | 28th Aug. Giddiness, sudden onset. | | Occipital headache. | Nausea | | Slight 2nd day. | | | + no coma. | + | + |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| | No evidence of muscular rigidity, but loss of power in muscles. | | No | | Slight | No eruption, but marks – flea bites. | | | Came from Barnstaple about 10 days before commencement of illness. Is said to have gone to Bucks Cross on 22nd (?) August, but had no communication, and was at least ½-mile from another case, reported from Weliand Cottage, Bucks Cross. Patient asthmatic, has had adenoids removed. Anorexia. House in good condition. |
| | About September 2 mother noticed loss of use of right leg and called in doctor. | | None | | | None | | | Father a timekeeper. House in good condition. |
| | Evidence of muscular rigidity in muscles of the back. | | No | | + | None | | | Father a farm labourer. Fairly clean house. |
| Patellar absent. | | | None | | Neck was stiff. | None | | | Employed at a baker's, Teignmouth, 1st-25th August. Went home to Newton Abbot on 12th August for some hours. New house, in good condition. |
| Leg reflexes present. | Left arm now paralysed [9 Sept.]. | | None | | Retraction of head. | | | | Not known whether patient has associated with suspicious cases of illness, but many cyclists have been staying here from time to time. |
| Patellar reflexes absent but plantar present. | | | | | | | | | |
| Patellar and plantar reflexes exaggerated | Muscular rigidity – jaw and leg. | | | + difficulty in swallowing. | | | | | Patient is a district nurse. No recent illness, and no previous serious illness. Nursed a suspicious case about two weeks previously. [See text.] |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 98 | 1½, F. | 21st Aug. Sleepy and dull. | | No evidence. | | | | | | + | + | + |
| 99 | 4½, M. | 24th Aug. | Fever | | | | | | | + no coma. | | + |
| 100 | 23, M. | 20th Aug. "did not feel well." | Fever | + | Some nausea. | | | | | + no coma. | | + |
| 101 | 10, F. | 13th Sept. Onset sudden. | | + | + | | | | | | | |
| 102 | 6, M. | 13th Aug. | 102° on 18th Aug., then normal till the end. | + | None | No | No | Almost complete anæsthesia. | No | Present all the time. | | + |
| 103 | 2, M. | 5th June | Poorly and feverish; temperature not taken. | + | No | 8th June, in convulsions all day. | No | None noticed. | No | + | | |
| 104 | 5, M. | 7th Sept. | 102° | No | Not marked. | No | No | Excessive | No | No | No | No |
| 105 | 5, M. | 9th Sept. | 99°–102° | + | + | No | + | Marked | + | Marked | No | + |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| | Complete paralysis left thigh and leg. | | | | + | | | | Father a colt breaker. Dirty house. Child's two sisters staying at Kesmalton, West Putford, where the disease is present, but no contact proved. |
| | On 6th September he was noticed to be unable to walk properly and doctor was sent for on 10th September. Child has now paresis of back and legs and is much better. | | | | No retraction, but head and neck stiff. | | | | Father a police constable. Clean house. |
| | Stiff neck. | | | | | | | | Father a farmer. Clean house. |
| | | | | | | | | | Father a ganger on Railway. Sanitary condition of house good. Diphtheria 6 years ago. **Died 13th September.** |
| All lost | Complete of both arms and legs and sphincters. | No | No | Present at onset; Dysphagia. | Retracted, neck and back stiff and painful. | No | No | C. | **Died 22nd August.** No P.M. Clean family, some fleas. |
| | 6th June; both legs gave way; Paralysis of both legs, more especially left; retention of urine. | No | No | No | No | No | No | C. | House dirty and fleas. 15th September. Child has made good recovery, able to run about, but limps a little on left leg, which has wasted slightly. |
| Patellar and plantar absent. | All limbs — not sphincter. | No | No | No | + | No | No | C. | Flea infested. Patient much bitten. Lived in house 3 years. Patient and mother at Helston for a week in May. There is slight return of movement in hands, but not in legs. |
| Patellar absent. | Legs; neck .. | No | No | + | Marked | No | No | C. | Large farm house. Went seaside for few days in August. Improving—paralysis disappearing. |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 106 | 8, M. | 15th Sept. | 103°-104° | + | + | No | + | Marked | No | No | No | + |
| 107 | 30, M. | 16th Aug. | 102°-103·5° | Frontal headache on 18th and 19th. | No | No | No | Back and both legs. | No | No | + | + |
| 108 | 14, M. | 13th Sept. (Probably). | 101°-104° | Slight | Persistent for last 14 hours. | Twitching for 24 hours. | Slight | No | No | For 24 hours. | + | + |
| 109 | 4, M. | 16th Sept., ailing, irritable and feevish 4-5 days before. | 16th Sept., 102°; 17th Sept., 2 p.m., 102°; 10 p.m., 101°; 18th Sept., 100°; 19th Sept. and after, normal. | Complained for 3 days. | None; but nausea. | Much twitching at times, especially at night; passed off after the febrile stage. | Slight only. | Apparently none. | Very irritable for a week or more. | + | + | Very mar[illegible] |
| 110 | 4, F. | 14th Aug. | 101° | Slight | No | No | No | No | + | + | + | No |
| 111 * | 6, F. | 6th Sept., 1911. | 103°-89·4° | + | + | No | Slight | No | + | + | + | + |
| 112 | 8, M. | 4th Sept., 1911. | 101°-102·6° | + occipital | + | No | + At night. | No | + | + | No | No |
| 113 | 2, F. | 8th Sept, 1911, | | | | | | | | | | + |
| 114 | 29, M. | 15th Sept. | 101° (even 17th Sept.). | + | + | No | No | + | No | No | + | + |

* A case of acute poliomyelitis occurred in this house 8 or 9 years ago.

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Absent | Whole body .. | No | No | + | Marked | No | No | C. | Died 17th September. No P.M. This case was moribund from the first. Fleas plentiful. |
| Patellar and tendon reflexes absent on 18th. | Paresis both legs on 17th : paralysis of leg and bladder on 18th, extending to arms, thorax, &c., on 19th and 20th. | No | No | No | Not noted | None. Dusky "mottling" of skin of chest on 19th. | No | C. | Died 20th August. Respiratory failure. No P.M. Death certificate acute ascendary myelitis. Clean house. See text. |
| Normal | Nil | No | No | No | + | No | No | No | Died 16th September. No P.M. Came from S. Wales 7th September. Lumbar puncture. |
| Patellar reflexes, both exaggerated. | Paralysis of left facial nerve. | No | No | No | Slight, but distinct. | No | No | C. | Clean house. |
| Patellar absent. | Complete paralysis both legs. | No | No | No | No | No | | C. | Improving. Father a photographer. |
| Absent | Both legs and slightly left arm. | No | No | No | No | No | No | C. | Has visited Teignmouth, Budleigh Salterton and The Warren, one day each place. Clean house. |
| Absent | Right arm .. .. | No | No | Slight ; child has adenoids. | No, but very marked stiffness of neck and pain in movement. | No | Patch on left cheek and round angle of mouth. | C. | Father a farmer. Paresis left arm. Lumbar puncture on 25th September. 21 days previous to attack returned from his grandfather's farm at Woolfardisworthy. Clean house. |
| Patellar absent. | Paralysis both legs. | | | | | | | | Clean house. |
| Patellar absent. Plantar ++ | Left arm : intercostal paralysis : muscles of neck. | No | No | Slight | + | No | No | C. | Patient a farmer. Clean house. 13 days previous visited Bideford market. Died 18th September, 1911. |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 115 | 3, F. | 8th Aug. | | + | + | | | Pain all over, especially head and neck. | | + | Right side. | |
| 116 | 6. M. | 5th Aug. | 103·6° on 6th; 102·5°–102° on 7th; pulse, 130; 102·5° on 8th: pulse, 130°. | + | | Towards the end spasm of the jaw. | | Tender to touch along spine and over muscles of neck. | | | Absent | |
| 117 | 8½, F. | 14th Aug. | Fever | | | | | | | | Slight | + |
| 118 | 10 wk. M. | 16th Aug. | 102·5° | | + | | | | | + Comatose before death. | | |
| 119 | 2½ | ? 1st Aug. | | + | | | | | | | | |
| 120 | 5, M. | 10th July | | | | | | | | | | |
| 121 | 3, F. | 18th Aug. | 20th Aug., 101·4; pulse, 128. | + | + | | | Cannot bear to be moved. Tenderness down spine. | | ++ | Present on left. | No |
| 122 | 9, F. | 14th Sept. | 103° | + | + | No | No | + | No | + | + | No |
| 123 | 28, F. | 11th Aug. | 99° | + | No | No | No | Pain back of head. | | + | | + |
| 124 | 8, M. | 12th Aug. | 101° | | + | No | No | | + | No | + | + |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Patellar present at first absent on 14th Aug. Plantar, right exaggerated on 15th August. | Weakness both legs suffers on attempting to walk. | | | | No | | | | Clean house. |
| 8th August, patellar absent. | Loss power in occupital muscles and all limbs; "curious form of breathing." | | | | + | No | | C. | Died at Bristol; taken ill on way home in train after a fortnight's residence at Bude. Died 8th August. Lumbar puncture. |
| Deep reflexes absent. | Paresis both legs, paralysis 7th nerve and neck muscles. | | | | | | | | Father a rabbit trapper. |
| | "Laboured breathing." | | | | + | History of impetigo on face. | | No. | Father a farmer. Bottle fed. House swarming with flies. Manurial nuisances. Lumbar puncture. Died 17th August. P.M. |
| | 8th August, paralysis of both legs. | | | | | | | | |
| | ? Paralysis of muscles of respiration. | | | | | | | | Died 12th July. Death attributed to "Bronchopneumonia." Case 59 occurred in adjoining cottage. |
| Patellar absent 22nd Aug., and plantar right absent, left present. | Loss power both legs. | | No | No | Extreme on 23rd August. | 22nd Aug., Flush across forehead. | | C. | Dirty house. Insanitary surroundings. Died 20th Aug. Inquest, P.M. See text. |
| Absent | Paresis both legs and partial both arms. | Slight | No | No | + | No | No | C. | Father a farmer. Clean house. A son died from tetanus 18 months ago. One daughter has been staying at Port Isaac; returned a week ago. |
| | No | | | + | No | No | | C. | Recovered in 3 days. See 40 & 41 (mother of these cases). |
| | No | | | | Slight | No Insect bitten. | | C. | Illness lasted 36 hours. Brother of 40 & 41; son of 123. |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 125 | 4, M. | 18th Aug. | 101° on 4th day. | | + | + | Screaming. | No | + | No | + | + |
| 126 | 2 5/12, M. | 15th Aug. | 101·6 | + | No | No | No | Both legs. | + | + | | |
| 127 | 6, F. | 5th Sept. | | | | | | | | | | |
| 128 | 19, F. | 29th Aug. | Feverish | + | + | No | + | Pain in back and spine, and under left shoulder blade. | No | + | + | + |
| 129 | 3, M. | 25th Aug. | 100·4 | + | + | No | No | + | + | | No | No |
| 130 | 5, F. | 4th Sept. | | | | | | | | | | |
| 131 | 3 5/12, F. | 6th Sept. | | | | | | | | | | |
| 132 | 2 5/12, M. | 1st Sept. | | + | + | | No | | | + | | No |
| 133 | 4 5/12, F. | 15th Sept. | | + | No | | + | | | + | + | + |
| 134 | 13, M. | 9th July | | | | | | | | | | |
| 135 | 13, F. | 30th July | | | | | | | | | | |
| 136 | 28, F. | 29th Aug. | 99·2 | + | + | No | No | + Pain right leg, tingling over right side face, lower part. | | + | + | |
| 137 | 16, M. | 29th Aug. | | + | No | | No | | | + passing to coma. | + | + |
| 138 | 2 5/12, M. | 9th Sept. | 101°–103°. | + | + | No | No | + | + | No | No | No |
| 139 | 7, F. | 1st Sept. | | No | No | No | No | Right shoulder and right side neck. | No | No | No | |
| 140 | 25, M. | 28th Aug. | | | | | | | | | | |
| 141 | 24, M. | 9th Sept. | | | | | | | | | | |
| 142 | 2. F. | 10th Sept. | | No | No | No | No | | | No | No | No |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Absent | No | | | | No | Purpuric spots on buttocks. | | No | On 23rd Aug.—Screaming "tetanic spasms." 24th. — "Fit," lasted 10 mins. |
| Absent | Complete paralysis both legs in 3 days. | No | No | No | No | No | No | No | Father a brush-maker. Came from Newton Abbot. |
| | Legs, arms and intercostal muscles. | | | | | | | | Died 9th Sept. |
| Patellar absent. | Paresis left arm followed by right leg and then by left leg. | No | No | No | No | No | No | C. | Left Bristol on 23rd Aug. |
| Patellar absent. | Paresis of legs | No | No | No | + | No | No | No | |
| | All four limbs and intercostal muscles. | | | Slight | | | | | Two cases in one house. |
| | Chiefly arms, to a lesser extent legs. | | | Slight | | | | | |
| | 2nd week rigidity and paresis muscles of back and legs. | | No | | No | No | | | Father a postman. Clean house. |
| | Evidence of muscular rigidity. | | No | | + | No | | | Clean house. |
| | | | | | | | | | |
| | | | | | | | | | |
| Plantar absent, patellar present. | Face, right side, lower part, and right leg; principally muscles supplied by ant-tibial nerve. | No | No | No | No | No | No | No | Clean house Slight numbness in right leg remains.—30th Sept., 1911. Marked giddiness at commencement of illness. |
| | Evidence of muscular rigidity. | | No | No | + | | | | Died 30th Aug. Clean house. |
| Normal | Paralysis left leg. | No | No | No | No | No | No | C. | Father an "Armorer." |
| Left patellar absent on 18th Sept. | Left foot slightly dropped on 18th September. | No | No | No | Slight | No | No | No | Mother stayed in Newton Abbot from 8th-15th Sept. Recovering on 18th Sept. Father a mastermariner at sea. |
| | | | | | | | | | |
| | | | | | | | | | |
| | Loss of power in left leg passing to complete paralysis in three days. | | No | | No | No | | | Father outdoor labourer |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 143 | 6, M. | 4th Sept. | 102° | + | | + of limbs. | No | In limbs. | | | No | No |
| 144 | 6, M. | 27th May | 101–103 | + | No | No | No | Over legs and back. | + | No | + | + |
| 145 | 1, M. | 29th June | + | + | No | No | No | + | + | + | + | + |
| 146 | 1½, M. | 8th June | 100–102·4 | + | No | No | No | Over back, abdomen and legs. | + | No | + | + |
| 147 | 16, F. | 18th June | Normal | No | No | No | No | Over left arm and shoulder. | No | No | No | No |
| 148 | 30, F. | 11th July | 99–103 | + | No | Twitchings. | + | Limbs and abdomen, pain in all joints. | + | + | No | + |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes. | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Normal | Inability to stand upright. | | No | Slight | No | No | | D. | Father a road-man. |
| Absent | Both arms, both legs. | No | No | No | + | No | + | C. | Condition on 14th September.—Left arm completely paralysed with the exception of the flexors of the thumb, ring and index fingers. Left leg completely paralysed. Right leg muscles of thigh normal; all muscles below the knee atrophied with the exception of the plantar muscles. Clean house. A week prior to child's illness a horse fell down in stable and was only raised with difficulty. As it could not stand it was shot without inspection by a vet. This was the first paralysis in Penryn. Stable near West Street. |
| + | Paralysis right deltoid and biceps. | No | No | No | No | Urticarial eruption. | No | C. | Atrophy of the deltoid muscle. |
| Absent | Both legs 14th June. Passed no urine for 36 hours and then for 24 hours. | + | No | No | No | Urticarial eruption. | No | C. | Clean house. Father, a grocer, called regularly at house of No. 144 for orders. Sept. 11th. — Recovered except peronei of left leg. |
| + | Deltoid left arm | + | + | + | No | Papular spots. | No | No | Works in a laundry. One sister works in a laundry, another, domestic servant at a school. No cases amongst pupils at this school. Recovered movement of arm. Clean house. |
| + | All four limbs. Right arm and pastic 23rd July. Urine and fæces passed involuntary. 28th July, well marked squint; left pupil more dilated than right. 30th squint disappeared. | No | No | + | + | Erythema over abdomen. | No | C. | Died 2nd August. Encephaloid type. No P.M. Death certificate signed "Intestinal toxæmia." Father a schoolmaster of a private school. Two other school masters of a national school live in house. Clean house. |

| No. | Age and Sex. | Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching or Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Drowsiness. | Kernig's Sign. | Tache Cérébrale. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 149 | 1 11/12, M. | 3rd Aug. | 100° | | No | No | No | Present over buttocks and legs. | + | No | No | No |
| 150 | 1 11/12, M. | 18th July | 98·4 | | No | No | No | No | + | No | + | + |
| 151 | 2 3/12, F. | 3rd Aug. | 100·2 | + | + | No | No | Present over abdomen and legs. | + | No | No | No |
| 152 | 7, M. | 3rd Sept. | | + | No | No | No | + | + | No | + | No |
| 153 | 2, M. | 19th Aug. | | No | No | | No | No | | + for a week. | No | No |
| 154 | 4, M. | 10th Sept. | 102°–105° | + | No | No | No | "Marked" | No | + for a week. | No | No |

| Condition of Reflexes. | Paralysis. | Epistaxis. | Discharge from Throat or Nose. | Sore Throat. | Retraction of Head and Neck. | Rash. | Herpes | Constipation or Diarrhœa. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| Absent right leg. | Anterior tibial and peronei of right leg. | No | No | No | No | No | No | D. | Numerous fleas. Very dirty house. Recovered except for peronei. |
| At first + lost later. | Left arm July 22nd, left leg July 26th, right side face, which disappeared in five days. | No | No | No | + | Urticarial eruption over abdomen for 4 days. | No | D. followed by C. | Father a farmer. Mother was on a visit to Truro and went into a shop where there was said to be a case of poliomyelitis. Here the mother of the affected case handled this baby. The people of the shop and this household have the same milk supply Clean house. Recovery of leg and partial recovery of arm. |
| Absent | Both legs, left arm | No | No | No | No | No | No | C. | Fleas on patient. Dirty house. Recovery of right leg. Paralysis of peronei and anterior tibial remains. 3rd October. — Left arm paralysed except for flexors of fingers. Father a yachtsman. |
| Absent | Both legs .. .. | No | Slight | No | No | No | No | C. | Flea-bitten. |
| | Weakness of legs and arms, no evidence of muscular rigidity. | | No | | No | No | | | Father a carter. Sanitary condition of house good. |
| Patellar, eft absent, plantar present. | Some loss of power in left arm, marked in grip. | No | No | No | No | No | Slight | D, then C. | Clean house. |

# No. 2.

## Report to the Local Government Board by Dr. Reginald Farrar on outbreaks of disease affecting the cerebro-spinal system occurring in the Midland Counties and in Dorsetshire in 1910.

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During the summer of 1910 cases of illness affecting the cerebro-spinal system and apparently infectious occurred in the City of Nottingham, in the Bingham Rural District, in the north-west portion of the Melton Mowbray Rural District of Leicestershire, adjoining the Bingham Rural District, and in the Melton Mowbray Urban District. Similar cases occurred during the same period in certain places in Leicestershire comparatively remote from the area principally affected.

In common parlance and in newspaper reports these cases were attributed to "spotted fever," and by the medical practitioners in attendance were, in the majority of instances, diagnosed during the outbreak as cerebro-spinal fever. Careful analysis of the outbreak has, however, led me to the conclusion, for which I shall presently give reasons in detail, that while certain cases of genuine cerebro-spinal fever did occur during the period under consideration in the City of Nottingham, the majority of attacks were properly referable to epidemic polio-myelitis, an infective inflammation affecting chiefly the anterior cornua of the spinal cord, sporadic cases of which are generally known under the name of "infantile paralysis."

Recognition of the true nature of the outbreak was probably prejudiced by the fact that "cerebro-spinal fever" was at an early stage of its occurrence made a notifiable disease in the Bingham Rural District and in the Melton Mowbray Urban and Rural Districts. It was not made notifiable in the City of Nottingham, but several cases reported to be of this nature came to the knowledge of the medical officer of health.

I obtained notes of altogether 83 cases of febrile illness affecting the cerebro-spinal system occurring in the counties of Nottingham and Leicester between the first of May and the 31st of October, 1910. As I was in close touch with all the medical officers of health and most of the practitioners in the affected area, and as the outbreak attracted a considerable amount of public attention, I think I have probably obtained information of all, or nearly all, the cases that occurred.

A tabular analysis of all such cases of which I could obtain information appears at the end of this report. The numbers preceding the initials of certain cases referred to in this report are the numbers given to these cases in the first column of the table. In addition to the cases actually notified to the medical officers of health of the districts affected I have included in my tabular

analysis certain other cases which it was agreed by the medical officer and myself were properly referable to the disease under consideration, but have excluded several cases brought to my notice of which the diagnosis appeared to be doubtful.

In practically all the cases brought to my notice, save those in which death had occurred before I commenced this inquiry, I visited the house affected, usually in company with the medical attendant, and personally obtained information from the patient and his or her relatives.

Of the 83 cases of which I obtained information—

| | | |
|---|---|---|
| 11 | occurred in | the City of Nottingham. |
| 50 | „ | the Bingham and Melton Mowbray Rural Districts. |
| 15 | „ | the Melton Mowbray Urban District. |
| 7 | „ | places in Leicestershire, remote from the area principally affected. |
| 83 | | |

Of these cases 14 were directly fatal, while one died of "bronchitis" in December, about 6 months after her original attack, when convalescence was apparently well established.

The majority of the cases not fatal may be comprehended under a single description; they conformed, with unimportant variations, to a special type, the symptoms of which resembled those of inflammation of the anterior cornua of the spinal cord (polio-myelitis) rather than those ordinarily associated with cerebro-spinal meningitis. This, which for convenience of description I shall call the "paralytic" type, occurred with remarkable uniformity of symptoms in a great majority of cases during the outbreak under consideration. The course of the disease in such cases was as follows:—Sudden and acute febrile onset with a temperature of about 102°; sweating; headache and pains in the neck and sometimes in the back and limbs; nausea, and, in most cases, vomiting; delirium and marked mental irritability, the patient being often drowsy during the day-time and restless and fretful at night; great muscular hyperaesthesia, so that any attempt to shift the patient's position occasioned cries of pain; twitching of the muscles, especially during sleep; Kernig's sign and *tache cérébrale* well-marked in practically all cases; loss of patellar, plantar, and abdominal reflexes; generally constipation; a petechial rash occurred in some cases; in several a slight general enlargement of the lymphatic glands was observed. In some cases there was rigidity of the muscles of the neck with some retraction of the head, and, in some cases, opisthotonos affecting the whole spinal column, the limbs being flexed.

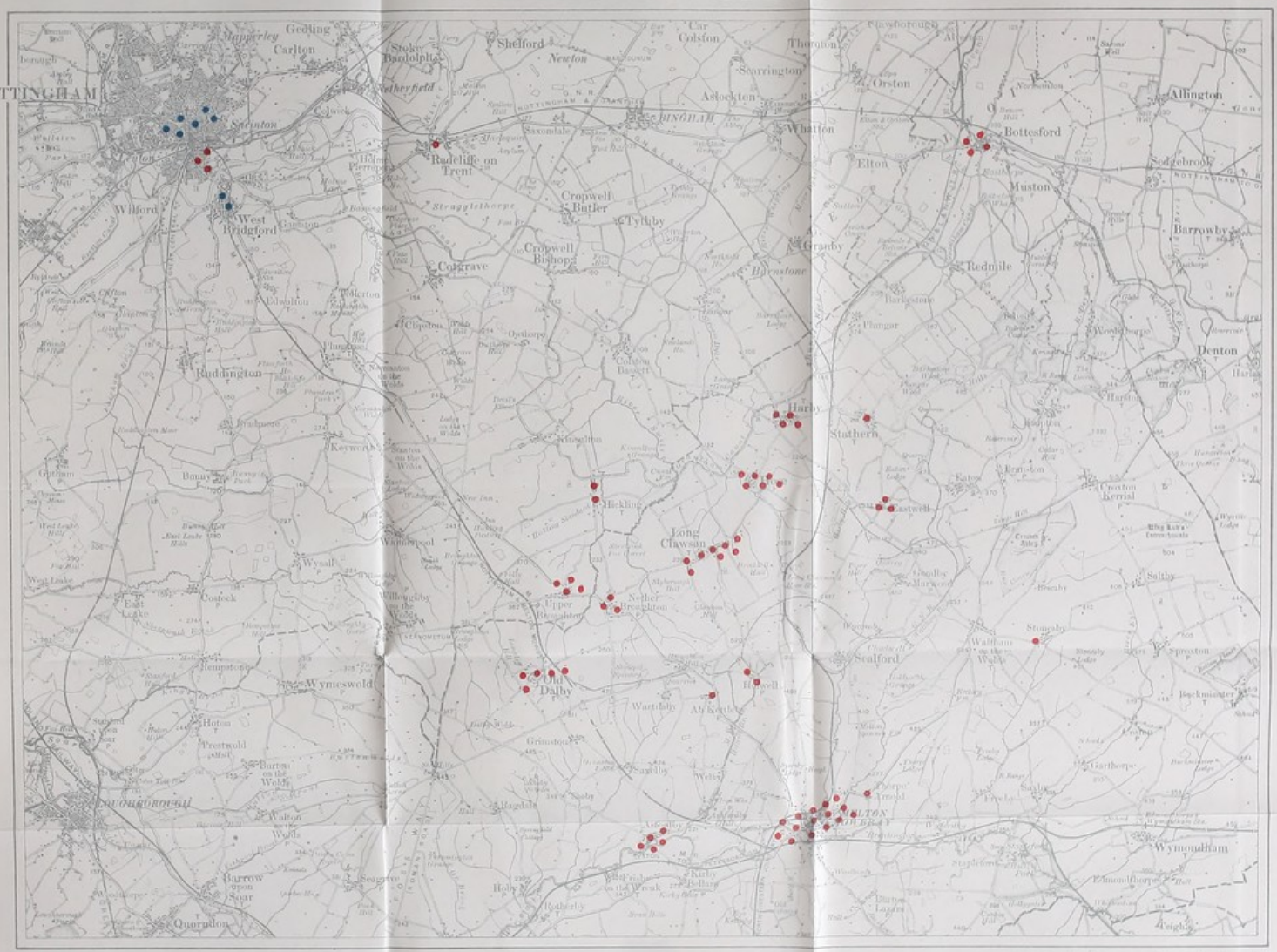
The febrile symptoms generally cleared up in the course of three or four days, sometimes less, and paralysis was manifest, usually about the time that the fever declined. In several cases, however, inability to turn in bed, pointing to some transient paralysis of the muscles of the trunk, was observed during the febrile stage. The paralysis affected sometimes the glosso-pharyngeal group of muscles, sometimes the oculo-motor muscles, causing squint, sometimes those of the neck, trunk, or arm, but in the majority of cases the

# MAP TO ILLUSTRATE DR. FARRAR'S REPORT.

Cases of Epidemic Polio-Myelitis marked Red.

