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THE NATURE OF POLIOMYELITIC INFECTION
AND THE TREATMENT OF THE ACUTE STAGES
OF THE DISEASE

by

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INTRODUCTION.

Poliomyelitis has been a subject of increasing interest to the Medical Profession in Northern Europe and America of recent years, on account of the great increase in the number of epidemics, and of the serious nature of the disease.

Investigations, both experimental and epidemiological, have been carried on in different countries, and, as a result of these, it has been established that the disease, which was formerly regarded as a primary cell-degeneration of the anterior horns of the spinal cord, giving rise to paralysis, and occurring only in children, is, in point of fact, an acute specific infection involving any region of the central nervous system, and occurring at any age.

In this thesis, I propose:-

- I. To give a short account of the recent experimental work done.
- II. To describe the nature of the infection, i.e. its pathology, as indicated by these researches.

- III. To discuss the epidemiology in this country, in the year 1911, with reference to the mode of spread of the virus, and
- IV. To describe a series of 60 cases admitted to the Hospital for Sick Children, Great Ormond Street, London, during the year 1912, with a view to illustrating the clinical manifestations, and indicating the treatment.
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I.

HISTORY OF RECENT INVESTIGATIONS.

Early Studies.

Poliomyelitis was first described in 1840 by Heine. The disease appeared in epidemic form in 1881, though not to any great extent until 1903, when 1,151 cases with 167 deaths, were recorded in Norway by Harbitz and Scheel. In 1905 another severe epidemic visited Scandinavia, and Harbitz, Scheel and Giersvold recorded 719 cases with 111 deaths from Norway, while from Sweden Wickman recorded 1,031 cases with 145 deaths. The latter observer, by a careful study of this epidemic in Sweden, was able to throw much new light on the nature of the disease. He was the first to point out its infectiveness, by tracing a connection between many of the cases. He also recognised

abortive and meningeal types of the disease, and concluded that one of the means of its dissemination, at least, was through healthy human carriers.

Inoculation Experiments.

The mystery surrounding the disease was not elucidated till 1909, when Landsteiner and Popper succeeded in conveying Poliomyelitis to monkeys. Flexner and Lewis carried the investigation further, later on in the same year. These observers used, as did Landsteiner and Popper, an emulsion of the spinal cord of a human fatal case. They injected this into the subdural space instead of into the peritoneal cavity as the former observers had done. Then by taking the spinal cord of affected monkeys and by injecting the emulsion again by the same route, they succeeded in carrying the disease, identical with that in man, through an indefinite series of monkeys.

These experiments then established, not only the existence of a specific virus, but also its infective nature.

Further experiments by Flexner and Lewis showed that an emulsion of infected spinal cord with saline solution when passed through a Berkefeld filter, was capable of transmitting the disease. This fact proved the parasite of Poliomyelitis to be filterable.

Cultivation.

All attempts to cultivate the specific organism

however, were fruitless, in spite of the trial of a variety of media and cultural conditions. At last this has been accomplished by Flexner and Noguchi, who have recently reported their success; and monkeys inoculated with the cultures have developed typical Poliomyelitis.

II.

NATURE OF INFECTION.

(A) Nature of Virus.

The organism is so minute that it has not been found possible to detect it by the maximum power of modern microscopy. There are eighteen infective diseases whose organisms belong presumably to this class of "invisible virus". These viruses are likewise filterable: they have been passed through the finest porcelain filters, and the filtrate has produced the particular disease.

Flexner has pointed out that the Poliomyelitic virus is intermediate in size between those viruses that pass the finest, and those that pass the coarsest Berkefeld filter, because the filtrate obtained after passage from the finer varieties is slower in its action in the production of disease, than is that from the coarser ones, and still slower than the unfiltered emulsion.

Immunization.

One important observation has been made in this connection, namely, that the number of organisms in the filtrate may be so far reduced, that it is possible to bring about, on inoculation, a degree of infection so slight that no symptoms result, but a high degree of immunity is established. This forms a basis of a possible specific treatment by vaccine.

Preservation.

Flexner has preserved the virus in an active state for several months, in a 50% solution of glycerine and water.

Resistance.

The same observer, with Lewis, has shown that drying for 24 days at a temperature of 25° Cent. did not affect the virulence.

Leiner and Wiesner found that the virus was destroyed by a temperature of 55° Cent. in half an hour, but was not destroyed by a temperature of -8° C. Its resistance to chemical action has been demonstrated by Landsteiner and Levaditi. They showed that the virus was killed by a 0.2% solution of potassium permang., at a temperature of 39° C. in one hour; that 6% peroxide of hydrogen at the same temperature, killed it in three-quarters of an hour; but that 1½% Carbolic Acid did not destroy it.

Cultural Characteristics.

The cultural characteristics are described by Flexner and Noguchi. They describe minute colonies clouding the tube, the colonies themselves being composed of rounded bodies occurring singly, doubly, in chains or in masses; the bodies staining a pale reddish violet by Giemsa's stain.

One of the observers (Noguchi) has also found similar bodies in films taken directly from the nervous tissues.

C. f. Rabies.

The analogies of this virus to that of Rabies is very striking and worthy of note.

The rabic virus is also one of the filterable viruses. It can be kept in glycerin without affecting its virulence. It is found in the brain, spinal cord and nerves, but not in the blood, lymph or internal organs, which corresponds to the distribution of the Poliomyelitic virus as will be pointed out later. It has been experimentally inoculated successfully into the cerebrum, but has failed to produce the disease when given by the mouth. In connection with this latter characteristic Flexner has shown that the secretions of the stomach apparently render the virus of Poliomyelitis inert. Both viruses give rise to paralysis. The pathological effects produced in the central nervous system

by the Rabic virus, very much resemble those resulting from Poliomyelitic infection, and will be described under that heading.