" Cerebro-Spinal Fever " Blue.

SCALE—TWO MILES TO ONE INCH.






Printed at the Ordnance Survey Office, Southampton, 1911

muscles of the leg. In many instances the trunk muscles or those of both legs, or one leg and one arm, were at first paralysed, but usually the chief manifestations of paralysis subsided in the course of a few days, leaving only a limited group of muscles paralysed, or partly paralysed. In some cases the subsequent recovery from paralysis has been complete, in others a greater or less degree of paralysis persisted at the end of December, 1910, and a few of these last are still paralysed at the date of the issue of this report. In some households transient febrile attacks not causing paralysis, which may have been abortive cases of the same disease, occurred among the relatives of those attacked during the outbreak.

I shall discuss presently the question whether cases of the "paralytic" type occurring during this outbreak were due to meningococcus infection, or were cases of epidemic poliomyelitis, and attributable to a distinct infection.

The outbreak may now be considered in detail.

## 1. Nottingham Cases.

Though cerebro-spinal fever was not made notifiable in this city, Dr. Boobbyer, the medical officer of health, kindly furnished me with notes of 11 cases provisionally so diagnosed which were brought under his notice between May 1st and October 26th, 1910, and afforded me facilities for seeing some of these cases. Of these 11 cases one was found *post mortem* to be a case of streptococcus infection of the cerebro-spinal meninges secondary to chronic middle-ear disease, another was probably a case of tubercular meningitis. I have, therefore, not included these two cases in my table. Dr. Blurton has furnished me with notes of two cases, one fatal, the other mild, occurring in a single household at West Bridgford, a suburb of Nottingham, which appeared to be properly referable to epidemic disease of the cerebro-spinal system. I have included these latter in my table.

Including these two Bridgford cases, I have thus notes of 11 cases of this nature occurring in Nottingham. Of these 11 cases six were fatal: one was severe but terminated in complete recovery; one was of a mild "abortive" type; the remaining three cases, two of them being brothers, were of the "paralytic" type.*

Dr. Boobbyer informs me that he has reason to believe that other non-fatal cases which were not brought to his notice occurred in Nottingham during the period under consideration.

The information I have obtained as to the six fatal cases occurring in this city is as follows:—

1. *E. O.*, 15. *F.*—Attacked on April 27th with violent headache and vomiting. Delirium with hebetude supervened. May 1st, admitted to Nottingham General Hospital; seen by Dr. Jacob; temperature 104°; pulse irregular and unequal; severe pain in head and at back of neck; marked opisthotonos; hyperæsthesia; Kernig's sign; knee jerks and Achilles jerks absent; plantar reflexes variable, abdominal reflexes greater

* *Note.*—It may be noted that in one of these cases (9. *P. F.*) the blood agglutinated a culture of the meningo-coccus in a dilution of 1 in 20, but in respect of this micro-organism the agglutination test is thought to be of doubtful value.

on the right side than the left ; mottled rash fading on pressure ; there was an offensive discharge from one ear, but no tenderness on pressure over the mastoid process ; a radical mastoid operation was performed but nothing found to account for symptoms (no necrosis) ; lumbar puncture with injection of Flexner's serum was performed on several (at least 5) occasions and on each occasion with good temporary results ; while in the City Isolation Hospital, to which she was transferred on June 9th, the patient developed epileptiform convulsions (no previous history of epilepsy) haematemesis and haematuria, and died in convulsions on August 20th. The temperature throughout ranged from 99° to 104° and was "pyaemic" in character some days before death occurred.

The fluid extracted by lumbar puncture was a turbid poly-nuclear exudate in which a "gram-negative" diplococcus was discovered by Dr. Jacob.

3. *E. S.*, 29, *M.*—Fell ill on July 29th with violent headache, high fever, rigors, hyperaesthesia ; the temperature ranged between 100° and 105° ; Kernig's sign ; opisthotonos ; strabismus ; ecchymotic rash ; died in a state of coma August 2nd.

Lumbar puncture was performed during life and a "gram-negative" diplococcus found.

4. *P. C.*, $\frac{7}{12}$, *M.*—Nasal discharge five or six weeks before his attack, which apparently got better, then came on again. Attacked August 7th, after a fall from his cot ; delirium, moaning and occasionally screaming most of the night. Admitted Nottingham Children's Hospital August 8th; retraction of head and opisthotonos ; epileptiform convulsions ; strabismus ; paralysis of left arm and leg ; mottled rash ; leucocytosis ; temperature ranged from 99·4° to 102·8° ; lumbar puncture performed and a diplococcus found ; the retraction of the head ceased after lumbar puncture ; death in a state of coma August 9th.

5. *R.T.*, 12, *M.*—September 1st, acute febrile onset with headache, rigors, opisthotonos, and rigidity ; Kernig's sign and *tache cérébrale ;* patellar, plantar, and abdominal reflexes absent ; "pyæmic" range of temperature, over 105° at death ; lumbar puncture September 4th, Flexner's serum 20 c.c. injected ; developed epileptiform convulsions ; died September 6th.

A diplococcus found in the cerebro-spinal fluid, which was turbid and contained 85 per cent. of polymorphic leucocytes, 15 per cent. lymphocytes.

Had been in a Training Institute for Boys since July 26th. The other boys in the same dormitory were quarantined and kept under observation for 10 days. None of them developed the complaint.

6. *C. W.*, 27, *M.*—Attacked September 25th, admitted Nottingham General Hospital October 1st, transferred to City Isolation Hospital October 4th ; headache ; marked opisthotonos ; Kernig's sign and *tache cérébrale* well-marked ; muttering delirium ; range of temperature 101° to 105° ; death in a state of coma October 18th.

Lumbar puncture was performed during life and a diplococcus found in the cerebro-spinal fluid.

7. *A. B.*, 2, *F.*—Attacked July 24th. Headache ; vomiting ; delirium; twitching of arms : retraction of head and marked opisthotonos ; died July 30th.

A sister of this patient, 8, *C. B.*, passed through a mild abortive attack a few days earlier.

A "gram-negative" diplococcus was discovered in the spinal fluid extracted by lumbar puncture from the following case which terminated in complete recovery :—

2. *E. H.*, 9, *F.*—Admitted to Nottingham General Hospital after a fall from a wall on May 31st ; transferred to City Isolation Hospital on June 9th ; discharged cured on July 27th. Temperature of intermittent febrile type up to July 11th and after that date even and subnormal. The symptoms during the acute stage of her illness comprised headache ; twitching and convulsions ; hyperaesthesia and irritability ; retraction of the head and spinal opisthotonos. Kernig's sign was present and tendon reflexes were absent. Slight paraplegia and strabismus were observed. Lumbar puncture

was performed on several occasions. A "gram-negative" diplococcus was found in the spinal fluid. Recovery was eventually complete, all the above symptoms clearing up by July 11th.

Thus, six cases occurred in Nottingham during the period under consideration, in which a "gram-negative" diplococcus was found in the fluid extracted by lumbar puncture. Five of these cases were fatal, and the symptoms in all were those characteristic of cerebro-spinal fever. The diplococcus found was morphologically identical with the *meningococcus* of Weichselbaum, and though it was not isolated and identified by cultural tests, it is probable that it was the same organism. The seventh case, 7. A. B., was a fatal case and the symptoms were compatible with the diagnosis of cerebro-spinal fever. A sister of this patient suffered from what seems to have been a mild abortive attack of the same disease.

Consideration of the facts has led me to the conclusion that the above were cases of true cerebro-spinal fever, specifically distinct from the cases of "paralytic" type that occurred during the same period in the Vale of Belvoir and in other parts of Leicestershire, and which I believe to have been cases of epidemic polio-myelitis. I shall therefore exclude them from further consideration in connection with the latter outbreak, together with case 83. A. W., that of a young servant girl, who was attacked at Barlestone and died in the Leicester Infirmary, and whose symptoms were considered to be those of fulminant cerebro-spinal fever.

## 2. Other Midland Cases.

There remain 74 cases (including three cases of "paralytic" type occurring in Nottingham) which may now be separately considered.

They were distributed as follows :—

| | |
|---|---|
| Bingham and Melton Mowbray Rural Districts ... | 50 |
| Melton Mowbray Urban District ... ... | 15 |
| Belvoir Rural District ... ... ... ... | 4 |
| Nottingham City ... ... ... ... ... | 3 |
| Atherstone Rural District ... ... ... | 2 |
| | 74 |

Sixty-one of the above cases occurred within an area of about 50 square miles, viz., 15 in the Melton Mowbray Urban District and 46 in a chain of villages in the Bingham and Melton Mowbray Rural Districts. The area thus enclosed is roughly elliptical in shape, and has the town of Melton Mowbray at its southern extremity. It comprises a considerable portion of the country known as the Vale of Belvoir. Two cases occurred at Stonesby and one at Sproxton, villages in the Melton Mowbray Rural District, slightly outside the above-mentioned area, one at Radcliffe-on-Trent in the Bingham Rural District (26. B. M. : the infection in this case was almost certainly contracted in Hose), four at Bottesford, in the Belvoir Rural District, the western boundary of which is contiguous with the Melton Mowbray Rural District, and three in Nottingham, which is contiguous on the south with the north-western corner of the Bingham Rural District. The two remaining cases occurred at Oakthorpe, in the Atherstone Rural

District, which is in Leicestershire, but at a considerable distance from the area principally affected.

Within the Bingham and Melton Mowbray Rural Districts cases occurred in the following parishes :—

| | Cases. | Fatal. |
|---|---|---|
| Eastwell ... ... | 3 | — |
| Harby ... ... | 4 | — |
| Hose ... ... ... | 6 | 1 |
| Old Dalby ... ... | 5 | 2 |
| Long Clawson ... | 7 | — |
| Upper Broughton ... | 4 | 1 |
| Nether Broughton ... | 3 | — |
| Hickling ... ... | 3 | — |
| Holwell ... ... | 2 | — |
| Asfordby ... ... | 5 | 1 |
| Stonesby ... ... | 2 | — |

Stathern, Radcliffe-on-Trent, Kettleby, Grimstone, Sproxton, and Thorpe Arnold, one case each, those at Radcliffe-on-Trent and at Kettleby being fatal cases.

It may here be noted that six of these patients lived in lone farm lodges remote from other dwellings.

As will be seen from the dates of attack given in the tabular statement of cases the invasion of the area affected seems, as regards the earlier cases, to have been of an "explosive" character, attacking several villages almost simultaneously, rather than a successive invasion of village after village; but within the villages invaded many of the cases afford strong evidence of infection spreading from one patient to another. The question of infectivity in this outbreak will be separately discussed.

Of the above 74 cases seven were directly fatal, while one child (27. *O. S.*) died of "bronchitis" in December when convalescence was apparently well established. Two cases were relatively severe, one of them terminating in complete recovery, while in the other convalescence was prolonged and was incomplete at the date of writing. The remaining 64 cases were practically all of the "paralytic" type characterised by an acute febrile stage, more or less severe but of brief duration, seldom exceeding three or four days, with subsequent motor paralysis, affecting usually the muscles of the leg below the knee. In 37 of these cases recovery was complete by the end of December, in 24 there was still at that date a greater or less degree of paralysis with wasting of the affected muscles which seemed likely to be permanent.* As regards the three remaining cases my information is incomplete.

The cases in point of time occurred as follows :—

7 in June.
20 „ July.
34 „ August.
9 „ September.
4 „ October.

* Since drafting this report I have ascertained that some degree of paralysis has persisted in several of these cases down to September, 1911.

The following clinical notes illustrate the course of the disease in some of the fatal and of the more severe cases, as well as in some of the milder cases of paralytic type.

21. *M. S.*, 6, *F. Hose.*—Attacked *July* 31*st*, headache, fever, vomiting, twitching, sweating; *August* 3*rd*, pains in the legs and loss of power in the right leg; Kernig's sign and *tache cérébrale* well marked; knees drawn up and head drawn forward; great rigidity; *August* 7*th*, lumbar puncture and injection of Flexner's serum, 15 c.c., which appeared to relieve the pain and rigidity, temperature ranging from 100° to 103°; *August* 8*th*, rigidity gone, pain relieved; respiration 60, pulse 130 feeble, temperature 97·6°; *August* 10*th*, died of "broncho-pneumonia." This child was a first cousin of 20 *E. C.*, who was attacked on July 24th. The children were playmates and used to exchange visits. She was also a playmate of 22. *W. C.* She had attended a Band of Hope tea at Hose on July 25th.

28. *S. S.*, 36, *F. Old Dalby.*—Attacked *August* 12*th*, pains in head, back, and limbs; slight stiffness in the neck; temperature 100°; pulse 112; loss of knee-jerks; Kernig's sign well marked; *tache cérébrale.* For some days continued to work and refused treatment. *August* 16*th*, temperature 100°, but said she felt better and did a day's washing, still refusing treatment; *August* 18*th*, attacked with "peritonitis"; *August* 19*th*, died. This woman is the mother of 27. *O. S.*, 7. *F.*, who was attacked on *June* 24*th*. The child, who slept in the same bed with her father and mother, passed through an acute but brief febrile attack, and developed paralysis of both legs. There was still weakness and some wasting of the muscles of the legs, but the child had otherwise recovered when she was attacked with bronchitis in December and died. The bronchitis was thought to be independent of her original attack. When tested in September the child's blood gave some, though incomplete, evidence of agglutination with meningo-coccus culture. The house was the village post-office.

29. *C. T.*, $1\frac{7}{12}$, *F. Old Dalby.*—July 26th, acute febrile onset, twitching, nystagmus, convulsions. The legs "seemed to give way." July 27th, died in coma before the arrival of the medical attendant. The child lived in a lone lodge, and so far as I could ascertain had not come into contact with other cases.

40. *J. W.*, 6, *M. Upper Broughton.*—Said to have had a "bilious attack" on July 26th from which he apparently recovered. Seemed to be quite well on July 30th and was playing all day in the hay-field with 41. *L. P.* (who was attacked on August 6th). He appeared to be well on the morning of July 31st, but was attacked in the afternoon of the same day with acute cerebral symptoms and quickly became comatose When seen by Dr. Atkinson he was comatose and twitching all over, and died within half-an-hour of his visit. The child returned on July 20th from a visit to West Bridgford. The other children of the family escaped attack.

49. *F. G.*, 5½, *M. Kettleby.*—Acute febrile onset with pains in the limbs and joints; temperature 104°; lay with knees drawn up; marked hyperæsthesia and irritability; died July 29th; there was no heart lesion; there was a small patch of pneumonia but this was not, in the opinion of the medical attendant, sufficient to account for death. The case was not at the time recognised as one of polio-myelitis, but in the light of subsequent events appears to have been of this nature. A woman (51. *A. G.*) who attended the funeral on August 1st and mixed with the crowd round the coffin was attacked on August 9th; a schoolmate of *F. G.'s* (50. *D. H.*) who had been closely associated with him in school a day or two before his attack was attacked on August 7th. Both these latter cases were of the "paralytic" type.

Two months previously a sister of *F. G.* suffered from "pneumonia" with a good deal of spinal pain.

37. *C. S.*, 15, *M. Long Clawson.*—Attacked *August* 23*rd* with headache and vomiting, *August* 24*th*, temperature 102°; pulse 100; rigidity of neck with some retraction; Kernig's sign; *tache cérébrale;* absence of reflexes; in the evening temperature 104°; lumbar puncture and injection of Flexner's serum 15 c.c.; *August* 25*th*, temperature 103°; pulse 100;

paralysis of pharynx, difficulty in swallowing, saliva running out of mouth; respiration jerky, 16 to 20, of "Cheyne Stokes" character; twitching, especially of the face; delirium; great dyspnœa, suffocation feared; second lumbar puncture and injection of Flexner's serum 15 c.c.; 7.30, temperature 100°; breathing easier; *August 26th*, temperature 99°; respirations 20–28, jerky; peculiar *tache cérébrale*, consisting of a thin red line with a broad white margin, the *tache* having a breadth of some half an inch; still difficulty in swallowing, fed by tube and with nutrient suppositories; *August 28th*, temperature 98°; partial right ptosis and squint; at 6.30 Dr. Jacob did a third lumbar puncture. Paralysis of the pharynx persisted for several weeks, necessitating artificial feeding; there was incontinence of urine for several days; inability to articulate, ptosis, facial paralysis and paralysis of both legs; there was considerable emaciation and convalescence was protracted; at the end of December there was still left facial paralysis, and such a degree of paralysis in both legs that he was unable to stand.

This patient occupied the same bed with his brother, 36, *L. S.*, who was attacked three days earlier, viz., on August 20th, and passed through a mild attack of the "paralytic" type, terminating in complete recovery.

42. *J. E.*, 37. *M.*, *Upper Broughton.*—Attacked *August 26th*, febrile onset with headache and vomiting; *August 28th*, seen by Dr. Atkinson; headache; rigidity of neck; Kernig's sign; *tache cérébrale*, absence of reflexes; enlargement of inguinal, axillary, and cervical glands; temperature 102°; lumbar puncture and Flexner's serum 15 c.c. injected; *August 29th*, violent headache and vomiting, retraction of head, general stiffness and rigidity of spine; temperature 102°; Flexner's serum again injected into the spinal canal, the spinal fluid was opalescent and under pressure; *August 30th*, temperature 99°; expressed himself as relieved by the injections; the rigidity had passed off; *August 31st*, a third lumbar puncture; *September 1st*, temperature 98°; slight headache and delirium, but general improvement of all symptoms; there was general weakness of the legs during the attack, and he was unable to turn over in bed. The patient made a complete recovery and by the middle of December he had resumed his work as an ironstone labourer. He had attended at Dr. Atkinson's surgery in Long Clawson, on Augut 22nd, four days before his attack.

47. *L. W.*, 6, *F. Hickling.*—*August 13th*, acute febrile onset with headache, vomiting, and twitching; *August 17th*, seen by Dr. Atkinson, temperature 99·4°, pulse 120, delirium, Kernig's sign, *tache cérébrale*; knee jerks and chest reflexes absent, plantar reflex extends; *August 18th*, temperature 103·2°, pulse 160, aggravation of pain and delirium; lumbar puncture and Flexner's serum 15 c.c. injected; the fluid was turbid and under high pressure—squirted out from the trocar; *August 19th*, temperature 99°, pulse 120; retraction of head noted; *August 20th*, temperature 99°, pulse 120; feels better, no pain; *August 21st, 22nd and 23rd*, temperature 97°; paralysis of muscles of neck, unable to lift head from the pillow save by raising the shoulders and swinging it up; slight paralysis of trunk and limbs; glands of neck, axilla and groin enlarged; *September 1st*, able to sit up in bed; *September 10th*, able to walk; convalescent but still weak.

Recovery was complete before the end of the year and no trace of paralysis remained.

The spinal fluid was turbid, contained leucocytes and polymorphonuclear cells, albumen, but no sugar.

71. *L. G.*, 3, *M. Melton Mowbray.*—Attacked *October 1st;* medically attended *October 3rd;* acute febrile onset, temperature 99·9°; apparent headache; vomiting; hyperæsthesia; irritability; twitching during sleep; very restless at night; difficulty in swallowing supervened, with regurgitation of food through nose. (The boy had diphtheria in the summer and was at first thought to be suffering from diphtheritic paralysis).

Admitted to Melton Isolation Hospital, *October 7th.* Slight retraction of head; back slightly arched, neck and back rigid "moved all in one piece;" left facial paralysis; pupils uneven; glosso-pharyngeal area insensitive; abdominal reflexes abolished, patellar reflexes diminished; Kernig's sign well marked; rash over back and buttocks, a well-marked clearly defined eruption, not raised, spots varying in size from a lentil to a

split pea, some cherry red, others dark mulberry, not fading on pressure ; *October 8th*, absolute regurgitation of food necessitating nasal feeding ; definite opisthotonos ; rosy spots appearing on shoulders, rash on lower part of trunk fading ; *October 10th*, signs of paresis, both lower limbs ; "Cheyne Stokes" breathing ; *October 13th*, all active and acute symptoms have passed off, but there is still some weakness of the back and lower limbs, especially of the right leg.

*December 13th*, recovery apparently complete. 70. *W. M.*, a next-door neighbour and playmate was attacked on August 18th, and it is not improbable that *L. G.* contracted the disease from this child ; on the other hand *L. G.* has five brothers and sisters, one of whom, a boy of four, is his bed-fellow ; none of them contracted the disease.

74. *A. G.*, 33, *F.*—Was rather "run down" previously to her acute attack. *August 31st*, acute febrile onset, temperature 102° ; headache and pain in the back of the neck ; vomiting ; febrile symptoms lasted about three days; *September 3rd*, inability to articulate or to swallow food ; regurgitation of food through nostrils ; no Kernig's sign ; no loss of knee-jerks ; right facial paralysis ; palate insensitive ; *December 21st*, can swallow liquids with difficulty, but not solids. Has three children, none of whom were affected.

76. *H. E.*, 16, *F.*—Mentally deficient since birth. *September 13th*, headache, pain in back ; vomiting ; inability to swallow food ; *September 19th*, admitted to Melton Isolation Hospital ; drowsy and stupid ; brows knitted ; left internal squint ; pupils unequal, left dilated and sluggish ; twitching ; Kernig's sign ; *tache cérébrale ;* marked opisthotonos, lumbo-dorsal arch accentuated ; patellar reflexes exaggerated ; hyperæsthesia ; inability to articulate or to swallow food ; loss of control over sphincters. *September 23rd*, all reflexes normal, but Kernig's sign persists.

Discharged convalescent at the end of four weeks.

*December 21st.* Recovery complete.

Has two brothers, neither of whom was affected.

It has been already stated that the outbreak was originally regarded by most of the medical practitioners in the areas affected as one of cerebro-spinal fever, and that cerebro-spinal fever was at an early stage made notifiable, under the provisions of the Infectious Diseases Notification Act, in the Bingham and Melton Mowbray Rural and the Melton Mowbray Urban Districts. This circumstance together with the fact that cases were simultaneously occurring in Nottingham which appear to have been cases of true cerebro-spinal fever, and in which a "gram-negative" diplococcus was discovered in the spinal fluid withdrawn by lumbar puncture, has probably prejudiced the view taken of the nature of the outbreak.

I have already indicated that nine cases—viz., eight cases occurring in Nottingham and one at Barlestone—may be regarded as presumably cases of true cerebro-spinal fever, leaving 74 cases—occurring for the most part outside the city of Nottingham, which seem to belong to a different category.

Of these 74 cases, those that proved rapidly fatal and some of the more severe non-fatal cases could not on clinical grounds alone be readily differentiated from cases of true cerebro-spinal fever, but the large majority (say 65) were of a milder type, which if occurring sporadically certainly would not have been so regarded. These latter cases were characterized by a brief febrile onset followed by paralyses of longer or shorter duration, but were associated, both in respect of time and of circumstances affording chances of infection, with the more severe cases (resembling cerebro-spinal fever) in such a manner as to make it appear highly probable that both types were due to the same infection. I regard these 74 cases

as a group constituting an outbreak of epidemic poliomyelitis on the following grounds :—

1. The febrile onset was comparatively short and mild, and the signs of meningeal irritation were less severe than is ordinarily the case in outbreaks of cerebro-spinal fever.

2. Practically all the cases were characterized by a selective action of the infective virus upon the anterior cornua of the spinal cord, resulting in paralyses of muscles or groups of muscles in the upper or lower limbs. This feature is characteristic of epidemic polio-myelitis, but does not commonly occur in epidemics of cerebro-spinal fever.

3. The fatality of the outbreak was only 10·8 per cent. (if we include the death of 27. O. S., who died of "bronchitis" when convalescence was apparently well established). This fatality-rate would be exceptionally low for an outbreak of cerebro-spinal fever, but is such as commonly occurs in outbreaks of epidemic polio-myelitis.

4. The high incidence of the disease on children is characteristic of polio-myelitis rather than of cerebro-spinal fever.

The ages of those attacked were as follows :—

| | Cases. |
|---|---|
| Under 1 year old ... ... ... ... | 0 |
| Over 1 and under 5 years old ... ... ... | 31 |
| „ 5 „ 10 „ ... ... ... | 21 |
| „ 10 „ 15 „ ... ... ... | 10 |
| „ 15 „ 20 „ ... ... ... | 4 |
| „ 20 „ 40 „ ... ... ... | 7 |
| „ 40 „ 50 „ ... ... ... | 1 |
| | 74 |

Thus 52, equal to a rate of 70·3 per cent. of all cases occurred in children under 10 years of age, and 62, equal to a rate of 83·8 per cent. of all cases in children under 15.

The high rate of attack among children cannot be attributed wholly to school infection (though this factor may perhaps have played some part at the beginning of the outbreak), as the majority of cases occurred during the months of August and September, during which time the schools in the Melton Mowbray Urban and Rural Districts and the Bingham Rural District remained closed. On the other hand several school treats, flower shows, galas, &c., occurred in the affected area during the month of August, some of which occasioned the close aggregation of young children, and in several instances attacks of the disease have been attributed with a reasonable degree of probability to infection contracted on such occasions.

5. The outbreak occurred during the summer months. This circumstance corresponds with the usual seasonal prevalence of epidemic polio-myelitis, whereas outbreaks of cerebro-spinal fever usually occur in the winter.

6. In most of those attacked the clinical *ensemble* (particularly in the milder cases, which constituted the great majority) was that of epidemic polio-myelitis rather than of cerebro-spinal fever.

## Nature and Degree of Paralysis observed during the Outbreak.

Of the non-fatal cases, recovery from paralysis was stated to be complete at the end of December in 37 instances; in 24 some amount of paralysis was persistent at this date. As regards the few remaining cases, I have been unable to obtain definite information.

As above mentioned, the muscles paralysed were various: paralysis of the muscles of the neck and trunk was a transient symptom in several cases, but in no instance proved permanent; transient paralysis of the sphincters of the rectum and bladder was observed in a few cases; in four cases there was paralysis of the muscles of the glosso-pharynx with inability to articulate or to swallow; (at the end of December, in one of these cases, only liquids could be swallowed and this affection threatened to become permanent; in the others the paralysis had passed off). Strabismus was noted in 14 cases; facial paralysis in 6; paralysis of the arm, chiefly of the deltoid muscle, in six. In by far the largest number of cases paralysis occurred in the legs, the anterior tibial and peroneal groups of muscles being those most frequently affected.

In nearly all cases tendon-reflexes, particularly the patellar reflexes, were in abeyance.

*Cases in which a rash was observed.*—Herpes labialis, which is often observed in similar outbreaks, was in this outbreak observed in two cases only, viz., 10. *F. C.* and 16 *T. W.* In view of the fact that this outbreak was popularly known by the name "spotted fever," it will be interesting to note the cases in which any form of skin affection was observed.

These cases were 17,* as follows:—

[N.] 1. *E. O.*, "mottled rash fading on pressure."
[N.] 3. *E. S.*, "ecchymotic rash."
[N.] 4. *P. C.*, "mottled rash, no ecchymosis."
[N.] 6. *C. W.*, "mottled rash."
10. *F. C.*, "herpes labialis."
16. *T. W.*, "eruption on trunk resembling varicella, herpes on lips."
23. *G. A.*, "petechial rash."
26. *B. M.*, "rash on extremities."
34. *R. G.*, "rash resembling that of enteric fever."
35. *A. R.*, "covered from top to toe with an eruption of dull red spots resembling those of enteric fever, fading on pressure."
38. *P. M.*, "erythematous patches and blotches all over body."
50. *D. H.*, "petechial rash on chest and abdomen."
58. *H. B.*, "well-marked brownish spots all over back, not fading on pressure."
66. *P. B.*, "indefinite rose spots on abdomen."

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* *Note.*—The Nottingham cases presumed to be cerebro-spinal fever are included, but marked [N.].

71. *L. G.*, "eruption of rose spots, not fading on pressure, over shoulders, back, and buttocks."

Thus, in 17 cases, of which four (viz., Cases 1, 3, 4 and 6) are regarded in this report as cases of cerebro-spinal fever, some form of rash was observed.

## Infectivity in this Outbreak.

From the public health standpoint the most important matter for consideration is the degree of infectivity of the disease.

Incidence of the disease upon "contacts" during the outbreak under consideration occurred in a fashion so irregular as to appear almost capricious; the closest association, even sleeping in the same bed, often failed to communicate the disease among members of the same family, while on the other hand under some circumstances the disease seemed to be communicable in a relatively high degree; the facts appeared also to indicate that unrecognised "carrier" cases may play an important part in the transmission of the disease.

Dual attacks, involving 14 cases occurred in seven houses (equal to 10·4 per cent. of all the houses invaded), viz.—

| | | | |
|---|---|---|---|
| 10. F. C. | 13. F. G. | 27. O. S. | 30. A. G. |
| 11. S. C. | 14. M. G. | 28. S. S. | 31. F. G. |
| 36. L. S. | 77. D. B. | 79. F. M. | |
| 37. C. S. | 78. C. B. | 80. A. M. | |

The following instances illustrate attacks of "paralytic" type affecting brothers and sisters at short intervals:—

10. *F. C.*, attacked September 3rd; his brother, 11. *S. C.*, September 10th.

30. *A. G.*, attacked August 24th; his sister, 31. *F. G.*, August 31st; a third child in this family was thought to have had a mild abortive attack in the same week.

77. *D. B.*, attacked September 5th; her brother, 78. *C. B.*, on September 9th.

79. *F. M.*, attacked June 6th; his sister, 80. *A. M.*, June 8th.

In other instances cases of "paralytic" or abortive type have been associated with severe or fatal cases.

36. *L. S.* was attacked on August 20th, a mild, almost abortive, case of "paralytic" type; his brother, 37. *C. S.*, who slept in the same bed, on August 23rd, his illness being very severe.

In two instances there were considerable intervals between attacks in the same house.

13. *F. G.* Sudden appearance of facial paralysis on July 5th; his mother 14. *M.G.* was attacked on August 24th.

27. *O. S.* was attacked on June 24th; the case was of "paralytic" type, but the child died of bronchitis, not, apparently, connected with the original disease, in December; she slept in the same bed with her mother, 28. *S. S.*, who was attacked on August 12th and died on August 19th.

Several instances occurred in which children who were playmates and near or next-door neighbours contracted the disease within shorter or longer intervals of one another.

It has already been mentioned that 20. *E. C.* attacked July 24th, 21. *M. S.* attacked July 25th, and 22. *W. C.* attacked August 3rd, were playmates. *E. C.* and *M. S.* were first cousins, and also attended the same Sunday school class.

18. *M. C.* and 19. *A. M.* of Harby, were both attacked on August 13th. The children are next-door neighbours and playmates. Both went to Clawson Flower Show on August 11th, when owing to heavy rain a large number of children were closely crowded together in a small tent. *M. C.* often visits Hose to see her aunt, who is a washerwoman there, and on these occasions *M. C.* plays with some of the Hose children.

44. *E. M.* attacked July 25th and 45. *B. G.* attacked August 1st, both of Nether Broughton, are first cousins and often meet and play together.

62. *R. R.* attacked at some time in June, and 63. *J. M.* attacked August 5th, both of Melton Mowbray, are next-door neighbours; his sister frequently, and *J. M.* himself occasionally, used to visit the home of *R. R.* and play with her.

67. *J. T.* attacked July 27th and 68. *F. S.* attacked July 30th, both of Melton Mowbray, live within a few doors of one another, and are constant playmates.

70. *W. M.* attacked August 18th, and 71. *L. G.* attacked October 1st, both of Melton Mowbray, are next-door neighbours, and frequently play together in the garden.

65. *H. C.* attacked June 23rd, and 66. *P. B.* attacked September 5th, both of Melton Mowbray, lived in the same small yard and were frequent playmates.

81. *F. M.* and 82. *C. N.*, both of Bottesford, are next-door neighbours. During *F. M.*'s illness *C. N.* eluded her mother and ran in to see him: two days later she had a febrile attack followed by paralysis of one leg.

The clearest suggestion of infection between playmates was afforded by the case of 40. *J. W.* and 41. *L. P.*, both of Upper Broughton.

*J. W.* (who returned from a visit to West Bridgford on July 20th) had a "bilious attack" on July 26th, from which he apparently recovered. On July 30th he was playing all day in the hayfield with *L. P.*, the children rolling and tumbling over one another. On July 31st *J. W.* was again attacked with acute symptoms, passed rapidly into a comatose condition, and died in a few hours: his companion, *L. P.*, was attacked with acute febrile symptoms on August 6th, and developed paralysis of the deltoid, which still persisted at the end of December.

The following cases in which there was definite evidence of exposure to infection may also be quoted:—

26. *B. M.*, 13, cycled from Radcliffe-on-Trent, a distance of about 10 miles, to visit her grandmother at Hose on July 24th, returning the same evening. The grandmother, Mrs. M., resides next door to the home of 23. *G. A.* who was attacked on July 20th. The A. children are constantly "in and out of" Mrs. M.'s house, and one of the children, E. A., who appears to have had a slight abortive attack, was ill and asleep in a chair in this house during *B. M.*'s visit. *B. M.*, who had thus cycled some 20 miles on July 24th, was attacked on July 31st and died on August 5th.

72. *R. S.* was governess to 54. *S. Y.*, who died on August 24th. She last gave him a lesson on August 17th, when he was evidently "sickening"; on August 18th the child was definitely attacked with acute symptoms. *R. S.* was herself attacked with febrile symptoms on August 22nd.

The four following instances appeared to point to the possible influence of "carrier" cases.

On August 1st, 51. *A. G.* attended the funeral of 49. *F. G.* who died on July 29th, and mingled with the crowd round the grave, in close contact with the relatives of *F. G.*, among whom may have been a "carrier." *A. G.* was attacked on August 9th. As she seldom leaves her home, it seems not unlikely that she contracted the infection on the occasion of the funeral.

73. *L. T.* resides a little way outside Melton Mowbray. The last occasion before his illness that he was in the town was on August 25th, when he mixed with a dense crowd, watching a wedding procession outside the church. He was attacked on August 31st, not having been into the town in the meanwhile.

21. *M. S.* of Hose died on August 10th. On August 14th her father visited Mr. S. of Long Clawson, the father of 36. *L. S.* and and 37. *C. S.*, who is a correspondent of a local newspaper, to give him some particulars of his daughter's death. As the two men were writing together at the table, *L. S.* leaned for some minutes over his father's shoulder between them, and his mother noticed the circumstance. *L. S.* was attacked on August 20th, and his brother *C. S.* who sleeps in the same bed on August 23rd.

13. *F. G.*, attacked July 5th, and his mother 14. *M. G.*, attacked August 24th, reside in a remote farm lodge in Eastwell parish, which they seldom leave, but *F. G.'s* brother B. G. used to play with 12. *W. W.* (attacked June 12th) and had wheeled *W. W.* about in his mail-cart while he was convalescent from his attack.

In several other instances cases were connected one with another, or through a third person, by demonstrable associations which though individually of too slender a nature to be adduced as positive evidence of infection seemed collectively too significant to be dismissed as the mere coincidences of village life.

It is reasonable to suppose that the infection may be conveyed as a "droplet" infection.

In outbreaks of cerebro-spinal fever the meningococcus has been in some instances found in the noses and throats of patients and "contacts." I saw comparatively few cases during the acute stage of their illness, but sent several recently-taken swabs from the throats of convalescent patients and "contacts" to Dr. Mervyn Gordon for examination, with negative results as regards the meningococcus which would probably have been found in some of these swabs had the outbreak been one of cerebro-spinal fever.

The theory has been adduced that the disease may be transmitted by fleas. I could obtain no evidence in support of this theory beyond the fact that a few children were said to have been flea-bitten within a few days before their attack, and it was conjectured that infective fleas were contracted on the occasion of certain popular gatherings, galas, &c. On the other hand it is equally possible that the

infection was communicated in the form of a "droplet" infection by "carrier" cases on such occasions.

In view of the fact that the outbreak affected several villages almost simultaneously, and that certain attacks occurred in remote farmhouses under which the chances of personal infection from other cases were not obvious, it has been suggested that infection might have been spread, possibly through the agency of flies, by tub-closet manure from Nottingham, which is spread by some farmers over their fields in the districts affected. I know of no positive evidence in support of this theory, but it is certainly the fact that a large amount of tub-closet manure has been sent from Nottingham into the district affected.

I am indebted to the medical officer of health for Nottingham for the following particulars of night soil sent to the Melton district from April to September :—

| Station. | Date. | Quantity. |
|---|---|---|
| | | Tons. |
| Old Dalby ... ... ... ... | April ... ... ... | 104 |
| " ... ... ... ... | May ... ... ... | 98 |
| " ... ... ... ... | September ... ... | 320 |
| Long Clawson ... ... ... | April ... ... ... | 34 |
| John-O'-Gaunt ... ... ... | June ... ... ... | 50 |
| Waltham ... ... ... ... | May ... ... ... | 50 |
| " ... ... ... ... | June ... ... ... | 40 |
| " ... .. ... ... | September ... ... | 16 |
| Edmonthorpe and Wymondham ... | April ... .. ... | 8 |
| Grimston ... ... ... ... | " ... .. ... | 16 |
| Eaton Siding ... ... ... | " ... ... ... | 32 |
| " " ... ... ... | May ... ... ... | 24 |
| Saxby ... ... ... ... | April ... ... ... | 16 |

I have made enquiries into the prevalence of infantile paralysis in Nottingham during recent years, and have obtained the following figures showing the number of cases of infantile paralysis admitted to the Nottingham Children's Hospital since 1906, viz. :—

| | | |
|---|---|---|
| 1906 ... ... ... ... | 25 | cases. |
| 1907 ... ... ... ... | 21 | " |
| 1908 ... ... ... ... | 38 | " |
| 1909 ... ... ... ... | 26 | " |
| 1910 ... ... ... ... | 49 | " |

The number of cases, 49, admitted during 1910 thus shows a significant increase over the average of the four preceding years, 27·5.

In view of the fact that both cerebro-spinal fever and epidemic polio-myelitis can be experimentally communicated to animals, the following information, kindly communicated to me by Dr. Kentish Wright, Resident Medical Officer of the Bagthorpe Isolation Hospital, Nottingham, is worth recording.

In December last he saw at Upper Broughton (one of the affected villages) one of last season's lambs which was partially paralysed in its hind legs, the right being slightly worse than the left. It seemed unable to clear the ground as it walked, and when running down hill both legs occasionally gave way. The lamb (one

of twins) was apparently quite normal at birth, and the paralysis was first noticed after three or four days. It was then hardly able to stand, so that considerable improvement had taken place between the onset of the disease and Dr. Kentish Wright's observation of the animal. The farmer who owned the animal stated that he had two other lambs similarly affected during 1910, but they were much worse and died or were slaughtered. He said that cases of "ricketty" lambs occurred every year and were frequently to be seen in Melton Market where they were sold to the butchers. He had never heard of an adult sheep being affected and could not say whether there were more cases than usual this year.

*Pathology.*

The pathological results obtained in this outbreak cannot be regarded as conclusive.

In four cases (42. *J. E.*, 37. *C. S.*, 47. *L. W.* and 54. *S.Y.*) the cerebro-spinal fluid withdrawn by lumbar puncture was turbid and was found by Dr. Jacob to contain an excess of polynuclear leucocytes; in none of these could the meningococcus be identified; in other cases (30. *A. G.*, 34. *R. G.*, 35. *A. R.*, 38. *P. M.* and 57. *H. H.*), although no microscopical examination was made, it is recorded that the cerebro-spinal fluid so withdrawn was turbid and under high pressure.

In three cases of the "paralytic" type, 9. *P. F.*, 77. *D. B.* and 78. *C. B.*, the blood definitely agglutinated a culture of the meningococcus in one hour, in a dilution of 1 in 20, and in a fourth case 27. *O. S.*, there was some "clumping" though less definite than in the foregoing. This test is not thought to have much positive value.

I have notes of the injection of Flexner's serum in several cases in which distinct improvement is stated by the medical attendant to have followed the injection. In considering the significance of the improvement, however, allowance should be made for the beneficial effects that might be expected from the relief of pressure on the spinal cord occasioned by the mere withdrawal of fluid.

As already mentioned a large number of faucial swabs taken by myself were examined by Dr. Mervyn Gordon with negative results as regards the presence of the meningococcus.

## Conclusion.

Pathological investigation, so far as it was carried out in respect of the 74 cases under consideration, gave no support to the view that any of them were cases of cerebro-spinal fever.

The diagnosis of cerebro-spinal fever was strongly upheld in respect of the more severe cases by some of the medical men who attended them. On clinical grounds alone it is not easy to differentiate between cerebro-spinal fever and the more severe bulbar or encephalic types of epidemic poliomyelitis. These more severe cases, however, were in many instances closely associated with milder cases of purely "paralytic" type, which conformed clinically in all respects to the description of poliomyelitis, and for the reasons above set forth I have no hesitation in concluding that the outbreak was one of epidemic poliomyelitis and not of cerebro-spinal fever.

## Cerne Outbreak.

Between September 29th and October 21st, 1910, an outbreak of disease, characterized by initial febrile symptoms with subsequent paralysis, occurred in the Cerne Rural District of Dorsetshire, at Cerne Abbas and at Up Cerne, a hamlet about a mile from the former. There were altogether 16 known cases, viz., 10 with one death at Cerne Abbas, four with one death at Up Cerne, and two in outlying cottages. These cases conformed with remarkable uniformity to a very definite type, the symptoms being as follows :— Acute febrile onset with moderately high temperature, not usually exceeding when recorded 102° ; headache and generally some pain in the back of the neck, down the spine, and in the limbs ; some retraction of the head was observed in a few cases, but in no instance was there severe opisthotonos ; vomiting occurred at the outset in some cases ; there was generally drowsiness and in some cases delirium, but not coma ; slight twitching in some cases but no convulsions ; some hyperæsthesia, but mental irritability was not marked. In the majority of cases Kernig's sign was not, it seems, looked for in the acute stage of the disease, but in three or four cases, which he had the opportunity of seeing during the first few days of their illness, my colleague, Dr. Mivart tested for this sign with negative results. When I saw the cases on November 18th and 19th, I did not find Kernig's sign present in a single instance. On the other hand, in the outbreak in the Midlands above described, I found Kernig's sign persistent in a large number of cases several weeks after the date of the acute attack. No observations were made as to the occurrence of *tache cérébrale* during the acute stage of this illness.

In all cases but one paralysis manifested itself at an early stage of the illness, as a rule within the first three days. In some instances a transient paralysis of the trunk muscles was observed during the acute stage, but this rapidly passed off, and paralysis of longer duration, affecting different groups of muscles in the extremities supervened.

The tendency was for several groups of muscles to be paralysed in the first instance, recovery occurring rapidly in some groups and more slowly in others ; in some cases it seems probable that a greater or less degree of permanent paralysis will persist.

The more permanent degree of paralysis affected the legs in 12 cases, the muscles of the neck and trunk in three, and of the arms in one ; in one case only was no degree of paralysis observed.

Of the 14 non-fatal cases recovery was reported as complete by January 9th in nine, and incomplete in five ; in three at least of these latter it seems likely that some degree of permanent paralysis will persist.

The history of the two fatal cases was as follows :—

*3. *A. W. V.*, 6, *M.*—Quite well and at school on *September 30th* ; attacked *October 1st* ; giddiness and pain in back of head ; supported the back of the neck with his hands ; *October 2nd*, partly insensible, delirum, twitching, hyperæsthesia, pains in the limbs ; seen by Dr. Dalton on *October 3rd* ; paralysis supervened on second or third

* The first figure is the number of the case in the tabular statement of "Cerne Cases," below.

day ; right arm, muscles of neck, back, and legs affected ; gradually improved and was recovering power in the legs and arms when abdominal, respiratory, and cardiac paralysis supervened ; died of cardiac failure on *October* 11*th*.

A younger brother of this patient, 4. *L. V.*, 4, *M.*, was attacked on October 9th ; very feverish ; twitching ; hyperæsthesia ; complained of pain in the back ; the illness passed off in three or four days, leaving no paralysis.

The only other child in the family was a baby who escaped attack.

11. *A. J. L.*, 14, *M.*—Acute attack *October* 22*nd* ; had been noticed to be run down on the previous day ; on *October* 22*nd* loss of power was observed in both legs ; Dr. Dalton was not summoned to see him till *October* 24*th*, when he observed no rigidity, and the initial symptoms, which do not appear to have been very acute, had subsided ; the patient died rather suddenly of cardiac failure on *October* 25*th*.

Two other children of this family had been previously attacked, viz., 9. *L. L.*, on *October* 7*th*, and 10. *F. T. L.*, on *October* 12*th*. The only other child of the family was away from home at the time of the outbreak.

Two of the non-fatal cases may be described as typical of the other cases among children.

7. *F. J. R.*, $2\frac{0}{12}$ *M.*, *Up Cerne*.—Only child ; was quite well on *October* 8*th* ; *October* 9*th*, attacked suddenly ; feverish ; temperature 102° ; vomited ; complained of pain under tongue ; twitching ; drowsiness ; irritability ; delirium ; retraction of head ; tongue coated ; paralysis of both legs, back, and right arm. During the first few days an herpetiform eruption was observed on the buttocks and down the thighs (this is the only case in which any rash was noted) ; the febrile symptoms subsided in the course of a few days ; the paralysis gradually improved ; when I saw him on *November* 17*th*, the muscles of the back and arm had recovered but there was fairly complete paralysis of both legs. On *January* 9*th* I was informed that some improvement had taken place but that both legs were still paralysed.

This child had been playing with 4. *L. V.*, brother of 3. *A. W. V.*, during the week, and certainly on October 6th. There is frequent communication between the two families.

1. *P. W.*, $3\frac{2}{12}$ *M.*, *Cerne*.—Attacked, *September* 29*th* ; was quite well the previous day ; headache ; vomiting ; delirium ; *September* 30*th*, temperature 103° ; was delirious for three days, and recovered gradually ; when I saw him on *November* 17*th* there was still a considerable amount of paralysis in both legs and the patellar reflexes were absent. On *January* 9*th* it was reported that there was gradual improvement, but he was still unable to walk.

The other members of this family are aged 17, 7, and 4. The child of 7, 2. *R. W.*, was attacked. The other children escaped. The two children attacked did not sleep together.

Only one adult was attacked in this outbreak, viz. :—

14. *G. T.* 36. *M.*, *Cerne*, a coachman. *October* 12*th* helped J. B., father of 13. *A. B.*, to clip a horse. *October* 14*th*, got wet and

chilled while driving. *October* 15*th*, attacked with aching pains in back which kept getting worse for three or four days. *October* 18*th*, kept his bed on this day for the first time with sickness, fever, and acrid sweats; was almost completely paralysed in both legs. *November* 18*th*, was beginning to recover slowly from the paralysis, could move his legs slightly from side to side but had almost complete paralysis of the extensor muscles and loss of patellar reflexes. *January* 31*st*, is in St. Thomas's Hospital with a considerable degree of paralysis in both legs but can walk a little if supported. It appears likely that a greater or less degree of permanent paralysis will be left.

It was practically impossible to isolate this man from his family, none of whom were attacked; but the wife and children were supplied at an early stage with a solution of permanganate of potash to use as a gargle.

The ages of the patients attacked were as follows :—

| | |
|---|---|
| Under 1 year old ... ... ... ... | 1 |
| Over 1 and under 5 years old ... ... | 8 |
| „ 5 „ „ 10 „ ... ... | 5 |
| „ 10 „ „ 15 „ ... ... | 1 |
| Adult ... ... ... ... ... | 1 |
| | 16 |

The infectivity in this outbreak was relatively high. Multiple attacks occurred in three households among brothers and sisters, viz., three cases in one household and two in each of two others.

There is a possible link, if incubation may be disregarded, between the Cerne and Up Cerne cases in the fact that Mrs. V. mother of 3. *A. W. V.* and 4. *L. V.*, on *September* 29*th*, when accompanied by *A. W. V.* met and spoke with Mrs. W. mother of 1. *P. W.* and 2. *R. W.* who was carrying the younger child *P. W.* in her arms. *P. W.* was noticed to be ill and was definitely attacked the same evening. *A. W. V.* was attacked on *October* 1*st* and his brother *L. V.* on *October* 9*th*.

As already mentioned 7. *F. J. R.*, who was attacked on *October* 9*th*, had been playing during the week, and certainly on *October* 6*th*, with 4. *L. V.*, who was also attacked on *October* 9*th*. There is frequent intercourse between the two families.

8. *F. J. B.*, of Up Cerne, was attacked on *October* 12*th*. He had been playing with 7. *F. J. R.* on *October* 8*th* and on this occasion Mrs. R., mother of *F. J. R.*, held him for some time on her knee.

5. *E. H.* was attacked on *October* 5*th* and 6. *T. S.* on *October* 8*th*. The H. and S. families live in adjacent cottages, within 10 yards of one another, and the children are constant playmates.

The influence of a "carrier" is perhaps indicated in the case of 14. *G. T.* J. B., the father of 13. *A. B.*, who was attacked on *October* 11*th* as already mentioned, helped *G. T.* to clip a horse on *October* 12*th*. *G. T.* was attacked on *October* 15*th*, after being exposed to chill and wet on the previous day.

Other cases are possibly attributable to school influence.

Dr. Dalton supplied the households in which cases occurred with a solution of permanganate of potash for use as a gargle. It is probable that this precaution tended to limit the outbreak.

I have not been able to discover how the outbreak originated. Cerne is a remote village but is a frequent resort of visitors from Dorchester and Weymouth, including many sailors who come in brakes on Sundays.

The symptoms of meningeal irritation were certainly less severe in this outbreak than in that occurring in the Midlands, and there were no cases resembling of cerebro-spinal fever. The two fatal cases died from cardiac paralysis and without the grave symptoms indicating profound cerebral and meningeal disturbance which are usual in the latter disease.

Dr. Mervyn Gordon has examined specimens of blood from nine of these patients, and in no instance did he obtain positive evidence of agglutination with a culture of the meningococcus.

On the whole the facts of the Cerne outbreak appear to point strongly to the conclusion that it was one of epidemic polio-myelitis.

In conclusion it is my pleasant duty to express my thanks to the Medical Officers of Health for the City of Nottingham, the Bingham, Melton Mowbray, and Cerne Rural Districts, and the Melton Mowbray Urban District, to Drs. Jacob and Kentish Wright of Nottingham, to Dr. Atkinson of Long Clawson, to all the medical men practising in Melton Mowbray, and to others who rendered me valuable assistance in collecting material for this report.

REGINALD FARRAR.

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Tabular analyses of the cases occurring in the Midlands and Cerne respectively follow this report.

# MIDLANDS.—CASES.

| Reference Number. | Initials of Name, Age and Sex. | Residence, and Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Condition as to Reflexes. | Kernig's Sign. | Tache Cérébrale. | Retraction of head Opisthotonos. | Paralysis. | Rash including Herpes. | Lumbar Puncture performed. | Result of Case and Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | E. O., 15, F. | Nottingham, April 27th. | 99°-104° Pyæmic temperature some days before death. | ++ | + | ++ | + | ++ | + | All reflexes absent or diminished. | + | | ++ | | Mottled rash, fading on pressure. | On several occasions Flexner's serum injected at least five times, always with good temporary results. | Developed epileptiform seizures, hæmatemesis and hæmaturia. Died in convulsions Aug. 20th. Meningococcus found. |
| 2 | E. H., 9, F. | Nottingham, May 31st. | Intermittent febrile followed by even sub-normal temperature after July 11th. | + | | ++ | | + | + | Absent | + | | ++ | Slight paraplegia and strabismus. | No rash. | On several occasions Flexner's serum injected. No marked effect. | Complete recovery. |
| 3 | E. S., 29, M. | West Bridgford, Nottingham, July 29th. | 100° to 105° | ++ | | | + | ++ | | | + | | ++ | Strabismus. | Ecchymotic rash. | Yes. | Death in coma, Aug. 2nd. Meningococcus found. |
| 4 | P. C., 7/12, M. | Nottingham, Aug. 7th. | 99·4° 102·8° | + | | ++ | + | + | + | | + | | ++ | Left arm and leg. Strabismus. | Mottled rash. No ecchymosis. | Yes. Some apparent improvement followed. Fluid under pressure. | Death in coma and convulsions, Aug. 9th. Meningococcus found. |
| 5 | R. T., 12, M. | Nottingham, Sept. 1st. | Pyæmic, over 105° at death. | + | | ++ | + | | | Patellar plantar and abdominal reflexes absent. | ++ | ++ | ++ | | None observed. | Yes. Sept. 20th. Flexner's serum 20 cc. injected. | Death in epileptiform convulsions, Sept. 6th. Meningococcus found. |

## MIDLANDS.—CASES—*continued.*

| Reference Number. | Initials of Name, Age and Sex. | Residence, and Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Condition as to Reflexes. | Kernig's Sign. | Tache Cérébrale. | Retraction of head Opisthotonos. | Paralysis. | Rash including Herpes. | Lumbar Puncture performed. | Result of Case and Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 6 | C. W., 27, M. | Nottingham, Sept. 25th. | 101°-105° | + | | + | ++ | | | Variable from day to day. | + | + | ++ | Transient facial and other paresis. | Mottled rash. | Yes | Death in coma, Oct. 18th. Meningococcus found. |
| 7 | A. B., 2, F. | WestBridgford July 24th. | 101°-104° | + | + | + | + | | | | | | ++ | | None | No | Died July 30th. |
| 8 | C. B., 5, F. | WestBridgford, July 20th. | Febrile | + | | | | | | | | | | | | No | Recovery complete. Abortive type. |
| 9 | P. F., 13, M. | Nottingham, Aug. 20th. | — | + | + | | + | + | | Patellar reflexes in abeyance. | + | — | + | Marked paraplegia, passing off and leaving some permanent paralysis of right leg. | None observed. | | Recovery, but some permanent paralysis of right leg. Blood agglutinated culture of meningococcus Jan. 20th. |
| 10 | F.C., 2, M. | Nottingham, Sept. 3rd. | 101° to 102° for two days, afterwards sub-normal. | + | + | | | | | Patellar reflexes absent | + | | ++ | Marked paresis of both legs passing off, but leaving some permanent paralysis and atrophy of peroneal and anterior tibial muscles. | No rash, but herpes labialis. | | Recovery, with some permanent paralysis and atrophy. |
| 11 | S. C., 2, M. | Nottingham (Same address as 7, F. C.), Sept. 10th. | 100°-102° when first admitted to hospital, afterwards normal. | + | No. | | | | | | + | | + | Paralysis of right peroneal muscles remaining permanent. Some transient strabismus. | No rash. | | Recovery, with some paralysis of peroneal muscles remaining. |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 12 | W. W., 3, M. | Eastwell, June 12th. | Febrile | | | | | | | | | | | Paralysis of right leg first observed 19th June. Anterior tibeal group still paralysed in December. | | No | Recovery with paralysis of right anterior tibeal muscles. |
| 13 | F. G., 1, M. | Eastwell, July 5th. | | | | | | | | | | | | Sudden appearance of facial paralysis. | | No | Complete recovery. |
| 14 | M. G., 39, F. | Eastwell, Aug. 24th. | Febrile | + | + | | | | | Patellar reflexes in abeyance. | | | + | Prostration with some transient paresis of legs. | | | Complete recovery. |
| 15 | W. M., 3, F. | Stathern, June 23rd. | Febrile | + | + | + | | | | – | – | – | + | Transient strabismus. Paresis of both legs. Subsequently of the left only. | | No | Recovery stated to be complete by end of Aug. |
| 16 | T. W., 4, M. | Harby, July 8th. | Febrile, 102° | + | + | + | + | | + | Patellar reflexes absent. | – | – | – | Both legs paralysed at first. Afterwards paralysis with wasting of left leg. | Eruption on trunk resembling varicella; herpes on lips. | No | Recovery with some permanent paralysis and wasting of left leg. |
| 17 | H. F., 32, M. | Harby, July 26th. | Febrile | + | + | | | | | Reflexes in abeyance. | + | + | + | Paralysis of right deltoid muscle. | | No | Recovery with wasting and paralysis of right deltoid at end of December. |
| 18 | M. C, 12, F. | Harby, Aug. 13th. | Febrile | + | + | | | | | Reflexes in abeyance. | + | + | | | | No | Complete recovery. |
| 19 | A. M., 13, M. | Harby, Aug. 13th. | Febrile | + | + | + | | | | Reflexes absent. | + | + | | Slight paresis of legs. | | No | Complete recovery. |
| 20 | E. C., 6, M. | Hose, July 24th. | Febrile | + | + | + | | | | Patellar Reflexes absent. | | | | Strabismus left eye. No other paralysis. | | | Complete recovery. |
| 21 | M. S., 6, F. | Hose July 31st. | Febrile, 100°-103° at first. Later sub-normal. | + | + | + | + | | | | + | + | | Paralysis right leg. | | Yes. Aug. 7th. Flexner's serum 15 cc. injected. | Died of "broncho-pneumonia," Aug. 10th. |
| 22 | W. C., 4, M. | Hose, Aug. 3rd. | Febrile, afterwards sub-normal. | + | + | + | + | + | + | Absent | + | + | + | Slight loss of power in legs. | | No | Complete recovery save for some mental excitability remaining in December. |

## MIDLANDS.—CASES—*continued.*

| Reference Number. | Initials of Name, Age and Sex. | Residence, and Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Condition as to Reflexes. | Kernig's Sign. | Tache Cérébrale. | Retraction of head Opisthotonos. | Paralysis. | Rash including Herpes. | Lumber Puncture performed. | Result of Case and Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 23 | G. A., 2, M. | Hose, July 20th. | Febrile | + | | + | + | + | + | Patellar reflexes absent. | + | + | + | Muscles of right leg | Petechial rash. | No | Recovery with some weakness and wasting of right leg. |
| 24 | E. T., 9, M. | Hose, Aug. 11th. | Febrile | + | + | | | | | Absent | + | + | + | Weakness of right leg; walked with a reeling gait. | | No | Recovery complete by December. |
| 25 | H. H., $1\frac{2}{12}$, M. | Hose, Aug. 20th. | Febrile, afterwards subnormal. | | | + | + | | | All reflexes absent. | | | + | Slight paralysis of leg. | | No | Complete recovery. |
| 26 | B. M., 13, F. | Radcliffe-on-Trent, July 31st. | 103° to 105·4° | + | + | + | + | + | + | | | | + | Paraplegia commencing on 4th day of illness. | On extremities. | No | Died August 5th. Was able to converse rationally up to half an hour before death. |
| 27 | O. S., 7, F. | Old Dalby, June 24th. | Febrile | + | | | | | | In abeyance | Not looked for. | Not looked for. | | Paresis both legs | | No | Recovery, but some weakness and wasting in two legs. Died of bronchitis in December. |
| 28 | S. S., 36, F. | Old Dalby, Aug. 12th. | Febrile 100° | + | + | | | | | Absent | + | + | + | | | No | Died August 19th. |
| 29 | C. T., $1\frac{7}{12}$, F. | Old Dalby, July 26th. | Febrile | | | ++ | + | | | | | | | Paralysis of both legs | | No | Died in coma, July 27th, before medical attendant arrived. |

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| 30 | A. G., $2\frac{6}{12}$, M. | Old Dalby, Aug. 24th. | Febrile 101° | + | | | + | + | | Absent | + | + | | Slight paresis of legs | | Aug. 25th Spinal fluid under high pressure. Flexner's serum 15 cc. injected. | Complete and uneventful recovery. |
| 31 | F. G., 5, F. | Old Dalby, Aug. 31st. | Febrile 100° | + | | | | | | | | | | Slight paresis of legs | | Aug. 31st. Spinal fluid not under pressure. | Complete and uneventful recovery. |
| 32 | E. T., 2, M. | Long Clawson, July 30th. | Febrile 100° | | | + | + | | | Reflexes absent. | + | + | + | Paresis of legs. | | July 31st. Flexner's serum 15 cc. injected. | Complete recovery. Immediate improvement followed injection of Flexner's serum. |
| 33 | A. S., 2, M. | Long Clawson, Aug. 2nd. | Sub-normal when first attended. | + | | + | | | | Patellar reflexes absent, | + | + | + | Strabismus. Paresis of legs. | | | Complete recovery. |
| 34 | R. G., $2\frac{5}{12}$, F. | Long Clawson, Aug. 13th. | Febrile 100° to 102°. | + | | + | | + | ++ | All reflexes absent. | + | + | + | Some paresis of trunk muscles and left leg. | Rash resembling that of enteric fever. | On each occasion, Aug. 20th and Aug. 23rd, Flexner's serum 15 cc. injected. Fluid turbid and under pressure. | Complete recovery. |
| 35 | A. R., 12, F. | Long Clawson, Aug. 23rd. | Febrile 101° | + | | + | | + | | Reflexes variable. | + | + | + | No paralytic symptoms. | Covered from top to toe with an eruption of dull red spots resembling those of enteric fading on pressure. | Aug. 25th. Spinal fluid turbid and under high pressure. Flexner's serum 15 cc. injected. | Complete recovery. Injection of Flexner's serum, followed in a few hours by marked relief of all symptoms. |
| 36 | L. S., 9, M. | Long Clawson, Aug. 20th. | Febrile | + | | | | | | Reflexes absent. | + | + | + | Transient paralysis of legs. | | No. | Complete recovery. |

## MIDLANDS.—CASES—*continued.*

| Reference Number. | Initials of Name, Age and Sex. | Residence, and Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Condition as to Reflexes. | Kernig's Sign. | Tache Cérébrale. | Retraction of head Opisthotonos. | Paralysis. | Rash including Herpes. | Lumbar Puncture performed. | Result of Case and Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 37 | C. S., 15, M. | Long Clawson, Aug. 23rd. | Febrile 102°-104°. | + | + | + | + | | | Patellar and abdominal reflexes absent, plantar normal. | + | ++ | + | Ptosis and strabismus right side. Paralysis of left facial nerve. Paralysis of legs. Paralysis of pharynx and of bladder. | | Aug. 24th, 25th and 27th. Flexner's serum 15 cc. injected on each occasion. | Very slow convalescence. At the end of December there was still considerable paresis, with wasting of both legs, so that he could not stand, and left facial paralysis. |
| 38 | P. M., 2, F. | Long Clawson, Aug. 30th. | Febrile 100°-102°. | + | | + | + | ++ | ++ | | + | + | ++ | Paresis of trunk muscles at first; afterwards of right arm and leg. | Erythematous patches and blotches all over body. | Sept. 4th, 6th and 7th. Flexner's serum 15 cc. injected on each occasion. Fluid under pressure, but clear on 4th, turbid on 6th. | Recovery, with wasting and some paralysis of right deltoid and anterior tibial muscles of right leg. |
| 39 | W. B., 2, M. | Upper Broughton, July 20th. | Febrile | | | | | + | ++ | | + | — | — | Paresis of both legs and wasting with dropped-foot of the right. | | No. | Recovery, with weakness of both legs and wasting of the right with dropped-foot. |
| 40 | J. W., 6, M. | Upper Broughton, July 20th. | Febrile | | + | ++ | + | | | | | | | | | No. | Death in coma, July 31st. |
| 41 | L. P., 8, M. | Upper Broughton, Aug. 6th. | Febrile 100° | + | | + | + | | | Absent. | + | | | Paralysis of right upper arm, wasting of deltoid. | | No. | Recovery, with some permanent paralysis. |

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| 42 | T. E., 37, M. | Upper Broughton, Aug. 26th. | Febrile, 100° to 102°. | + | + | | + | | | All reflexes absent. | + | + | ++ | Paralysis of trunk muscles during acute stage. Paresis of both legs during convalescence, with subsequent complete recovery. | | Aug. 28th, 29th and 31st. Spinal fluid under pressure and turbid. Flexner's serum 15 cc. injected on each occasion. | Complete recovery. Resumed work as ironstone labourer in December. |
| 43 | R. G., 15, F. | Nether Broughton, July 16th. | | | | | | | | | | | | Paralysis with dropped foot on right leg. | | No | In December was still suffering from paralysis with dragging of one foot. |
| 44 | E. M., 10, M. | Nether Broughton, July 25th. | Febrile | + | | | | | | | | | + | Slight paresis of legs. | | No | Complete recovery. |
| 45 | B. G., 4, M. | Nether Broughton, Aug. 1st. | Febrile | + | + | + | + | | | Absent | | | + | Slight loss of power in one arm. | | No | Complete recovery. |
| 46 | G. H., 5, M. | Hickling, Aug. 1st. | Febrile, afterwards sub-normal. | + | | + | + | | | Absent | + | + | | Temporary paralysis of pharynx. Paresis of legs. | | Aug. 6th. ½ fluid withdrawn. Flexner's serum 10 cc. injected. | Complete recovery. Immediate relief followed lumbar puncture and injection of Flexner's serum. |
| 47 | L. W., 6, F. | Hickling, Aug. 13th. | Febrile, 99·4-103·2°. | + | + | + | + | | | Patella absent, reflexes afterwards exaggerated. | + | + | + | Paralysis of muscles of neck. During the first few days there was also slight paralysis of trunk and limbs. | | Aug. 18th. Spinal fluid turbid and under high pressure. Flexner's serum 15 cc. injected. | Complete recovery. Rapid improvement followed lumbar puncture and injection of Flexner's serum. |
| 48 | M. G., 16, F. | Hickling, Aug. 26th. | Febrile | + | | | ++ | | | | + | + | + | | | Aug. 29th. Flexner's serum injected. | Complete recovery, but headache persisted for some weeks. |
| 49 | F. G., 5½, M. | Kettleby, July 24th. | Febrile, 104°. | + | | | | + | + | | | | | | | No | Died July 29th. |

## MIDLANDS.—CASES—*continued.*

| Reference Number. | Initials of Name, Age and Sex. | Residence, and Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Condition as to Reflexes. | Kernig's Sign. | Tache Cérébrale. | Retraction of head Opisthotonos. | Paralysis. | Rash including Herpes. | Lumber Puncture performed. | Result of Case and Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 50 | D. H., 11, M. | Holwell, Aug. 7th. | Febrile at first, later sub-normal. | + | + | | | | | All reflexes absent. | + | + | + | Paresis of trunk and leg muscles. | Petechial rash on chest and abdomen. | No | Complete recovery. |
| 51 | A. G., 27, F. | Holwell, Aug. 9th. | Febrile 101°. Sub-normal after lumbar puncture. | + | + | | | | | Absent at first, afterwards exaggerated. | + | + | + | Paralysis of legs | | Aug, 12th. Flexner's serum 15 cc. injected. | Convalescent in December but still some weakness and pain in lumbar region. Distinct improvement the day after lumbar puncture was performed. |
| 52 | L. D., 4, F. | Asfordby. July 31st. | Febrile 104-6°. The fever subsided in about 5 days. | + | | + | + | + | + | | + | + | + | Strabismus—transient. | | No | Complete recovery but a condition of mental irritability continued for some time. |
| 53 | T. P., 6, M. | Asfordby, Aug. 15th. | Febrile | + | | + | | | | | + | + | + | No paralysis observed at any time. | | No | Complete recovery, the symptoms subsided in a few days. |
| 54 | S. Y., 9, M. | Asfordby, Aug. 17th. | Febrile | + | | + | + | + | | | | | + | — | | Twice | Died August 24th. |
| 55 | S. T., 3½, M. | Asfordby, Aug. 21st. | Febrile 102° | + | + | + | + | + | + | | | | | No paralytic symptoms. | | | Complete recovery. |

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| 56 | E. L., 1½, M. | Asfordby, ? | | | | | | | | | | | | Paralysis at first slight, then complete of the left leg, was first noticed on August 25th. | | | At the end of December, 1910, there was still partial paralysis of the left leg. |
| 57 | H. H., 9, M. | Stonesby, Oct. 21st. | | + | + | + | + | + | + | Superficial and deep reflexes all diminished or abolished. | + | + | ++ | Paralysis right arm and leg, the paralysis of the leg remaining permanent. | | Oct. 24th. Spinal fluid turbid. | At the end of December convalescent, but still considerable loss of power in right leg. |
| 58 | H. B., 11, M. | Stonesby, Oct. 10th. | | | | | | | | — | — | — | — | Paresis of both legs, persistent at end of December. | Well-marked brownish spots all over back not fading on pressure. | | Convalescent but had a considerable amount of paresis in both legs at the end of December. |
| 59 | L. H., 4, M. | Grimstone | Febrile, sub-normal for several days after admission to hospital. | | | | | + | | — | — | — | — | Severe paralysis of both legs persisting at end of December. | | | At end of December both legs were paralysed; there was a very little power over the left leg, none at all over the right. |
| 60 | T. K., 36, M. | Sproxton, ? | | | | | | | | Patellar reflexes sluggish. | — | — | + | Strabismus slight paralysis of neck of bladder. | | | Still has strabismus and general weakness at end of December. |
| 61 | J. N., 3, F. | Thorpe Arnold, July 11th. | Febrile | | | | | + | + | | + | + | | Paralysis of both legs at first limited, afterwards to peroneal and anterior tibial muscles. | | No | Convalescence, with paralysis of peroneal and anterior tibial muscles of both legs persisting in December. |
| 62 | R. R., 9, F. | Melton Mowbray, June ? | No febrile stage observed. | | | | | | | | | | | Paralysis of left leg. | | No | Recovery, with paralysis and 1¼ inch wasting of left leg below the knee. |
| 63 | J. M., 2¾, M. | Melton Mowbray, Aug. 5th. | Febrile | | | | | + | + | In December left patellar reflex exaggerated, right normal. | + | | | Paralysis of left leg. Strabismus. | | No | Recovery, with some weakness and wasting of the left leg. |

## MIDLANDS.—CASES—*continued.*

| Reference Number. | Initials of Name, Age and Sex. | Residence, and Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Condition as to Reflexes. | Kernig's Sign. | Tache Cérébrale. | Retraction of head Opisthotonos. | Paralysis. | Rash including Herpes. | Lumber Puncture performed. | Result of Case and Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 64 | E. M., 3, F. | Melton Mowbray, July 14th. | Febrile | + | + | + | + | + | + | | | | ++ | Temporary paralysis of legs. Strabismus. | | No | Complete recovery save for slight strabismus. |
| 65 | H. C., 9, M. | Melton Mowbray, June 23rd. | Febrile, 102° | + | | + | + | + | + | | | | | | | No | Complete recovery. |
| 66 | P. B., 13, M. | Melton Mowbray, Sept. 5th. | Febrile, 103°, afterwards sub-normal, 95°. | + | + | + | + | + | | Absence of patellar reflexes. | + | + | + | Slight left internal squint. | Indefinite rose spots on abdomen. | No | When seen late in September had slight internal squint, absence of patella reflexes and tender spots in cervical and lumbar regions. |
| 97 | J. T., 3, M. | Melton Mowbray, July 27th. | Febrile | + | + | + | | + | + | | + | | | Paralysis of both legs, particularly the left, facial paralysis, strabismus. | | No | Recovery with some loss of power in the left leg in December |
| 68 | F. S., 4, M. | Melton Mowbray, July 30th. | Febrile, 103° | + | | + | | | | | + | + | | Paralysis of right leg and arm, lasting 2 or 3 weeks. | | No | Complete recovery. |
| 69 | E. D., 11, F. | Melton Mowbray, July 31st. | Febrile | + | + | | | | | Absence of patellar reflexes. | + | | | Some paralysis in both legs which persisted for some weeks. | | No | Complete recovery. |
| 70 | W. M., 4, M. | Melton Mowbray, Aug. 18th. | Febrile | + | + | | + | + | | | + | + | + | Slight strabismus | | No | Complete recovery. |

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| 71 | L. G., 3, M. | Melton Mowbray, Oct. 1st. | Febrile | + | + | + | | + | + | Abdominal reflexes abolished; patellar diminished. | + | + | ++ | Paralysis of pharynx necessitating nasal feeding for some days, some paresis of trunk and lower limbs, left facial paralysis. | Eruption of rose spots not fading on pressure over shoulders, back, and buttocks. | No. | Complete recovery by end of December. |
| 72 | R. S., 45, F. | Melton Mowbray, Aug. 22nd. | Febrile T. 101°. | | | | | | | | | | | No paralysis observed. | | No. | Illness attributed to "lumbago." Tedious convalescence. |
| 73 | L. T., 6, M. | Melton Mowbray. Aug. 31st. | Febrile 102·4° | + | + | + | | + | | Stated to be normal. | + | | + | Marked paralysis of both legs, worst on the right side with practically complete paralysis of all the muscles below the knees. | | No. | Recovery with almost complete paralysis in both legs below the knees persisting in December. |
| 74 | A. G., 33, F. | Melton Mowbray, Aug. 31st | Febrile 102° | + | + | | | | | No loss of patellar reflex. | Stated to be absent. | − | + | Glossopharyngeal paralysis with inability to articulate or swallow. Fluids regurgitated through nose. Right facial paralysis, palate insensitive. | | No. | When seen in December had still some difficulty in articulation; could swallow liquids with difficulty but not solids. |
| 75 | H. K., $1\frac{2}{12}$. M. | Melton Mowbray, Sept. 9th. | Febrile | + | + | + | | + | | | | | + | Paralysis of both legs, the left being the worst. | | No. | Recovery. |
| 76 | H. E., 16, F. | Melton Mowbray. Sept. 13th. | Febrile | + | + | + | + | + | | Patellar reflexes at first exeggerated, afterwards normal. | + | + | ++ | Paralysis of glossopharynx, strabismus; paralysis of sphincters. | | No. | Complete recovery. Discharged from Isolation Hospital. Convalescent at the end of four weeks. |
| 77 | D. B., 8, F. | Oakthorpe, Sept. 5th. | Febrile | + | + | + | | | + | Patellar reflexes, left diminished, right absent. | + | − | − | Paresis of both legs. | | No | Recovery. |
| 78 | C. B., 5, M. | Oakthorpe, Sept. 9th. | Febrile 102° | + | | | | + | + | Patellar reflexes absent. | + | + | + | Paresis of both legs. | | No | Recovery. |

## MIDLANDS.—CASES—*continued.*

| Reference Number. | Initials of Name, Age and Sex. | Residence, and Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching Convulsions. | Delirium. | Hyperæsthesie. | Irritability. | Condition as to Reflexes. | Kernig's Sign. | Tache Cérébrale. | Retraction of head Opisthotonos. | Paralysis. | Rash including Herpes. | Lumbar Puncture performed. | Result of Case and Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 79 | F. M., 8, M. | Bottesford, June 6th. | Febrile about 103° for three days. | + | + | | | | | Present throughout. | | | | No paralysis observed. | | | Complete recovery. |
| 80 | A. M., 6, F. | Bottesford, June 8th. | Febrile for several days 100° to 104°. | | + | + | + | | | Patellar reflexes absent at first. | + | | + | At first paralysis of head, back and leg muscles. Later, paralysis with wasting of the extensors of the right leg. | None | No | Feb. 2nd. The left leg has completely recovered. the right leg is steadily improving under massage and electricity. |
| 81 | F. M., 4, M. | Bottesford, Aug. | Febrile two days. | + | | | | | | | | | + | Paralysis noticed about third day of attack. | | | — |
| 82 | C. N., 2½, F. | Bottesford, Aug. | Febrile | + | | | | | | | — | — | + | Paralysis, left leg | | | — |
| 83 | A. W., 15, F. | Barlestone. Aug. | Febrile | + | | + | + | | | | | | + | | | A turbid exudate extracted by lumbar puncture. | Died Aug. 30th. |

## CERNE.—CASES.

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | P. W., $3\frac{2}{12}$, M. | Cerne, Sept. 29th. | Febrile, 103° | + | + | | + | | | Patellar reflexes absent. | — | | | Paralysis both legs | | | Jan. 9th. Gradual improvement, but still unable to walk. |

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| 2 | R. W., 7, M. | Cerne, Oct. 1st. | Febrile, 102° | + | + | | | | | In Nov. patellar reflex full on the right side, absent on the left. | | | | Paralysis both legs | | | Complete recovery. |
| 3 | A.W.V., 6, M. | Up Cerne, Oct. 1st. | Febrile | + | | + | + | + | | | | | + | Paralysis of right arm and muscles of neck, back and legs. Finally abdominal respiratory and cardiac paralysis set in. | | | Died Oct. 11th of cardiac failure. |
| 4 | L. V., 4, M. | Up Cerne, Oct. 9th. | Febrile | + | | + | | + | | | | | | No paralysis observed | | | Complete recovery. The illness passed off in 2 or 3 days, leaving no paralysis. |
| 5 | E. H., 8, F. | Cerne, Oct. 5th. | Febrile | + | + | + | + | | | | | | | Loss of power in trunk muscles especially of left side. Later some paralysis of legs. | | | Jan. 9th. Recovery almost complete, but falls down easily if pushed. |
| 6 | T. S., 3¼, M. | Cerne, Oct. 8th. | Febrile | + | | | | | | | | | | Sudden loss of power in legs, Oct. 8th. | | | Complete recovery. |
| 7 | F.J.R., 2¹⁰/₁₂, M. | Up Cerne, Oct. 9th. | Febrile, 102° | + | + | + | + | + | + | | | | | Paralysis of muscles of legs, back and right arm. On Nov. 17th muscles of back and arm had recovered, but there was fairly complete paralysis of both legs. | Herpetiform eruption on buttock and down thighs. | | Jan. 9th. The arm has recovered. There is still paralysis of both legs, but some improvement has taken place. |
| 8 | F.J.B., 2¹⁰/₁₂, M. | Up Cerne, Oct. 12th. | Febrile | + | + | + | | | + | | | | + | Paresis of muscles of back and neck, could stand but not walk. | | | Complete recovery. |
| 9 | L. L., 2¹⁰/₁₂, F. | Cerne, Oct. 7th. | Febrile | + | | + | | + | | | | | + | Loss of power in muscles of back and neck, no affection of legs. | | | Recovery appeared to be complete by Nov. 18th. |

## CERNE.—CASES—*continued.*

| Reference Number. | Initials of Name, Age and Sex. | Residence and Date of Onset. | Range of Temperature. | Headache. | Vomiting. | Twitching Convulsions. | Delirium. | Hyperæsthesia. | Irritability. | Condition as to Reflexes. | Kernig's Signs. | Tache Cérébrale. | Retraction of head Opisthotonos. | Paralysis. | Rash including Herpes. | Lumbar Puncture performed. | Result of Case and Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 10 | F. T. L., $\frac{5}{12}$, M. | Cerne, Oct. 12th. | Febrile | | | | | + | + | Patellar reflexes absent. | | | + | Loss of power in muscles of back and legs. | | | On Nov. 18th there was still some paresis of the legs and loss of patella reflexes. Jan. 9th, recovery complete. |
| 11 | A. J. L., 14, M. | Cerne, Oct. 21st. | Febrile | | | | | | | | | | | Paralysis observed in both legs, Oct. 22nd, Cardiac failure, Oct. 25th. | | | Died suddenly from cardiac failure, Oct. 25th. |
| 12 | W. H., 5, M. | Cerne, Oct. 8th. | Febrile | + | | | | | | | | | | Loss of power in muscles of back and later of legs. | | | Recovery almost complete by Nov. 18th. Jan. 9th, recovery complete. |
| 13 | A. B., 3, M. | Cerne. Oct. 11th. | Febrile | + | + | | | | | | | | | Paralysis of muscles of back, subsequently of legs. | | | Complete recovery. |
| 14 | G. T., 36, M. | Cerne, Oct. 15th. | Febrile | + | + | | | | | Patellar reflexes absent. | | | | Almost complete paralysis of both legs. | | | Had almost complete paralysis of both legs. Jan. 9th, some improvement, can now walk slightly with help. |
| 15 | J. G., 1$\frac{5}{12}$, M. | Cerne, Oct. 8th. | Febrile | + | + | | | | + | | | | | There appeared to be slight paralysis of trunk muscles. | | | Recovery complete by Nov. 18th. |
| 16 | M. S., 5, F. | Cerne, Oct. 23rd. | Febrile | + | + | | | | + | | | | | Complete paralysis of both arms. | | | Jan. 9th, right arm still completely paralysed, left showing very slight improvement. |

## No. 3.

# Notes by Dr. Mervyn Gordon on Acute Poliomyelitis with reference to its Etiology, Histology, and as to Immunity.

The morphology of the virus of acute anterior poliomyelitis is still unknown ; while there are good grounds for suspecting it to be ultimately particulate, it is so minute that nobody has yet seen it with certainty : even with the highest microscopic powers available. Up to the present time, therefore, the chief method of investigating this disease has been by animal experiment. In these respects, as also in the character of the microscopic lesions found in the central nervous system, there is great similarity between poliomyelitis and rabies ; the virus of which also is " ultramicroscopic."

The following summary of the results of experimental investigations of poliomyelitis up to the present time is taken chiefly from a recent article by Landsteiner on the subject. It will be remembered that this observer and Popper were the first to publish the fact some two years since that acute poliomyelitis of man is communicable to the monkey ; although Flexner and Lewis working with material obtained during the recent epidemic in America appear to have discovered the same thing independently at about the same time as the Austrian observers.

### *Experimental Poliomyelitis.*

For the experimental study of this disease monkeys are used. A few investigators have succeeded in infecting rabbits, but the majority of those who have tried to do so have failed. As the results with rabbits are inconstant, and this animal, when infected, is liable not to exhibit characteristic signs of the disease, monkeys alone are available at the present time for the experimental study of poliomyelitis.

Owing to the fact that the spinal cord either of a fatal human case or of a previously infected monkey contains the virus in larger quantity than the other organs, an emulsion of the cord is generally used for the purpose of inoculation. Poliomyelitis, however, has been produced by inoculation of the cerebral cortex, or of the submaxillary lymphatic glands, or of the nasal mucous membrane of an animal affected by the disease. In exceptional cases the disease has been produced by inoculating the cerebrospinal fluid, or a large amount of blood from an infected case, but these materials contain very little virus ; and such experiments are, for the most part, negative. It is, perhaps, of interest in this relation to refer to the result of a recent investigation of the distribution of virus in the organs of a child who had died of acute poliomyelitis undertaken by Landsteiner, Levaditi, and Pastior. These investigators made saline emulsions of the various organs of the child and injected them into monkeys. The animals injected with the brain, the spleen, the salivary gland, and the mesenteric glands showed no symptoms ; on

the other hand, monkeys that received emulsions of the spinal cord, the tonsil, and the pharynx developed acute poliomyelitis. In the child in question the disease had begun with an acute angina.

Should any of the materials for inoculation be contaminated by bacteria, the emulsion can be freed of such by passing it through a filter such as the Chamberland or Berkefeld. These filters hold up the bacteria, but allow the virus of poliomyelitis to pass through.

The spinal cord to be used for purposes of injection is best taken from an animal at the height of the disease. With decline of the symptoms the virulence of the cord markedly diminishes. When obtaining material for inoculation, the cord is removed aseptically, ground up well in a mortar, and an emulsion of it made in 1 per cent. saline. In order to get rid of the larger particles, the emulsion may be filtered through gauze, or through filter paper. Frequently a very small dose of this emulsion succeeds in producing the disease, whereas too large a dose does not act so well.

In most of the successful experiments the virus has been injected intracerebrally, subdurally, intraperitoneally or intravenously. It may also be injected intraneurally: in which case the resulting paralysis may begin in regions supplied from that part of the cord where the injected nerve joins it. The disease has also been conveyed by injection into lymphatic glands or into the trachea, or into a loop of intestine. It has also been produced by inhalation, by introduction of the virus by means of a stomach tube, by painting it on to the nasal mucous membrane (Leiner and Wiesner), by injecting it into this mucous membrane, or by rubbing it on to the nasal mucous membrane after scarification (Flexner and Lewis, Levaditi and Landsteiner).

In place of the fresh material, good results may be obtained with the preserved virus. The virulence of the cord is maintained for at least a number of weeks if kept in the ice chest either in dried condition, or in concentrated glycerine, or in a mixture of 1 part glycerine to 2 parts saline (Flexner and Lewis, Römer and Joseph, Landsteiner and Levaditi).

### *Qualities of the Virus.*

The virus of poliomyelitis is a so-called "filtrable" virus (Flexner and Lewis, Leiner and Wiesner, Levaditi and Landsteiner), that is to say, it readily passes through filters such as the Chamberland, Berkefeld, Reichel, or Pukall filters which do not transmit the smallest visible bacteria.

The resistance of the virus to freezing and to glycerine has already been referred to, as also its resistance to drying. When dried over $H_2SO_4$ the material remains virulent for at least 14 days, and when dried over KOH for at least 24 days. When dried in vacuo (away from light) at room temperature it is potent for four weeks (Römer and Joseph).

On the other hand, the resistance to heat is small. Heating for half an hour at from 45°–55° C. destroys it (Leiner and Weisner, Flexner and Lewis).

According to present observations, the virus is destroyed by disinfection with menthol preparations, by potassium permanganate (1 : 1000 1 hour at 37° C.), by hydrogen peroxide (1 : 100), by formaldehyde vapour, by 1 per cent. thymol (1 hour), and by 0·5 per cent. phenol in three days at a low temperature. It must be borne in mind, however, that these figures are all founded on single experiments on monkeys, and that the more exact limits of the above disinfectants have not yet been ascertained.

Flexner and Lewis, and also Levaditi, attempted to obtain a culture of the virus by inoculating a filtrate of the cord emulsion into broth containing 10–25 per cent. of rabbits' or monkeys' serum. On incubation, the inoculated medium became slightly turbid, and this turbidity also developed in one or two sub-cultures ; but it is not yet proved that a real culture was here obtained. Preparations of the turbid fluid stained by Löffler's flagella stain, by Giemsa, or by prolonged staining with fuchsine showed the presence of very small corpuscles, round or oval in shape and frequently in pairs or in groups. Römer and Joseph, however, who examined the virus with the ultramicroscope, failed to find any corpuscles.

Levaditi and Landsteiner having found that the cords of monkeys infected with a small dose of virus that had been passed through Berkefeld or Chamberland filters were exceedingly infectious for other animals, concluded that poliomyelitis is due to a microbe which possesses the power of actively multiplying ; and not to a toxic product elaborated by this microbe and accumulated in the cord.

### *Experimental Poliomyelitis in the Monkey.*

Poliomyelitis in the monkey follows after an incubation period, which varies in different experiments from 4–46 days from the date of inoculation. If the virus is working well, the incubation period is not more than from 1–2 weeks.

After prodromal symptoms such as langour, diminished appetite, tremors of the head, which may last from a few hours to a day or so, pareses and paralyses set in. Generally, the paralysis occurs first in the lower extremities, and spreads up in the more severe cases to the upper extremities and to the muscles of the trunk and neck. In a small proportion of cases the muscles of the upper extremities and of the neck are affected first. Occasionally there is paralysis of some of the cranial nerves, e.g., the third (ptosis), and the seventh. In one monkey, described by Landsteiner and Levaditi, paralysis occurred in these two nerves, but not elsewhere. The clinical picture of bulbar paralysis is also seen in the monkey.

The paralyses are frequently complete. They are constantly "flaccid"—only in exceptional cases have spastic appearances been observed. The paralysed limbs feel cool to the touch. Very frequently the animals die with the progressing paralysis. If they survive, the paralyses endure after the disease has abated, and result in atrophy and contracture.

### *The Histological Diagnosis of Acute Poliomyelitis.*

Sure recognition of poliomyelitis is possible in fatal cases owing to the very characteristic histological lesions which occur in the cord.

The macroscopic changes in the central nervous system are slight, and consist only of some hyperæmia and œdema of the meninges and grey matter. An inconstant feature is hæmorrhage into the grey matter of the spinal cord.

In histological preparations one sees an infiltration of the meninges chiefly with mononuclear round cells. A similar infiltration is present in the neighbourhood of the anterior fissure, the vessels of which are frequently enclosed by a thickened mantle of cells which is continued in regard to their branches in the grey matter.

In the grey matter itself, especially in the anterior horns, this perivascular infiltration is seen, and also discrete groups of round cells which contain various kinds of mononuclear, and especially in the early stage of the disease, polynuclear elements. For the rest, the grey matter in the acute stage of the disease is swollen, and contains, in many cases, hæmorrhages.

Frequently there are also very striking changes in the motor cells of the anterior horns. These cells show different grades of destruction, *e.g.*, granular degeneration and vacuolation up to complete disintegration of both cytoplasm and nucleus—"neuronolysis." With this decay is associated the phenomenon of "neuronophagia," in which the motor cells are surrounded and infiltrated with partially phagocytic round cells and finally disappear completely, leaving in their place a heap of round cells.

Though much less intense than in the cord, similar inflammatory changes, *i.e.*, meningeal and perivascular infiltration, may be met with in some cases in the crura and in the cerebral cortex.

Owing to the profuseness of the change, there is, as a rule, no difficulty in the histological diagnosis of poliomyelitis. It should be borne in mind, however, that mere hyperæmia without the other changes is not sufficient evidence to justify a diagnosis of poliomyelitis.

No bodies precisely similar to Negri's corpuscles in rabies have yet been demonstrated in poliomyelitis. Bonhoff, however, has found certain inclusions in the nuclei of the glia cells which he claims to be specific evidence of poliomyelitis.

There is as yet no specific test *in vitro* by which poliomyelitis can be recognised. Attempts to diagnose this disease by the "Fixation of Complement" process have not been successful so far.

A table showing the grosser points of pathological distinction between meningitis of the commoner types and acute poliomyelitis appears below.

### *Active Immunity.*

Animals that have recovered from an attack of poliomyelitis are usually resistant to another dose of the virus (Flexner and Lewis, Landsteiner and Levaditi, Leiner and Wiesner, Römer and Joseph).

This immunity seems to appear early.

TABLE showing the Grosser Points of Pathological Distinction between Meningitis of the commoner types and Acute Poliomyelitis.

| Disease. | Cerebro-spinal Fluid. | | Meninges of the Brain and Cord. | Substance of the Cord. | Histological Examination of Cord. |
|---|---|---|---|---|---|
| | Cells present. | Bacteria. | | | |
| Cerebro-spinal Fever (Meningococcal Meningitis). Also 'Post-Basic' Meningitis. | Polymorphonuclear chiefly. | Diplococcus intracellularis (Meningococcus). | Purulent exudate in sub-arachnoid space, especially evident at posterior aspect of cord and at base of brain. May be present over vertex also. Films of the pus show polymorphonuclear cells, some of which contain meningococci. | Escapes, for the most part. | Unnecessary. |
| Septic Meningitis (including meningeal infection from pneumococcus, streptococcus, staphylococcus, B. influenzæ, B. coli, &c.). | Polymorphonuclear chiefly. | Pneumococcus, streptococcus, &c. | Purulent exudate with similar distribution to above. Films of the pus show polymorphonuclear cells and the infecting micro-organism in large numbers. | Escapes, for the most part. | Unnecessary. |
| Tuberculous Meningitis | Lymphocytes chiefly. | B. tuberculosis | Sero-fibrinous exudate, well marked at base of brain. Tubercles present in neighbourhood of blood vessels; especially conspicuous along those in Sylvian fissure. Films of the exudate show lymphocytes and tubercle bacilli. | Escapes, for the most part. | Unnecessary. |
| Poliomyelitis (Acute)... | Lymphocytes chiefly. | Nil | Some hyperæmia may be present, and a little clear or slightly turbid exudate. Films show lymphocytes; no bacteria. | The grey matter is congested. Exceptionally, minute hæmorrhages into it can be seen with the eye. | Imperative. The pathological diagnosis depends upon it. |

Levaditi and Landsteiner attempted actively to immunise animals with the dried cord after the well known method of Pasteur in reference to hydrophobia; but they failed to prevent poliomyelitis by this means.

Flexner and Lewis immunised animals by using gradually increasing doses of fully virulent cord.

Attempts to produce immunity by vaccination with the virus inactivated by heat have been made without success. Kraus, however, claims to have produced immunity in the monkey by subcutaneous injection of a vaccine consisting of virus weakened by treatment with phenol. His present vaccine appears to consist of an emulsion of a poliomyelitis cord which has been kept for 5 days in contact with saline containing from 1 to 1·5 per cent. of phenol.

Finally, it should be mentioned that Landsteiner and Levaditi and Römer and Joseph have succeeded in actively immunising monkeys by injecting "sensitised vaccine" after the method introduced by Besredka. "Sensitised vaccine" consists of a mixture of virus and immune serum. (*See below.*)

### *Passive Immunity in Poliomyelitis.*

The serum of an animal immunised against poliomyelitis possesses the capacity of inactivating the virus in vitro (Levaditi and Landsteiner, Römer and Joseph, Flexner and Lewis).

The laboratory procedure by which this important point may be proved is as follows. The virus—a 5 per cent. emulsion of poliomyelitis cord in salt solution—is mixed with an equal volume of immune serum. The mixture is then kept either for 4 hours at the room temperature and over-night in the ice chest, or it is placed in the incubator at 37° C. for 1 hour and then for 12–24 hours at 15° C. If the serum is not fresh, Netter and Levaditi add some fresh normal serum for complement. For the proof, these fluids, as also control mixtures of the virus mixed with normal serum, are injected intracerebrally into monkeys.

Levaditi and Landsteiner have applied this capacity of an immunised animal's serum of neutralising the virus of poliomyelitis as a test for that disease. If there has been an attack of poliomyelitis, the serum of the patient possesses the specific capacity of neutralising the virus. By means of this test a number of abortive and unrecognised cases of poliomyelitis have been identified. As the test, however, involves the sacrifice of at least two monkeys, one of which is used for the necessary control experiment with normal serum, it is not likely to come into general use.

Flexner and Lewis report that protection can be afforded by injection subdurally of immune monkey's serum immediately after the injection of a small dose of the virus into the nasal mucosa or even 18–24 hours after the injection intracerebrally of a small dose of the virus; the protection failing after this time, and after injection of a large dose of the virus. Liberal and repeated doses of the immune serum are needed to save the infected monkeys.

Finally Netter, Gendron and Touraine report the result of injection of immune serum into the subarachnoid space of four human

cases affected by acute anterior poliomyelitis. The serum used by them was human serum obtained from patients who had recovered from the disease. The injections were from 3–9 in number, and the average dose given was 9 c.c.

In the four cases of anterior poliomyelitis treated in this way the stage from the onset varied from 1–6 days, and the paralysis was still extending at the time when the serum was first applied.

We are told that one of these cases succumbed to respiratory paralysis; but that in the other three cases the march of the paralysis was arrested, and the most recent paralyses disappeared.

In the case of a youth of 16 the first two injections of serum produced an amelioration; but 36 hours afterwards a disquieting relapse supervened which, however, was arrested by a series of six injections of the serum.

From these facts there follows the necessary criticism that anterior poliomyelitis is in its present clinical aspects far too variable a disease, and that these cases are far too few in number for any general inferences to be drawn from them. But when this has been said, the experimental evidence referred to above still gives substantial ground for hope that the time is not far distant when this disease will be under control.

M. H. GORDON.

*References.*

The article by Landsteiner referred to can be seen in the first Supplementary Volume to the *Handbuch der Technik und Methodik der Immunistatsforschung* by *Kraus and Levaditi*, *Jena*, 1911, pp, 458–464, where references to the literature will be found.

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## No. 4.

# Report by Dr. Mervyn Gordon on the Result of the Examination of Materials received in connection with Dr. Reece's inquiries in Devon and Cornwall.

### 1. *Cerebrospinal fluids.*

The results obtained with cerebrospinal fluids from cases during the present outbreak have been individually reported. In general, it would appear that in cases of acute poliomyelitis the cerebrospinal fluid is most often clear; though exceptionally it may be slightly turbid.

The cells present are lymphocytes. Polymorphonuclear cells are conspicuously absent.

None of the well known bacteria of meningitis are found. When the fluid is not sterile, the micro-organism present consists of Staphylococcus epidermidis, or some other contamination.

From the observations at present made, it would seem that the reduction of Fehling's solution may furnish a useful distinction between the cerebrospinal fluid of poliomyelitis and of tuberculous meningitis respectively. Normal cerebrospinal fluid added to an equal amount of Fehling's solution, and boiled, reduces it. This reducing capacity is lost in most cases of meningitis—tuberculous or other—but appears to be retained in many cases of poliomyelitis. A larger number of cases, however, must be examined before the value of this test can be decided.

Early in the course of the present investigation Dr. A. E. Gow drew my attention to the fact that after removal of the suspended matter from a cerebrospinal fluid by centrifugalisation, important evidence is to be obtained by measuring the percentage of albumen in the remaining fluid by some such appliance as Aufrecht's well-known albuminometer. This point is still under investigation; but the method promises to be a valuable addition to the procedures at present in general use when examining cerebrospinal fluids.* It would appear that normal cerebrospinal fluid shows by Aufrecht's method about 0·05 per cent. of albumen. In the majority of cases of poliomyelitis examined so far there appears to have been a definite increase; the albumen being about 0·1 per cent. In many infections of the meninges, however, the albumen content of the cerebrospinal fluid rises above this figure. Thus a sample of cerebrospinal fluid from a case of meningococcal meningitis gave 0·4 per cent; another from a case of pneumococcal meningitis gave 0·55 per cent.; a third from a case of syphilitic meningitis gave 0·3 per cent. It would seem, then, that the percentage of albumen in a cerebrospinal fluid is an important point. There are, however, two drawbacks to the application of its increase as a gauge of meningitis. The first is that for the observation to be of value, the cerebrospinal fluid should be free of blood; for this, of course, tends

* The quantitative estimation of the albumen content of cerebrospinal fluid appears to have been in use in Germany for some years past, having been introduced by Nissl in 1904. In this country the majority of observers appear to have been satisfied hitherto with the rough estimation.

to vitiate the albumen reading. The second is that the albumen reading in tuberculous meningitis appears from present observations to be comparatively low—0·1 to 0·2 per cent. As the cells present in tuberculous meningitis are chiefly lymphocytes, and tubercle bacilli, though probably present in this fluid in all cases of tuberculous meningitis, are not always easy to demonstrate in films; it is clear that there may be considerable difficulty in distinguishing the cerebrospinal fluid of a case of tuberculous meningitis from that of a case of poliomyelitis.

Apart, however, from the differences clinically between the two conditions, tuberculous meningitis is at the present time invariably fatal, and a naked eye inspection of the central nervous system *post-mortem* readily distinguishes this condition from acute poliomyelitis. It may, however, be necessary to wait for the result of the histological examination before some fatal cases of acute illness accompanied by nervous symptoms, and found to show post-mortem marked hyperæmia of the central nervous system, can be differentiated from cases of acute poliomyelitis.

### 2. *The Histological Examination of Spinal Cords from suspected cases of Poliomyelitis.*

When reviewing the present state of knowledge of the pathology of acute poliomyelitis, it was mentioned that a sure recognition of this condition was possible from histological examination of the cord *post-mortem*.

While it is equally true that a sure differential diagnosis of this disease is possible *ante-mortem* in typical cases from the correlation of the clinical picture with the result of lumbar puncture, there is, nevertheless, in the present state of our knowledge still greater value from the epidemiological standpoint to be attached to the certainty that is the outcome of an examination *post-mortem*, when that examination is completed by microscopical examination of the cord.

During the present outbreak, cords from four cases have been examined histologically by me.

I. In the first of these cases decomposition had advanced to such a stage that the cord was disorganised; and though it was hardened and sections cut, the structure could not be made out histologically.

II. In a second case, however (Case No. 121), a piece of the cord was received in its membranes from Dr. Gray, of Holsworthy, for examination on August 26th. A part of this cord was hardened in formalin and sections cut, stained, and examined.

These sections showed that while there was some slight degree of meningitis present (evidenced by the dilation of the meningeal vessels, and the accumulation of a few lymphocytes here and there on the outside of the pia mater) such meningitis was of altogether minor degree. The grey matter, however, showed the classical lesions previously cited as justifying the histological diagnosis of poliomyelitis, viz., hyperæmia accompanied by well marked perivascular infiltration with lymphocytes and accumulation of the same in the grey matter, more especially in the neighbourhood of some of the large motor nerve cells. The latter showed granular

degeneration, and in some cases atrophy. Figures 1, 2, and 3 are from this case.

III. A third cord (Case No. 80) was received on September 2nd from Dr. Mosier, of Crediton.

When this cord was cut across, before being put into the hardening solution, hæmorrhages in the grey matter were distinctly visible with a hand lens.

On histological examination the characteristic lesions of acute poliomyelitis were found present. Perivascular infiltration with small round cells was particularly evident; Figures 4, 5, 6. In the upper part of the cervical cord a good deal of hæmorrhage had occurred in the grey matter. The nerve cells of the anterior horns had in some places disappeared.

In both of the above cases the distribution of the inflammation was by no means uniform throughout the grey matter of the respective cords. At one place the grey matter would show very little, if any, inflammatory change; and at another, perhaps a fraction of a millimetre away, well marked inflammatory change could be seen in one or both horns. Nor was the inflammatory change restricted to the anterior horns exclusively; at some places other parts of the grey matter were affected. Moreover, the white matter did not escape altogether, as Wickman has observed (*Studien über Poliomyelitis acuta, Berlin*, 1905).

IV. The fourth case in which the cord was submitted to histological examination was a patient (male, aged 27), under the care of Dr. T. B. A. Haggard, and seen by Dr. T. J. Horder in consultation with him. The case does not belong to Dr. Reece's series, but is included on account of its pathological interest. After a short illness, characterised by slight pyrexia, accompanied by loss of the plantar reflexes and of the knee jerks, and some sphincter trouble, a rapidly ascending paralysis of the Landry type set in, and the patient succumbed with bulbar paralysis, and failure of the respiratory muscles other than the diaphragm, which continued working till nearly the end.

The lumbar and dorsal portions of the cord were removed 26 hours after death, placed in 10 per cent. formalin, and afterwards submitted to histological examination. The appearances found are seen in the photographs reproduced in figures 7, 8, 9, and 10, which serve to illustrate the more marked microscopical changes in the cord in acute poliomyelitis. Figure 10 showing the infiltration of the degenerated nerve cells of the anterior horn with phagocytic lymphocytes portrays the "Neuronophagia" to which reference has already been made when dealing with the literature of the subject. Other sections of this cord show equally well the loss of substance of the nerve cells to which the term "Neuronolysis" has been given.

In fatal cases of suspected poliomyelitis it is advisable to place at once a piece of the cord in glycerin 1 part, to 1 per cent. saline two parts, in order that the material may be of use for animal experiment. The rest of the cord and, if possible, the bulb, pons, crura, and a piece of cortex from the cerebrum and cerebellum respectively, should be put in 10 per cent. formalin solution so as to be available for the histological examination.

### 3.—*The result of Animal Experiments with the material from the present Outbreak.*

I am indebted to the courtesy of Professor Levaditi of the Pasteur Institute in Paris for animal experiments with the material from the present outbreak.

Portions of the spinal cords of cases Nos. 80 and 121 of Dr. Reece's Series (in glycerin 1 part to 1 per cent. salt solution 2 parts) were forwarded to Professor Levaditi and were by him injected into monkeys.

Professor Levaditi reports on 22nd October that a monkey that received an emulsion of the cord of the case 121 developed typical acute poliomyelitis after an incubation period of 11 days. The monkey injected with the cord of case 80 also developed typical acute poliomyelitis. The experimental work is still proceeding, but these results would seem to establish the fact that the virus of the present outbreak is of full virulence.

M. H. GORDON.

PLATE I.

FIG. 1.

Horizontal section of a piece of dorsal cord from a fatal case of poliomyelitis, Case No. 121, at Holsworthy. At the site represented by the section the inflammatory change is more marked in the left anterior horn of grey matter than in the right.

Magnified × 20.

FIG. 2.

The left anterior horn of Fig. 1 under higher magnification. While the inflammatory exudate is well marked in the immediate neighbourhood of the blood-vessels, it is also scattered throughout the grey matter of this horn.

Magnified × 50.

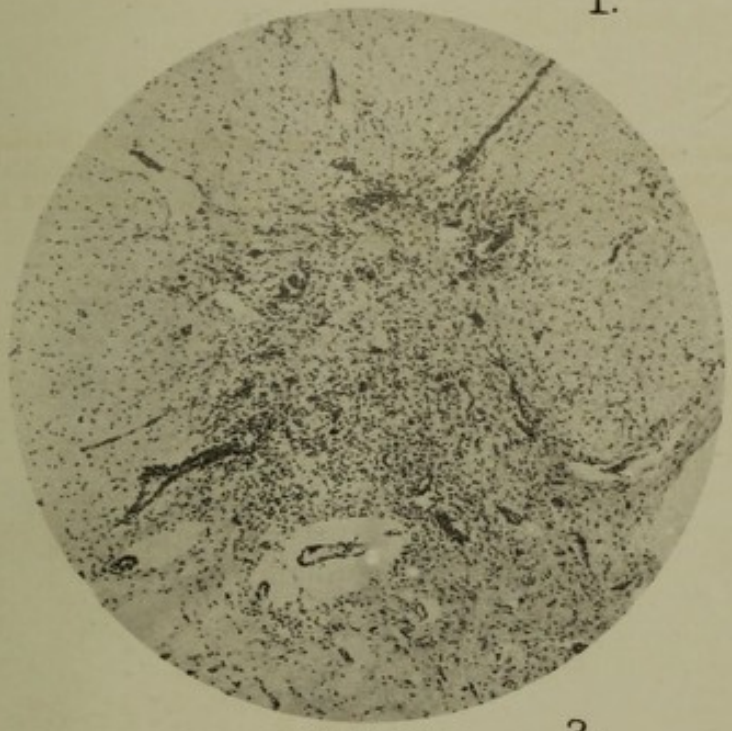
FIG. 3.

The right anterior horn of Fig. 1 under the same magnification. Here there is comparatively little change.

Magnified × 50.

*Plate 1.*

1.



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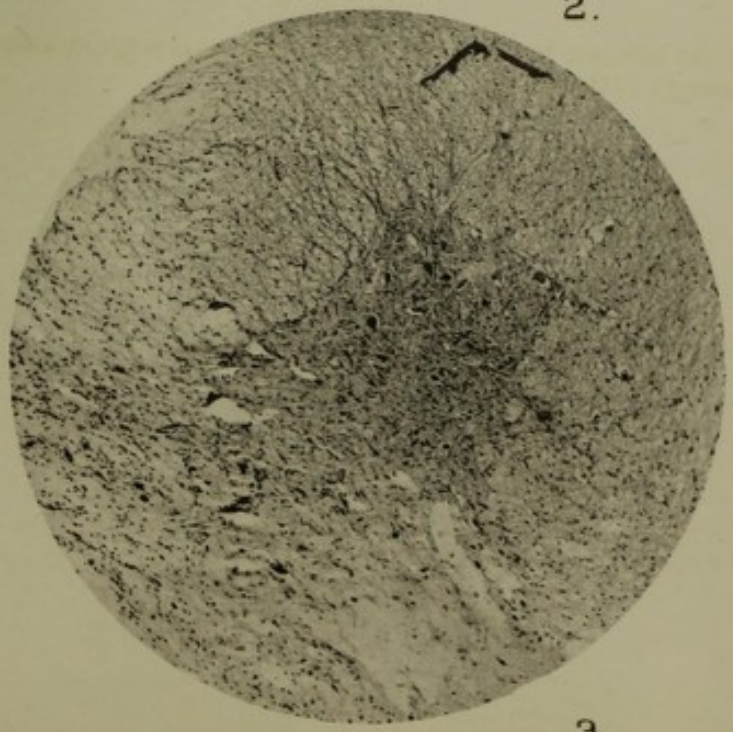

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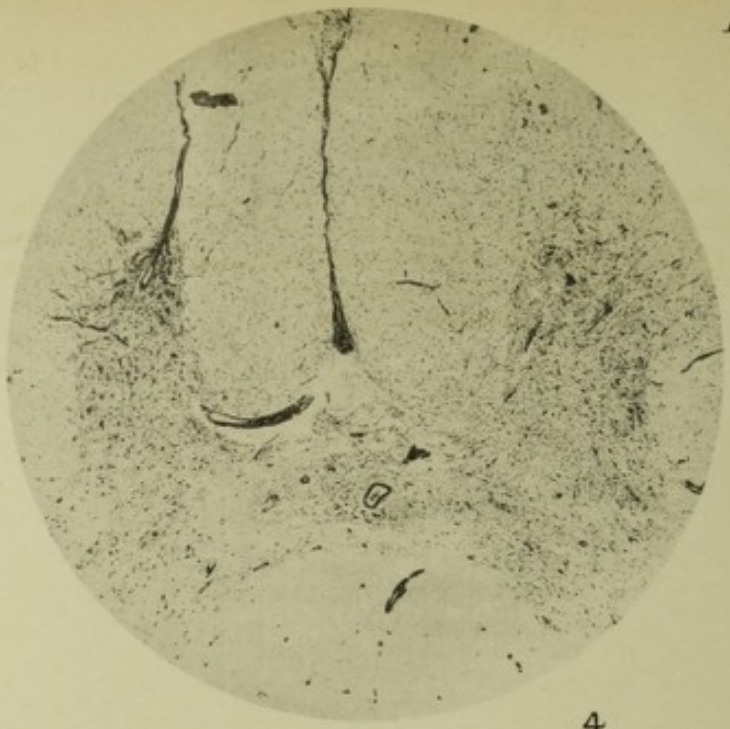

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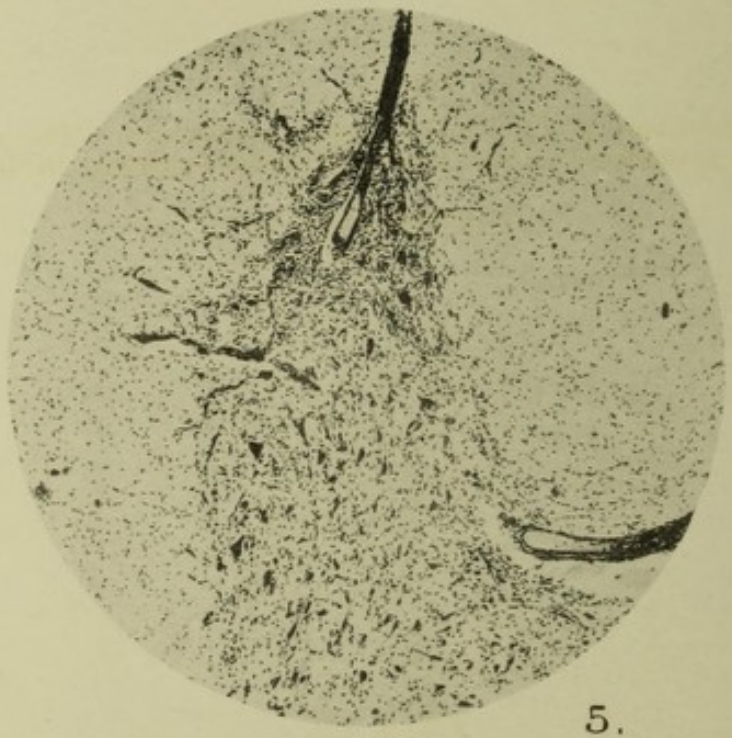

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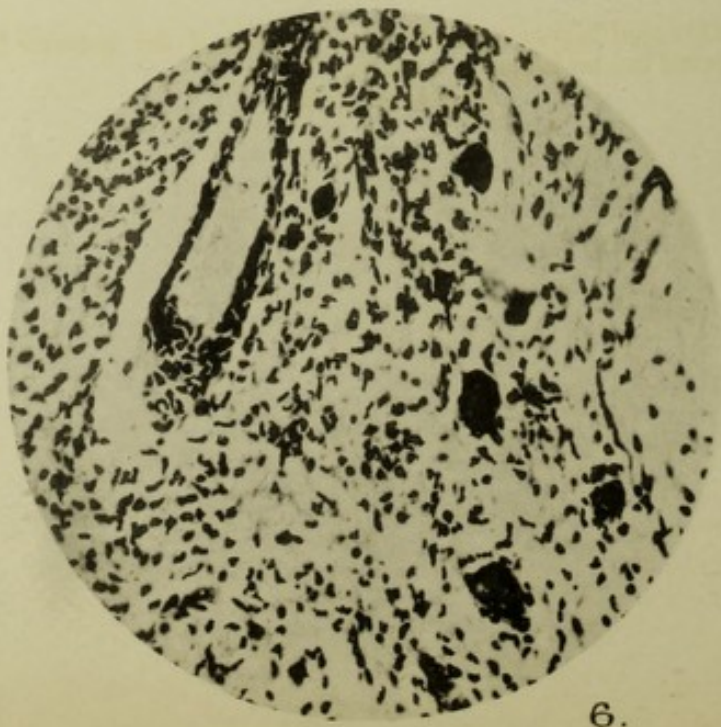


6.

PLATE II.

FIG. 4.

Cross-section of the dorsal cord of a fatal case of poliomyelitis, Case No. 80. Here there is also a contrast between the two horns. The blood-vessel of the anterior sulcus, and especially the blood-vessels supplying the left anterior horn, are picked out by an exudate of inflammatory cells.

Magnified × 20.

FIG. 5.

The left anterior horn of Fig. 4 under higher magnification. The inflammatory exudate is particularly evident around the vessel supplying the tip of this horn.

Magnified × 50.

FIG 6.

The same under still higher magnification. Some of the anterior horn nerve cells near the vessel are undergoing atrophy.

Magnified × 230.

PLATE III.

FIG. 7.

Cross-section of the lumbar cord of a recent fulminating case of poliomyelitis in a young adult residing in London. Foci of inflammatory exudate are seen scattered throughout the grey matter, and are especially well marked around the blood-vessels. The nerve cells of the anterior horns have been destroyed. The central canal of the cord has been blocked up by inflammatory exudate.

Magnified × 14.

FIG. 8.

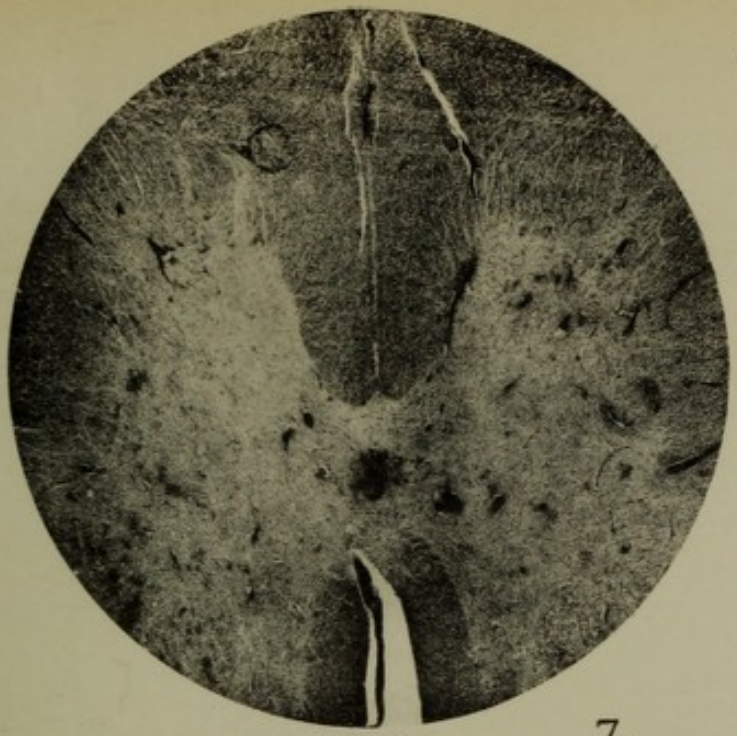
One of the anterior horns of Fig. 7 under higher magnification. No nerve cells can be made out. Here and there the place where a nerve cell has been is occupied by a focus of inflammatory cells.

Magnified × 50.

FIG. 9.

From a section of the same cord in the upper dorsal region. Three blood-vessels are seen in cross-section, and their perivascular lymph spaces are filled with an exudate of lymphocytes.

Magnified × 100.



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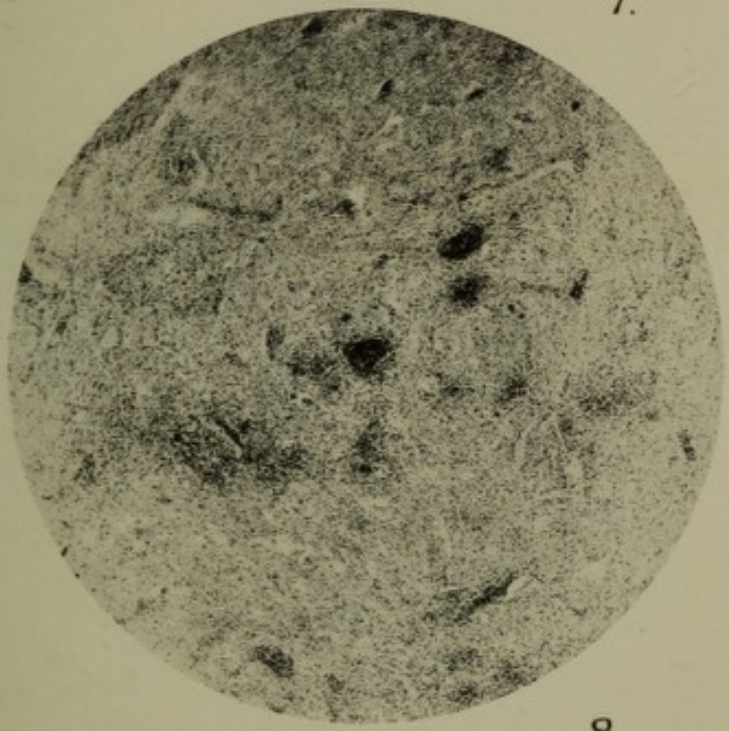

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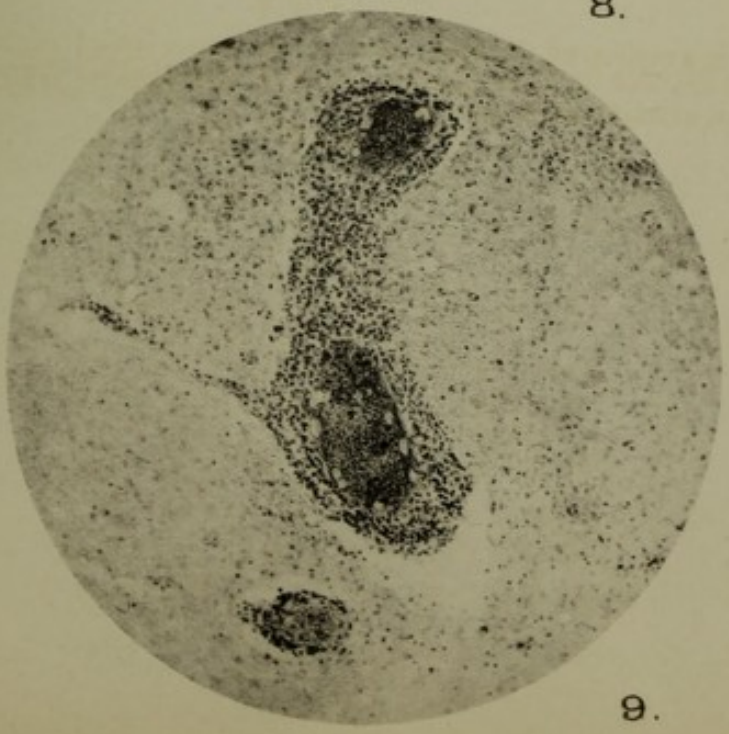

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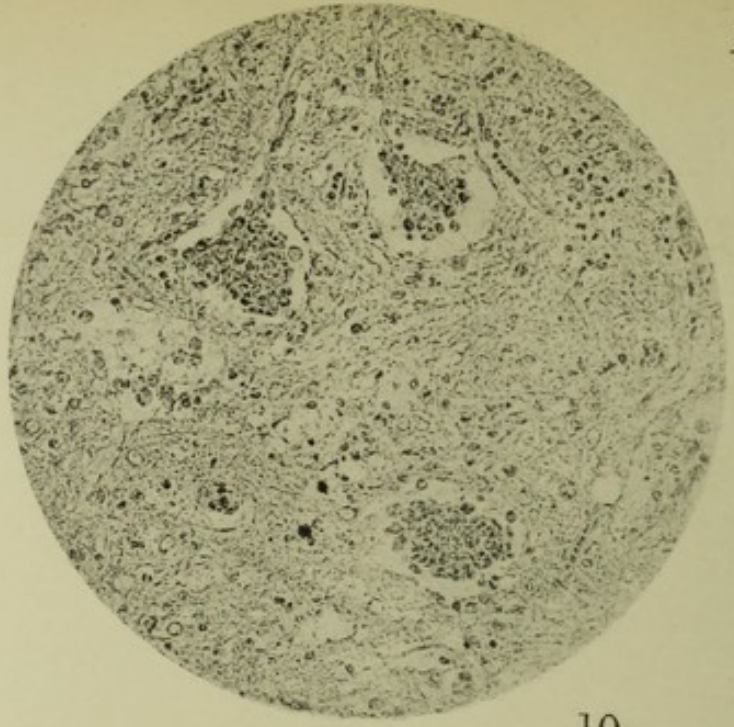

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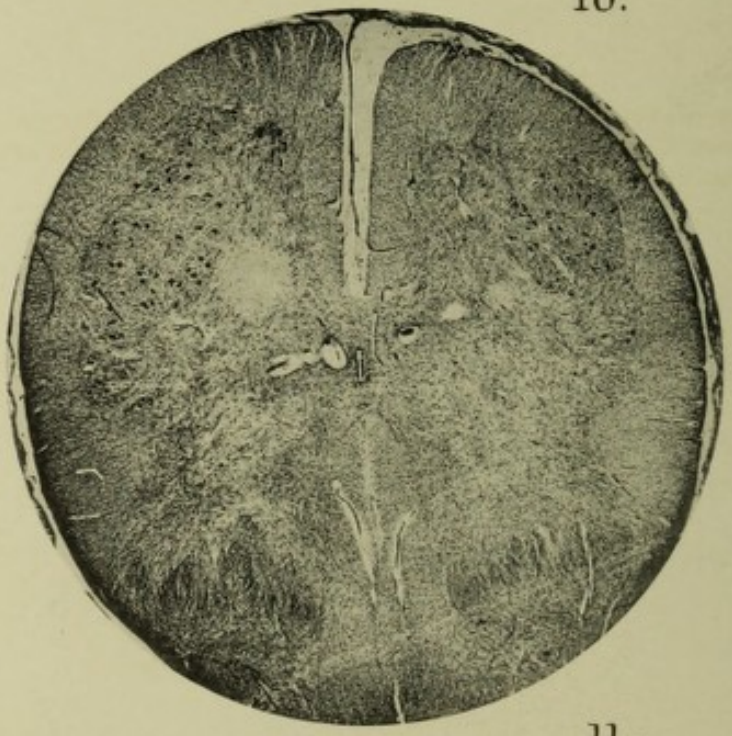

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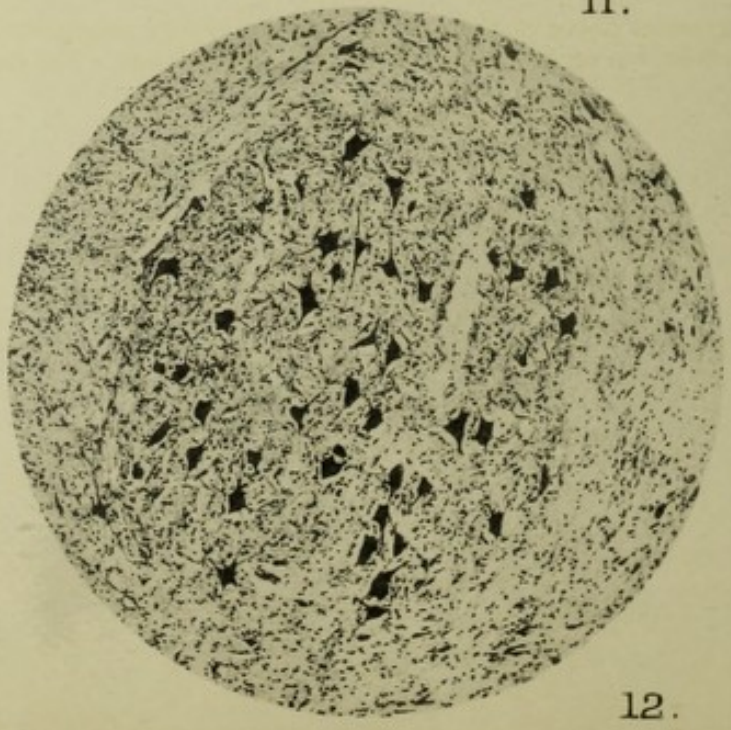

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PLATE IV.

FIG. 10.

From a section of the lumbar cord of the same case. The three anterior horn cells in the field are seen to be undergoing "neuronophagia."

Magnified × 175.

FIG. 11.

Cross-section of the lumbar cord of a case of acute cerebro-spinal fever. The grey matter is unaffected, and the anterior horn nerve cells can be made out. For comparison with Fig. 7.

Magnified × 14.

FIG. 12.

One of the anterior horns of the last figure under higher magnification. The anterior horn cells are as well seen as in the normal cord. For comparison with Fig. 8.

Magnified × 50.

PLATE V.

FIG. 13.

Section across the dorsal cord and membranes of case 121, showing the absence of meningeal exudate. The material had been forwarded in glycerine, and the cord has shrunken somewhat, and the pia mater has become detached.

Magnified × 8.

FIG. 14.

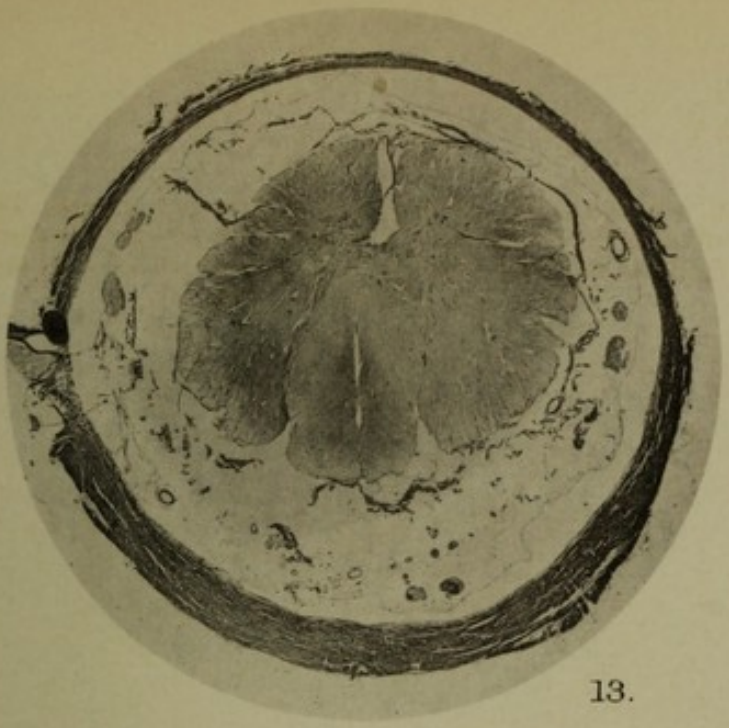
Section across the lower lumbar cord of a case of cerebrospinal fever, showing nerves in section and darker collections of pus in the sub-arachnoid space towards the posterior aspect.

Magnified × 8

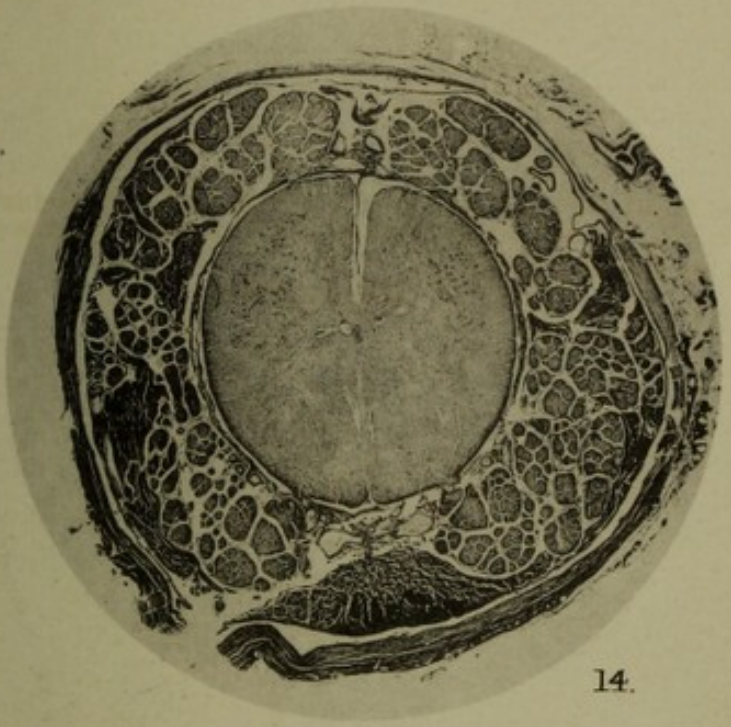
FIG. 15.

Section of commencement of the anterior sulcus in the lumbar cord of a case of tuberculous meningitis, showing the accumulation of lymphocytes outside the pia mater.

Magnified × 60.



13.



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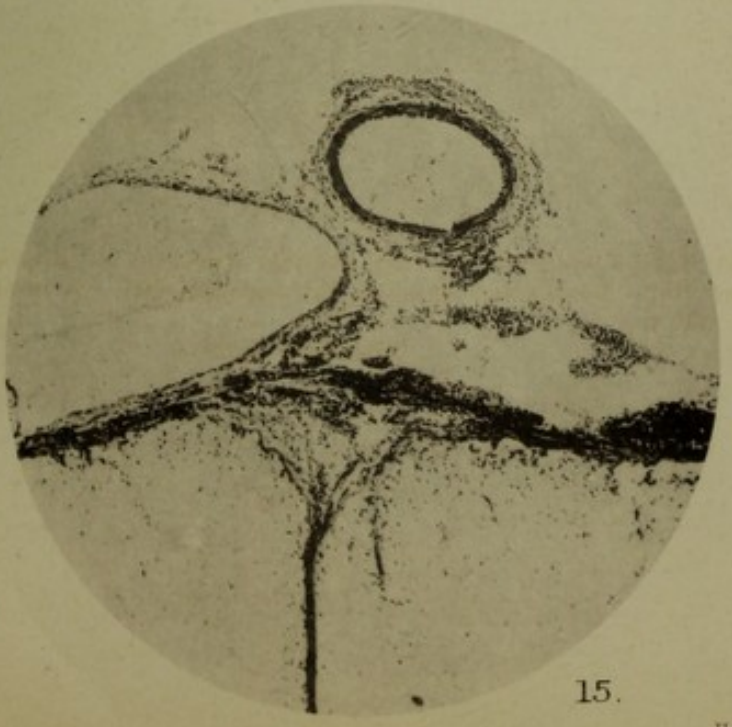
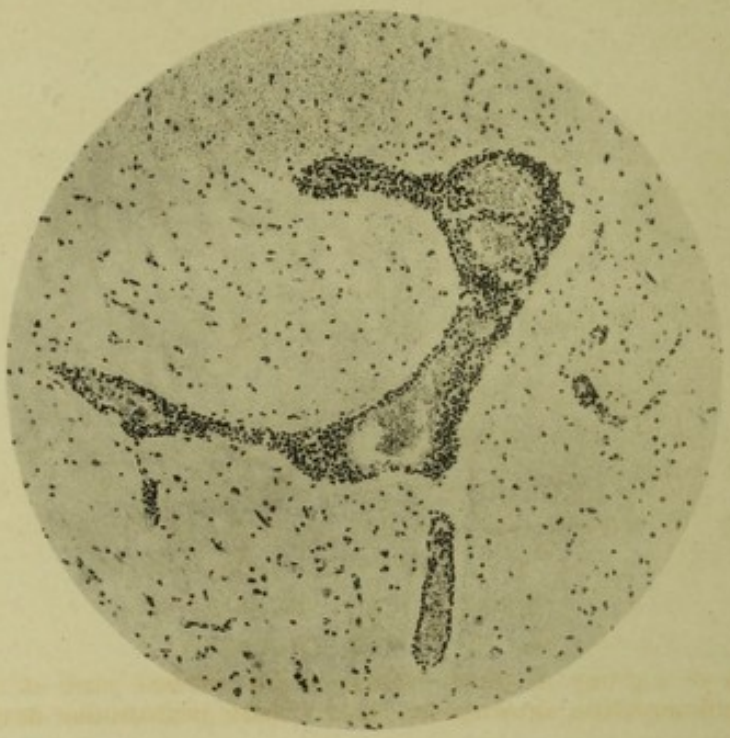


15.

Huth, London.



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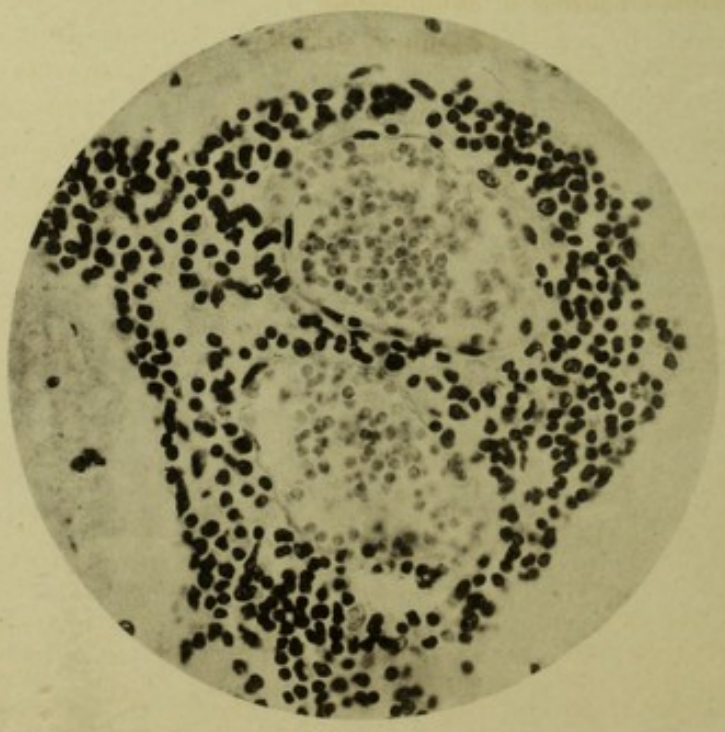

17.

Huth, London.

## PLATE VI.

FIG. 16.

Cross section of a group of blood vessels in the cervical cord of a further case of acute poliomyelitis, showing the characteristic perivascular exudate.

Magnified × 85.

FIG. 17.

Portion of the same section under increased magnification, showing that the perivascular exudate consists of lymphocytes.

Magnified × 300.

# No. 5.

## Clinical and Epidemiological Notes on Acute Poliomyelitis, by Dr. Hugh A. Macewen.

The following notes have been made as a result of a study of some of the principal writings on the clinical and epidemiological aspects of acute poliomyelitis. It should be understood that they do not relate to the results of personal observations or inquiries.

### PART I.—CLINICAL NOTES.

#### *Definition.*

Acute poliomyelitis is not a new disease. Its clinical features and pathological characteristics were fully described by Heine[1] in 1840, and it has long been recognised as a disease of children by the medical profession in this country, usually by the name of "infantile paralysis" or "anterior poliomyelitis."

In recent years certain districts in Europe, America and Australia have been invaded by an epidemic disease, the clinical and pathological characters of which strongly resemble those of infantile paralysis. This malady, appearing to be infectious in character, has been very carefully studied by many observers and is now known as acute anterior poliomyelitis, epidemic poliomyelitis or, on the continent, as Heine-Medin's disease.*

The term "acute anterior poliomyelitis," implying an acute inflammation localised in the anterior cornua of the grey matter of the spinal cord, though indicating the chief lesion, does not cover the whole of the conditions met with. In most cases it is not solely the anterior cornua that are affected—though they are the part of the nervous system that generally suffers most. The white matter of the cord may likewise be involved. Nor are the lesions confined to the spinal column, for the brain and pia mater may also participate in the morbid changes.

Poliomyelitis, to use the shorter and more convenient term, may be defined as a disease resulting from an acute, self-limiting, general infection, occurring sporadically and in epidemic form—its most marked symptoms being sudden paralysis emanating from lesions in the spinal cord and brain.

#### *Symptomatology.*

The disease presents such a variety of symptoms that it is impossible to present a single clinical picture. Different epidemics

* Wickman states that Netter and Levaditi have been able to demonstrate that the serum of a person who had three years before suffered from sporadic poliomyelitis was able to neutralize in vitro the virus of epidemic poliomyelitis. He believes that this, among other observations, conclusively proves that they are one and the same disease.

have been characterised by certain groups of prodromal or preliminary symptoms. There is generally an initial rise of temperature accompanied by symptoms which may not at first suggest any suspicion of the true nature of the disease,—drowsiness, irritability, malaise, vomiting and gastro-intestinal disturbances being very common.

The onset is frequently attributed by the patient's friends to a fall, a chill, or, in the case of infants, teething. Swimming or wading in cold water has been associated with the onset of attack in certain epidemics.

Shortly after the occurrence of primary symptoms, paralysis generally appears—such paralysis being seldom observed without such preliminary manifestations, though occasionally a child may go to bed apparently well and be found in the morning to be suffering from paralysis.

The following account of the clinical aspect of the disease has been taken largely from the works of Wickman[2], who is probably the greatest authority on the subject.

### *General Symptoms.*

*Onset.*—The onset of the disease is generally sudden, attacking persons in apparently good health. In a few cases the onset may be insidious in character.

*Fever.*—A sharp rise of temperature is, perhaps, the most constant symptom met with, though some cases may run their course without fever. The range of temperature varies considerably. According to Wickman, it ranges from 99° to 102·2° F. In the New York epidemic of 1907, the temperature was somewhat higher, varying from 101° to 104° F. in most cases. The duration of the fever is generally short. It seldom lasts longer than 2 to 7 days. The few temperature charts that have been published in connection with poliomyelitis show that the fever generally runs an irregular course.

The degree of fever seems to be no criterion of the severity of the attack. The temperature in the so-called abortive cases may be quite as high as in cases in which extensive paralysis or death results.

*Prostration.*—This is a frequent early symptom of the disease. Muscular weakness and extreme weariness are characteristic features of many cases.

*Headache.*—In a large proportion of cases headache accompanies the fever, though in young children it may be difficult to determine whether it is present or not. Wickman states that it is generally occipital in character, while in the New York epidemic general or frontal headaches seemed to predominate.

Wickman's observations led him to the conclusion that the headache in poliomyelitis is seldom so severe as that met with in cerebro-spinal meningitis, where the patient frequently shouts out owing to its intensity.

*Gastro-intestinal disturbances.*—These have been very prevalent in certain epidemics, so much so, indeed, that some observers have, on this account, come to the conclusion that the virus gains entrance to the body by way of the mouth. Krause[14] found digestive disturbances present at the onset of 90 per cent. of all cases in an epidemic which occurred in Germany in the neighbourhood of Hagen.

Vomiting is met with not infrequently. It is not, however, usually very severe in character nor of long duration. This serves to differentiate the disease from cerebro-spinal meningitis, in which the vomiting is generally more severe and prolonged.

Diarrhœa and constipation are common. Constipation was present as often as diarrhœa in those cases in which the condition of the bowels was recorded in the New York epidemic.

*Catarrhal conditions.*—It is stated by Wickman[23] that catarrhal conditions of the respiratory system are not of very frequent occurrence, though symptoms of catarrh and bronchitis may usher in an attack of the disease. Sore throat and tonsilitis have been noted in connection with certain epidemics.

*Sweating.*—Müller[3] states that excessive sweating was a characteristic early symptom of the cases studied by him.

*Skin eruptions.*—Skin eruptions occurred in 61 out of 742 cases in the New York epidemic. The eruption was most commonly papular and covered the entire body. A skin eruption is, however, by no means characteristic of poliomyelitis. Herpes is, according to Wickman, very seldom met with, though other observers have frequently noted its presence.

*Retention of urine.*—Retention of urine has been occasionally reported.

### *Nervous Symptoms.*

*Drowsiness.*—Drowsiness is of frequent occurrence especially in children. The patient falls asleep and may remain sleeping for several days, only waking when he is aroused for the purpose of taking nourishment. This somnolence may occasionally merge into coma, but, if so, the comatose stage is generally of short duration. Delirium is of rare occurrence.

*Irritability.*—Irritability or restlessness is also very common. The Collective Investigation Committee on the New York epidemic state that the most marked features of that epidemic were the irritative, nervous symptoms at the time of onset and during the first few days, giving rise frequently to great difficulty in diagnosis.

*Pain and tenderness.*—The same Committee remark—"Almost all of the cases had pain and tenderness during the first few days. In practically half the cases it was quite marked. In many cases it was excruciating. It occurred most often in the lower extremities, next in frequency in the spine and trunk, and still less frequently in the upper extremities and neck. In regard to the pain and

tenderness, a peculiar feature of this epidemic seemed to be the occurrence of pain at the back of the knees or below the knees and in the calf."

This pain and tenderness may be a very marked symptom of the early stages of the disease and may be so intense that the patient can scarcely tolerate the weight of the bed clothes nor remain in the same position in bed for any length of time.

Pain is frequently present in the back of the neck and along the spine, and may be very severe in character. It does not generally last very long, frequently subsiding with or before the onset of paralysis. Numbness is sometimes found, especially in older children and adults. Wickman observed a case in which there was diminution of the sense of pain from the hips downwards together with paralysis of the lower limbs. This patient had also difficulty in distinguishing between heat and cold when they were applied to his feet.

*Meningeal symptoms.*—A large number of cases of poliomyelitis seem to have an onset closely resembling that of cerebro-spinal meningitis.

Pain, stiffness, and even rigidity of the neck are sometimes met with, rendering it painful or impossible to bend the head forward. Sometimes, in severe cases, the head may even be retracted from contraction of the posterior muscles of the neck. While very marked retraction of the head, such as is met with in cerebro-spinal meningitis, is uncommon, many cases of poliomyelitis occur in which the head is maintained farther back than normal. The presence of a modified Kernig's sign (inability to completely extend the leg when the thigh is flexed) renders the diagnosis still more difficult.

It is possible, as has been pointed out by Wickman and others, that in some of these cases we are dealing with a true meningitis of independent origin in addition to poliomyelitis. On the other hand, post-mortem examinations have shown that the pia mater is congested and infiltrated in the acute stages of poliomyelitis. Under such circumstances some of the above symptoms are to be expected in true cases of the disease.

*Muscular twitchings.* — Muscular twitchings, jerking, or tremor of the limbs are sometimes observed in this disease. They generally occur before paralysis sets in, but may also be seen in cases that do not result in paralysis. Such manifestations are due to disturbances of the motor centres.

*Reflexes.*—Wickman[23] believes that the condition of the patellar reflex is of importance in the diagnosis of poliomyelitis and makes the following statement with regard to it :—

1. The patellar reflex disappears. This is the rule. It should be noted that loss of the patellar reflex may be the only recognisable symptom of the disease (Wickman, Ed. Müller, Zappert).
2. There may be a preliminary increase followed by loss of the patellar reflex (Wickman, Ed. Müller).
3. When the crus or bulb is involved, an increase of the patellar reflex in the otherwise normal legs may be observed (Wickman, Neurath, Zappert, Foerster, Ed. Müller).

4. In connection with paralysis and loss of reflex in one leg there may be an exaggeration of the reflex in the other apparently normal leg (Wickman, Zappert).
5. Increase of the patellar reflex in a paretic and clearly atrophied leg.

The Committee that investigated the New York epidemic state that "a study of the reflexes established the general doctrine that in poliomyelitis the deep reflexes in the parts paralysed are absent and that they are often absent in non-paralysed parts."

In summary of what has already been said the United States Public Health Bulletin, No. 44, February, 1911, may be quoted.

"The characteristic features of acute anterior poliomyelitis in the early stage are sudden onset with fever, gastro-enteric disturbances (vomiting, diarrhœa, constipation), occasionally sore throat, headache, restlessness followed by apathy, pains in the neck, back, and muscles ; muscular twitchings, exaggeration or abolition of tendon reflexes. Symptoms of a rather mild meningitis are present in a varying proportion of cases, and when present are rather characteristic."

"The clinical picture prior to the onset of paralysis may be that of an indefinite general infection or toxemia, gastro-enteritis, tonsilitis, multiple neuritis, meningitis or encephalitis."

### *Types of the Disease.*

There are, according to Wickman, eight principal types of the disease, and, as most other observers have confirmed and adopted his classification, it may be given here.

1. *The spinal poliomyelitic type.*—This is by far the most common and easily recognised form of the disease. After the commencement of such preliminary symptoms as have already been enumerated, paralysis sets in. It is sudden in onset and of a flaccid motor type which reaches its maximum, both in extent and degree, within one or two days, and then goes no further. After that a retrogression generally takes place so that the paralysis (should it persist) finally involves a considerably less area and is much less marked than in the acute stage. Sometimes complete recovery of the affected muscles takes place.

The extremities are the parts most frequently paralysed. Statistics show that the lower limbs are affected twice as frequently as the upper. Combinations may, however, occur as—both legs, one leg and one arm of the same or opposite sides, both arms and one leg, &c.

The permanent paralysis may affect the limbs as a whole ; as a rule, however, it is confined to certain groups of muscles, and these ultimately become atrophied as a result.*

Besides paralysis of the limbs, paresis of the muscles of the back, neck and abdomen is occasionally met with.

* It is stated by Flexner that the proportion of cripples as a result of this disease in 23,000 cases was from half to three-quarters of the whole.

2. *The ascending or descending type.*—This strongly resembles Landry's paralysis. The paralysis generally begins in the lower extremities and gradually ascends until most of the musculature of the body becomes involved. Such cases often succumb from paralysis of the respiratory muscles. Very occasionally the paralysis is descending instead of ascending.

3. *Bulbar or pontine form.*—This form is characterised by paralysis of the parts supplied by the cranial nerves which have their nuclei in the medulla or pons. Thus the face and eyes generally become affected and there is frequently difficulty in swallowing.

4. *Encephalitic form.*—Here we have to deal with initial changes in the cortex of the brain followed by spastic monoplegia or hemiplegia.

5. *The ataxic form* resembles an acute ataxia, the ataxic symptoms being due to lesions of the cerebellum.

6. *Polyneuritic type* presents a clinical picture of multiple neuritis and is characterised by hyper-sensibility of the nerves, which gives rise to pain when pressure is exercised.

7. *Meningeal form.*—In this form two types of cases are met with. (1) Those with initial symptoms of meningitis followed by spinal or bulbar paralysis. (2) Cases in which the symptoms of meningitis are followed by paralysis.

8. *Abortive forms,* characterised by fever, headache, and stiffness of the neck, but without paralysis. Such cases can only be diagnosed —even with probability—during the prevalence of epidemics.

## *Diagnosis.*

The diagnosis of poliomyelitis, especially in the early stages of the disease before paralysis has set in, may be a matter of considerable difficulty. A typical case, with sudden onset of flaccid paralysis of the limbs following on acute febrile disturbance, is generally sufficiently characteristic, but, as we have seen, the initial symptoms are so varied and the types of paralysis so numerous as often to obscure the true nature of the disease.

Wickman lays stress on the following symptoms as being characteristic :—1. Drowsiness ; 2. Pain and tenderness ; 3. Stiffness of the neck ; 4. Profuse perspiration. According to Müller[3] the three cardinal prodromal symptoms are :—1. Profuse perspiration ; 2. Hyperæsthesia ; 3. Leucopenia.

Among the conditions with which poliomyelitis is most likely to be confused may be mentioned epidemic cerebro-spinal meningitis, influenza, and tuberculous and acute or septic meningitis. The following rough differentiation taken largely from a table drawn up by Wickman, may, perhaps, help to distinguish poliomyelitis from cerebro-spinal meningitis.

| *Epidemic Poliomyelitis.* | *Epidemic Cerebro-spinal Meningitis.* |
|---|---|
| 1. The disease is of the nature of an acute poliomyelitis, resulting in paralysis emanating from lesions of the spine. | Most cases present very marked symptoms of meningitis. |
| 2. Paralysis generally affects the limbs and is, in many cases, permanent. | Paralysis is rare with the exception of a transitory paralysis of eye muscles. |
| 3. Fever lasts 3–7 days and then temperature becomes normal. | In most cases, that do not die during the first few days, fever is prolonged and becomes of an intermittent or remittent type. |
| 4. Headache frequently present, but not excessively severe. | Headache very severe, so that patient shouts out or screams. |
| 5. Drowsiness is frequently present, but the sensorium generally remains clear. | Coma is frequent. |
| 6. Herpes may occur. | Is commonly met with. |
| 7. Rashes very uncommon. | Found in some cases ("spotted fever"). |
| 8. Deafness an uncommon sequela. | Deafness occurs in a fair percentage of cases. |
| 9. The meninges are seldom inflamed. | In most cases a turbid or purulent fluid is found in cerebro-spinal canal. |
| 10. Cerebrospinal fluid obtained by lumbar puncture clear. | Turbid. |
| Albumen slightly increased. | Albumen increased to a much greater degree. |
| Lymphocytes found. | Polymorphonuclear leucocytes generally present. |
| Bacteria nil. | Meningococcus present. |
| 11. Lymphocytic infiltration of pia mater. | Polymorphonuclear infiltration. |
| 12. Blood count—leucopenia with relative increase of lymphocytes. | Number of leucocytes is high, and there is a relative increase of polymorphonuclear leucocytes. |
| 13. Epidemics occur in summer and autumn. | They generally occur in winter and spring. |

In cases of the meningeal type of poliomyelitis, lumbar puncture, and determination of the presence or absence of the meningococcus is practically the only available means of differentiation from cerebro-spinal meningitis during life.

Examination of sections of the cord under the microscope will always reveal the true nature of the disease in fatal cases of poliomyelitis.

In certain forms of influenza many of the symptoms seen in the early stages of poliomyelitis may be met with. Catarrhal conditions of the respiratory tract are, however, of frequent occurrence in

influenza whereas they are rare in poliomyelitis. Further, the prevalence of influenza is greater in winter and of poliomyelitis in summer.

Tubercular meningitis has generally a more gradual onset than poliomyelitis; the headache is less violent and the disease runs a more regular course. Nearly if not all the cases end fatally.

In acute meningitis there is generally some recognisable cause such as suppurative disease of the ear or nose, disease of bone, injury, pyæmia or other acute illness. Spinal symptoms are, moreover, less commonly present.

## Part 2.—Epidemiology.

Probably the first epidemic of poliomyelitis to be recorded occurred in Louisiana, though its true nature was not recognised. The following description of it is given by Colmer.[4] "While on a visit to the parish in the fall of 1841, my attention was called to a child, one year of age, slowly recovering from hemiplegia. The parents, who were people of intelligence and unquestioned veracity, stated that 8 or 10 cases of either hemiplegia or paraplegia had occurred during the preceding three or four months within a few miles of their home. Some of these had recovered completely, others were improving."

Bergenholz in 1881 recognised and described an outbreak of poliomyelitis. The first important work on the subject was, however, contributed by Medin, who described an epidemic of 43 cases in Stockholm in 1887. Since that time epidemics have been recognised and probably have occurred with increasing frequency in different parts of the world. Lovett[5] gives the number of cases of poliomyelitis reported in the literature of the world as occurring in epidemics by five-yearly periods from 1880 to 1909, as follows:—

| —— | Cases. | Epidemics. | Average No. of Cases. |
|---|---|---|---|
| 1880–1884 ... ... ... | 23 | 2 | 11·5 |
| 1885–1889 ... ... ... | 93 | 7 | 13·0 |
| 1890–1894 ... ... ... | 151 | 4 | 38·0 |
| 1895–1899 ... ... ... | 345 | 23 | 15·0 |
| 1900–1904 ... ... ... | 349 | 9 | 39·0 |
| 1905–1909 ... ... ... | 8,054 | 25 | 322·0 |

After making allowance for the increase due to greater accuracy of diagnosis, it seems probable that there has been a definite and progressive increase in the occurrence of epidemics of this disease in recent years.

Holt and Bartlett[16] have collected records of 35 epidemics of poliomyelitis which occurred up till the year 1907, the chief being those which took place in Norway and Sweden in 1905 and 1906. Since then larger or smaller outbreaks have been reported in England, Germany, Holland, Austria, France, America, Canada and Australia.

Among the better known epidemics may be mentioned:—The Swedish epidemic of 1905, reported by Wickman,[2] the New York epidemic[12] of 1907, reported by the Collective Investigation Committee, and the Massachusetts epidemic,[13] 1907, 1908 and 1909.

The disease appears to have been prevalent in Germany in 1909. Krause[14] reported an epidemic in Westphalia, and it is estimated that there must have been over 1,000 cases in Germany, during that year.[15] Zappert[11] collected 266 cases in Vienna and Lower Austria in 1908 and 1909, and 24 cases were also reported from Leyden in Holland.

It is officially stated that acute poliomyelitis has, within the last year, appeared at various places in Norway. In some localities the number of cases has been considerable and "worse than any outbreak since the epidemic of 1905."

Lovett has shown that the United States of America have suffered more severely than any other country, 5,500 of the 8,000 cases reported from 1905 to 1909 having occurred in America.

To show how widespread has been the prevalence of the disease in that country, the following may be quoted from the Public Health Bulletin, No. 44, Feb. 1911. "From 1907 to 1910 outbreaks have occurred in the following States in this country:—Connecticut, 1910; District of Columbia, 1910; Florida, 1907; Illinois, 1909; Iowa, 1908–1910; Kansas, 1909–1910; Massachusetts, 1907, 1908, 1909, 1910; Michigan, 1907–1908; Minnesota, 1908, 1909, 1910; Missouri(?), 1908; Nebraska, 1909; New York, 1907–1910; Oregon, 1910; Pennsylvania, 1910; South Dakota, 1910; Virginia, 1908–1910; Washington, 1910; Wisconsin, 1908."

The Forty-first Annual Report of the State Board of Health of Massachusetts contains the following statement:—"Of the 8,054 cases reported in the last five years (number, of course, only approximately correct), the United States contributed 5,514 cases, or about five-sevenths of the total number of cases. The bulk of these cases has, moreover, been reported from the Northern States, the outbreaks in the Southern States being insignificant. In the same way in Europe, Norway and Sweden contributed about 1,500 cases, and Germany practically the rest, except for an outbreak in Australia, which occurred in March, which is their early fall.

"As the literature has been very carefully gone over, and as, in the present state of interest in the subject, it seems fair to assume that large outbreaks in any civilized country have been reported, it would seem that the following conclusions were justified:—

"1. That outbreaks of infantile paralysis have been greatly increased in several parts of the world in the last five years in a measure not to be explained in any way by the increased interest in the disease.

"2. That it is more prevalent in cold than in warm countries.

"3. That from the northern part of the United States have been reported more cases than from any part of the world."

The first mention of epidemic poliomyelitis in Great Britain is recorded by Dr. Pasteur[19] in 1897, who discovered and described the disease in seven members of one family.

In 1898 a lecture was given by Dr. Buzzard[18] "On cases illustrating the Infective Origin of Infantile Paralysis," in which some striking examples are given.

Dr. Treves[25] reported an epidemic of eight cases in 1908 at Upminster, a small village in Essex.

The first epidemic of any magnitude occurred in Bristol in 1909, and was the subject of an article by Dr. George Parker in "The British Medical Journal," of March 18th, 1911. There were 37 cases.

Dr. F. E. Batten,[20] in a valuable contribution to the epidemiology of poliomyelitis states that Dr. Garrow in 1910 discovered 13 cases of poliomyelitis in the town of Maryport, in Cumberland, besides scattered cases in various villages in the west of Cumberland, and 37 permanent cripples at Barrow-in-Furness, which he attributed to results of the disease. Thirty-four cases were notified in the city of Carlisle in the same year.

A small but interesting outbreak, which occurred in Clackmannanshire, not far from the town of Tillicoultry, was studied by Drs. D. W. Currie and Edwin Bramwell, a report of which appears in "The Edinburgh Medical Journal" for October, 1911. They also bring forward evidence which they believe justifies the conclusion that acute poliomyelitis was more prevalent than usual in various parts of England and Scotland during the autumn of 1910, and, further, that this increased frequency was as pronounced, if not more so, in the smaller towns and rural districts as in the cities.

From a consideration of the reports of epidemics of this disease, certain characters in its epidemiology to which different writers have drawn attention, may be noted.

*Seasonal incidence.*—In temperate climates epidemic poliomyelitis generally occurs in summer and autumn.

In America epidemics are, almost without exception, confined to the warm season, *i.e.*, May to November, and in the southern hemisphere from January to April (a season corresponding to the late summer or "fall" in America).

While this is so, however, it does not preclude the possibility of cases occurring at other seasons. Thus, the 1,025 cases which occurred in Sweden in 1905 had, according to Wickman, the following monthly distribution:—

| | | | | | |
|---|---|---|---|---|---|
| January ... | 1 | May ... | 7 | September ... | 243 |
| February ... | 5 | June ... | 20 | October ... | 140 |
| March ... | 4 | July ... | 137 | November ... | 69 |
| April ... | 4 | August ... | 367 | December ... | 28 |

In the New York epidemic of 1907 the date of onset of the cases was as follows:—

| | | | | | |
|---|---|---|---|---|---|
| January ... | 5 | May ... | 10 | September ... | 218 |
| February ... | 3 | June ... | 59 | October ... | 71 |
| March ... | 4 | July ... | 133 | November ... | 20 |
| April ... | 3 | August ... | 188 | December ... | 4 |

Date not stated 24.

*Age.*—There has been considerable variation in the age incidence as reported in different epidemics. This is well illustrated by the following figures taken from the Report of the Collective Investigation Committee on the New York epidemic of 1907.

| — | Wickman's, 1905. | New York. | Rutland. | Göteborg. |
|---|---|---|---|---|
| Up to 3 years... ... | 169 | 463 | 90 | 11 |
| From 3 to 6 years ... | 181 | 197 | 90 | 5 |
| From 6 to 9 years ... | 154 | 40 | 15 | 2 |
| From 9 to 15 years ... | 165 | 21 | 15 | 0 |
| Fifteen and over ... | 199 | 8 | 15 | 2 |

| — | Stockholm. | Smedjeb ackmer. | Skelby. | Gloucester (U.S.A.). |
|---|---|---|---|---|
| Up to 3 years... ... | 34 | 30 | 10 | 16 |
| From 3 to 6 years ... | 12 | 30 | 0 | 10 |
| From 6 to 9 years ... | 1 | 20 | 2 | 3 |
| From 9 to 15 years ... | 1 | 20 | 5 | 2 |
| Fifteen and over ... | 5 | 0 | 3 | 0 |

It will thus be seen that the majority of cases are in children under 6 years of age. The New York epidemic of 1907 is characterised by the large percentage of the cases reported among children—90·5 per cent. occurring in the first six years of life. In the epidemic reported by Wickman in Sweden, on the other hand, there were only about 40 per cent. of cases in the first six years of age, and 10 per cent. among adults.

It would seem that, in epidemics in which a comparatively large proportion of the population in a given area is attacked, the proportion of adult cases is high.[21] In the epidemic of Nauru,[6] for example (an island in Melanesia, with a population of about 2,500), where 700 cases of this disease occurred, it is reported that the majority of cases were in adults.

*Sex.*—Such statistics as are available on this point show that more males are affected than females. In Minnesota in 1909 the ratio was 193 males to 139 females. In the Massachusetts epidemic of 1909, 363 males and 263 females were attacked.

Hill[7] has shown that in Minnesota, the proportion of males and females affected was more nearly equal in the first decade of life, but, that after 10 years of age males were affected in much greater proportion than females.

*Fatality.*—The recorded fatality varies in the different epidemics. It was 12 per cent. in Sweden; 14 per cent. in Norway; 5 per cent. in New York; 13·16 per cent. in Upper Austria; and 5 per cent. in the small epidemics observed by Netter in Paris and its suburbs. Holt[8] gives the fatality as ranging from 6 to 29 per cent., and the Iowa Health Bulletin XXIV, July, August, and September, 1910, gives a rate of from 8 to 20 per cent.

Much will, of course, depend upon the manner in which these

statistics are compiled, and as to whether the so-called abortive cases are included.

In certain epidemics the fatality among older children has been higher than among the younger members attacked.

*Incubation period.*—The incubation period of poliomyelitis has not yet been accurately determined. From observations of the time elapsing between the first and subsequent cases in those instances where two or more cases occurred in the same family or same house, Wickman came to the conclusion that the incubation period is usually from one to four days.

Experiments have shown that monkeys inoculated with the virus generally sicken from 5–15 days after inoculation. It has been demonstrated, however, that the period is capable of modification according to the dose administered and the thoroughness with which the virus has been filtered.

*Density of population.*—Apparently density of population bears no constant relation to the prevalence of epidemic poliomyelitis. In Vienna it was found that the disease was not more prone to occur in the poorest and most overcrowded quarters of the town than in the better and less crowded districts. It has been noted in Sweden and in the United States of America that epidemics are more severe in small towns and rural communities—the larger cities, as a rule, suffering less in proportion to population.

### *Infectivity.*

As Dr. Gordon points out, poliomyelitis has been produced in monkeys by inoculating them with the submaxillary lymphatic glands or nasal mucous membrane of an animal affected by the disease. The tonsil and pharynx of a child dead of the disease have also been used with positive results for the inoculation of monkeys. Further, the disease has been experimentally produced by inhalation, by introduction of the virus by means of a stomach tube, by painting it on to the nasal mucous membrane or by injecting or rubbing it into that membrane after scarification.

It is stated by Osgood and Lucas, however, that, during the experiments performed by them, no case of transmission from monkey to monkey occurred although the closest association with subjects in the acute stage had been maintained both by contact and by the use of the same feeding utensils and food.

The laboratory experiments referred to indicate that the disease is capable of being transmitted from person to person by direct contact, a conclusion already reached by Wickman and others from epidemiological studies.

Some of the facts upon which such an assumption is based may here be enumerated.

*Occurrence of multiple cases in houses.*—The number of cases occurring in the same house is of interest as bearing upon the communicability of the disease. Wickman gives the following figures for the Swedish epidemic of 1905 :—

| No. of cases in each house | 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|
| No. of houses ... ... | 627 | 97 | 39 | 14 | 7 | 1 |

In the above figures the so-called abortive cases are included. When these are left out the table becomes as follows for the houses in which at least two cases occurred :—

| No. of cases in each house ... ... | 2 | 3 | 4 | 5 |
|---|---|---|---|---|
| No. of houses ... ... ... ... | 76 | 15 | 4 | 2 |

In the New York epidemic the cases studied were distributed as follows :—

| No. of cases in each house ... ... ... | 1 | 2 | 3 |
|---|---|---|---|
| No. of houses ... ... ... ... ... | 700 | 18 | 5 |

The Committee who undertook the investigation of the New York epidemic make the following statement :—

"A study of the various epidemics leads to the conclusion that there is considerable variability both in the infectivity and the virulence of the causative agent. We have already noted the large proportion of multiple infections in the Swedish epidemic as compared to our own outbreak. So also in the small epidemic in St. Mary's, Canada, in 1908, out of a total of 17 cases, three instances were observed of two cases in one family." . . . "The degree of infectivity does not necessarily correspond to the degree of virulence. The virulence of the infection can be measured not only by the mortality of the disease, but also in many instances by the proportion of adults affected and by the period of incubation."

In addition to instances in which two or more cases have been observed in the same family or the same house, instances have frequently been reported, in different epidemics, in which cases among acquaintances or neighbours were noted.

*Infected houses.*—Cases are quoted by Wickman where the disease appeared successively for two or three years in the same house. He also records that persons, coming into a house previously invaded, themselves contracted the disease.

*School epidemics.*—The observations of Wickman led him to the conclusion that schools may play a part in the propagation of the disease.

At Trästena, where 49 cases occurred, Wickman traced the cause of contagion to children who attended a school in the neighbourhood.

Of those who took ill, four children belonged to the school-teacher; 25 to families of which the first infected child attended the same school; 12 to families who had healthy children attending school (the infected children being kept at home); lastly, four persons from families that had only indirect communication with the supposed seat of infection. This leaves a residue of only four cases who had apparently no ascertained connection with the school.

Of the 49 cases, 23 were of the abortive type and 26 suffered from paralysis. Eleven of the paralytic cases died. There were 102 houses in the village of Trästena, 19 of which were invaded. The numbers affected in each house were as follows :—

| No. of cases in each house ... | 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|
| No. of houses ... ... ... | 6 | 3 | 5 | 3 | 2 |

Netter came to a similar conclusion in connection with an outbreak of poliomyelitis at Créteil. Seven children were attacked

one after the other with infantile paralysis. Their homes were far apart and their parents did not visit one another. Three of the children went to the same school. Two of the others (too young to attend school) had a brother and cousin respectively attending the school. Netter was of opinion that the infection was spread at the school by the three scholars who had contracted the disease and by the two scholars who, without having the disease themselves, nevertheless infected their brothers and cousins.

*Transmission by abortive cases or by healthy carriers.*—Many writers consider that a principal share in the epidemic spread of poliomyelitis is borne by abortive cases, without characteristic paralysis or severe illness, and that even healthy persons, who have never had the disease, may harbour the infection and be able to transmit it to others—playing a role similar to "carriers" in enteric fever and diphtheria. As regards "carriers" these views appear so far to be based on circumstantial evidence. In view of the importance of the operation of abortive cases, the following notes are given with regard to their occurrence.

In epidemics, cases of the disease are met with showing much diversity in the degree and extent of the paralysis. In the same group may be found cases resulting in extensive and lasting paralysis; cases with permanent paralysis of slight extent; cases of transient paralysis recovering completely within a few weeks or even a few days; other cases in which there is no definite paralysis but only muscular weakness of short duration, and so on.

The diagnosis of poliomyelitis is these cases is suggested by their close association with typical cases of the disease rather than by the distinctiveness of their symptoms. The manifestations are, however, sufficiently similar to the pre-paralytic symptoms observed in paralytic cases, and generally sufficiently different from the symptoms observed in the more common infectious diseases.

It is further observed that monkeys inoculated with the virus of poliomyelitis occasionally pass through an indefinite illness without resultant paralysis, clinically similar to the abortive attacks observed in man.

The symptoms generally met with correspond closely with the initial symptoms of pronounced cases of the disease. Thus fever, sometimes of very short duration, is often observed. Headache is one of the most constant features, generally accompanied by an unusual degree of physical weakness. In some cases nausea and vomiting, associated with diarrhœa or constipation, are the most prominent symptoms. Drowsiness and irritability in children, and restlessness and mental anxiety in older persons are common.

Wickman describes four clinical types of abortive cases:—

(1.) With symptoms of general infection.
(2.) With gastro-intestinal disturbances.
(2.) With pain and hyperæsthesia (resembling neuritic influenza.)
(4.) With meningeal symptoms (severe occipital headache, pain, tenderness and rigidity in neck accompanied by pain and tenderness of the back.

It is difficult to determine, with accuracy, the frequency of abortive cases. Wickman found 15 per cent. in the Swedish epidemic of 1905.

The Massachusetts State Board record 49 cases of illness, possibly abortive cases of poliomyelitis, occurring in the same houses with the 150 pronounced cases.

In the epidemic which occurred in the island of Nauru in 1910 Müller states that many cases of the disease recovered without paralysis. Only 50 of the 700 attacked had paralysis remaining after three months.

Muller[24] lays stress upon the fact that the disease in Hesse-Nassau seemed to be conveyed less often by infected children than by their apparently healthy brothers and sisters. He is also of opinion that adults, who have been in contact with infection, may act as " carriers " of poliomyelitis, and gives some striking instances of circumstances pointing to such modes of infection.

*Experimental investigation of abortive cases.*—It is possible, by means of a neutralization test to determine, in a given instance, whether a person has been affected with poliomyelitis, even should no signs of paralysis have presented themselves.

The test is performed by mixing the blood serum of the suspected case with the filtered virus of poliomyelitis, and incubating the mixture at 37° C. for a few hours. It is then injected into a monkey.

Normal human serum has little or no power to neutralize the virus of poliomyelitis, while the serum of recovered cases possesses this power.

While such a test is useful to prove that abortive cases do occur, it is, needless to say, of no use as a means of routine diagnosis. The recognition of these cases, therefore, which may play a very important part in spreading the disease, must be attempted by clinical observations alone.

Osgood and Lucas[22] have demonstrated by experiment that the nasal mucous membrane of two monkeys inoculated with poliomyelitis remained infectious for six weeks and five and one-half months, respectively. This observation strengthens the suspicion of the existence of human carriers and indicates that prophylactic measures directed only to persons in the acute stage of the disease are unlikely to be successful.

*Food as a source of infection.*—Wickman believes that food, especially milk, may transmit the infection of poliomyelitis. In a small epidemic described by him, the first case became ill on 6th October, with high fever, drowsiness, and tremors, but without developing paralysis. The father of this patient was a dairyman. Another son of this dairyman became ill on the 20th October and exhibited paralysis of the legs. Eight other cases became ill. Four were simultaneously attacked on 20th October. These four belonged to three families, all of which obtained their milk supply from the above dairyman. Wickman is of opinion that milk was the source of infection in this instance.

*Other articles as a source of infection.*—The following observation of Wickman is of interest in this connection :—

On 27th July, in Söderköping, a young man of 22 years of age, became ill with poliomyelitis accompanied by paralysis of one leg. Some time after his illness he travelled to Stockholm and made some sketches while convalescing there. Some of these were

sent to an office for reproduction. On 25th September a lady of 34 years of age, who took charge of the sketches at the office, was attacked with paresis of both legs and complete paralysis of the left thigh.

The population of Stockholm is about 300,000. During the year 1905 there were only 11 known cases of poliomyelitis in the city. As no other source of infection could be traced, Wickman infers that the contagion was conveyed by the sketches.

*Dust.*—The occurrence of epidemic poliomyelitis in the hot, dry, dusty season has given rise to the surmise that dust may possibly be a factor in the spread of the disease. It has further been suggested (as the horse is said to suffer from a similar disease) that the infective agent in dust may be horse manure.

*Insect transmission.*—It has been suggested that insects may possibly play a part in the transmission of this disease. Flexner and Clark[9] have shown that house flies contaminated with the virus of poliomyelitis harbour the virus in a living and infectious state for at least 48 hours.

Transmission of infection by biting insects has also been suggested.

*Epidemics among domestic animals.*—It has been stated, in connection with a number of epidemics, that domestic animals—including chickens, dogs, horses, pigs, cattle, and sheep, were found, in the same district, to be suffering from paralytic diseases clinically similar to the disease prevailing among human beings.

The lumbar cord of a chicken, examined by Dana,[10] of New York, showed lesions resembling those of poliomyelitis.

A number of medical men in America have reported the occurrence of a disease in the horse resembling poliomyelitis. In this connection the observations of Dr. Shore,[20] who carries on veterinary practice, are of interest. He noted a disease appearing among one and two-year-old colts that showed symptoms corresponding closely to poliomyelitis. These cases always occur during the summer months, and the majority of them during August.

Krouse, of Bonn, reports the occurrence of a paralytic affection in chickens.

The similarity between poliomyelitis and rabies has led certain observers to the conclusion that the former must also be derived directly or indirectly from the lower animals, but nothing has so far been definitely ascertained with regard to the matter.

The Committee who investigated the New York epidemic state—"It was impossible to learn of any infection of the lower animals which might have had any relation to the disease in question."

### *Drug treatment of the disease.*

It was claimed by certain clinical observers[17] that urotropin administered in large doses had a beneficial effect on the disease. This led to experimental work being undertaken.

It is found that, when a large dose of urotropin is administered by the mouth to monkeys, its presence can be demonstrated, by chemical tests, in the cerebro-spinal fluid soon afterwards.

If the virus of poliomyelitis is injected into the nervous system of monkeys in which urotropin is already present in the cerebro-spinal fluid, and if the drug is administered by the mouth daily

thereafter, it is found in a proportion of the animals so treated but not in all that—

(1) The incubation period is prolonged.

(2) The onset of paralysis is entirely prevented.

Further, if urotropin be administered to monkeys at the same time as the protective serum is being used it seems to increase the action of the serum.

It is difficult to determine, clinically, in human beings, whether urotropin is of value, but the above experiments seem to show that its possible usefulness should not be lost sight of.

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