Serum Test.

It is possible now to establish the diagnosis in all cases by testing the serum of the patient under observation, for its viricidal power, as shown by Landsteiner and Levaditi.

A 50% emulsion of spinal cord containing the active virus is taken and mixed with an equal quantity of the serum to be tested, the mixture being made at a temperature of 34° C., and kept for several hours at room temperature. Of this mixture .6 - .8 c.c. is injected intracerebrally into one monkey, and the same amount of the virus alone, injected into another. If the case is one of active Poliomyelitis, the monkey injected with the serum plus the virus does not develop the disease, but the other one does become infected.

(B) Paths. of Infection.

Location of virus in body tissues.

Up to the present, the virus has been found in the tissues of the central nervous system, and peripheral nerves, and in the mesenteric lymph nodes of the internal organs, but ^{not} in the internal organs

themselves, nor yet in the bone marrow. It may still be possible, with cultural facilities, to demonstrate the virus even in these situations, though it apparently does not exist here in sufficient amount to produce the disease on inoculation. The virus has been found also in the mucous membrane of the nose and throat, in the nasal secretion, and in the secretions of the stomach and intestines. In all these sites, with the exception of the stomach, the virus multiplies very rapidly. The cerebro spinal fluid probably is a medium for the dissemination of the virus in the C.N.S., but apparently the virus does not multiply in this fluid. Neither does it survive in the blood for very long.

By whatever route the experimental inoculation is made, whether through the peritonium, cerebrum, peripheral nerves or skin, or by applying the infected material to the nasal mucosa, the ultimate distribution in the C.N.S. is the same in every case.

Mode of spread to C.N.S.

From the site of inoculation, the virus finds its way directly by the nervous tissues, or by the lymph channels accompanying the peripheral nerves, to the medulla, spinal cord, and meninges. Flexner founded this knowledge upon the observation of an interesting experiment. He destroyed a monkey 48 hours after inoculating its nasal mucous membrane

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with the virus, and after removing the brain and cord, he separately inoculated emulsions of the medulla, spinal cord and olfactory bulb into other monkeys, and produced the disease only in the monkey which had received the emulsion of the olfactory lobes. From this he concluded that infection must proceed along the course of the olfactory nerves, into the bulbs and C.N.S., since, had the infection travelled by the blood stream, the only other possible route, he would by that time have found it in the more distant parts of the N.S., the medulla and cord, as he pointed out, having a greater affinity for the virus.

In support of this, Landsteiner and Levaditi showed that paralysis always occurs in the limb corresponding to the nerve trunk injected with the virus, and Leiner and Wiesner found that, by sections of the nerve trunk above the point of inoculation, infection of the animal can be prevented.

It is known that monkeys cannot be infected through the unbroken skin, that infection cannot take place through the stomach or intestines, unless their function be interfered with by the administration of opium; and that the virus passes with difficulty, or not at all, through lung tissue.

Therefore it is reasonable to infer that one site at least, if not the main one, of accidental infection with Poliomyelitis is the nasal mucosa. This inference

is substantiated by what we have seen to be the means of transit of the virus along the course of the peripheral nerves, by the knowledge that the terminals of the olfactory nerves are the most exposed of any nerve terminals in the body, and are in intimate relation to the brain; and also by the fact that Flexner has been able to show that the disease can be produced in monkeys by merely painting the infected material on to the surface of the nasal mucous membrane.

Flexner has also demonstrated the virus in the nasal mucous membrane of monkeys experimentally inoculated - and Wickman in Sweden has found the virus in the nasal secretion of a human abortive case - seven months after the onset of the illness.

Thus the nasal mucosa can be the mode of exit.

The establishment of this mode of entrance and of exit is of considerable importance since it provides the possibility of checking the spread of epidemics, by nasal antiseptic douching.

(C) Incubation Period.

With regard to this, there seems to be no definite limits established. Flexner gives the interval as being from two days to five weeks in monkeys.

The incubation period both in man and monkeys is apparently dependent on the amount and virulence

of the virus and the power of resistance on the part of the subject. It was shown under the heading (A) that the smaller the amount of virus inoculated, the longer was the interval before evidence of the disease was observed. It is well known that the virus in different countries does vary in its power of infectiveness in monkeys, and in all probability different strains occur in human infection. The individual resistance in this, as in all other infections, is a variable factor, and has to be taken into account.

During the period of incubation the virus multiplies at the site of inoculation, and travels along its nervous path to that situation in the brain or spinal cord where it is to produce its destructive work.

(D) Anatomical Changes.

The effects produced in man by accidental infection, and in monkeys by experimental inoculation of the virus, are identical.

The most characteristic changes are seen in the spinal cord, and are of two types:-

- (1) A mono-nuclear cell in filtration of the pia arachnoid, surrounding the vessels of the cord, and passing in with them to the grey and white matter:
- (2) A degeneration of the nerve cells in the grey matter of the anterior, and more rarely of the posterior horns.

Sometimes the one change is more marked than the other, when there is much injury to the nerve cells, paralysis is more marked; when the lining membrane of the vessels is more especially affected, the clinical signs are apt to simulate meningitis.

The nerve cells are probably subjected to a further degree of injury through the interference of the vascular supply, by the cellular invasion.

Accompanying these changes haemorrhage, hyperaemia and oedema of the tissues are found.

III.

EPIDEMIOLOGY IN GREAT BRITAIN.

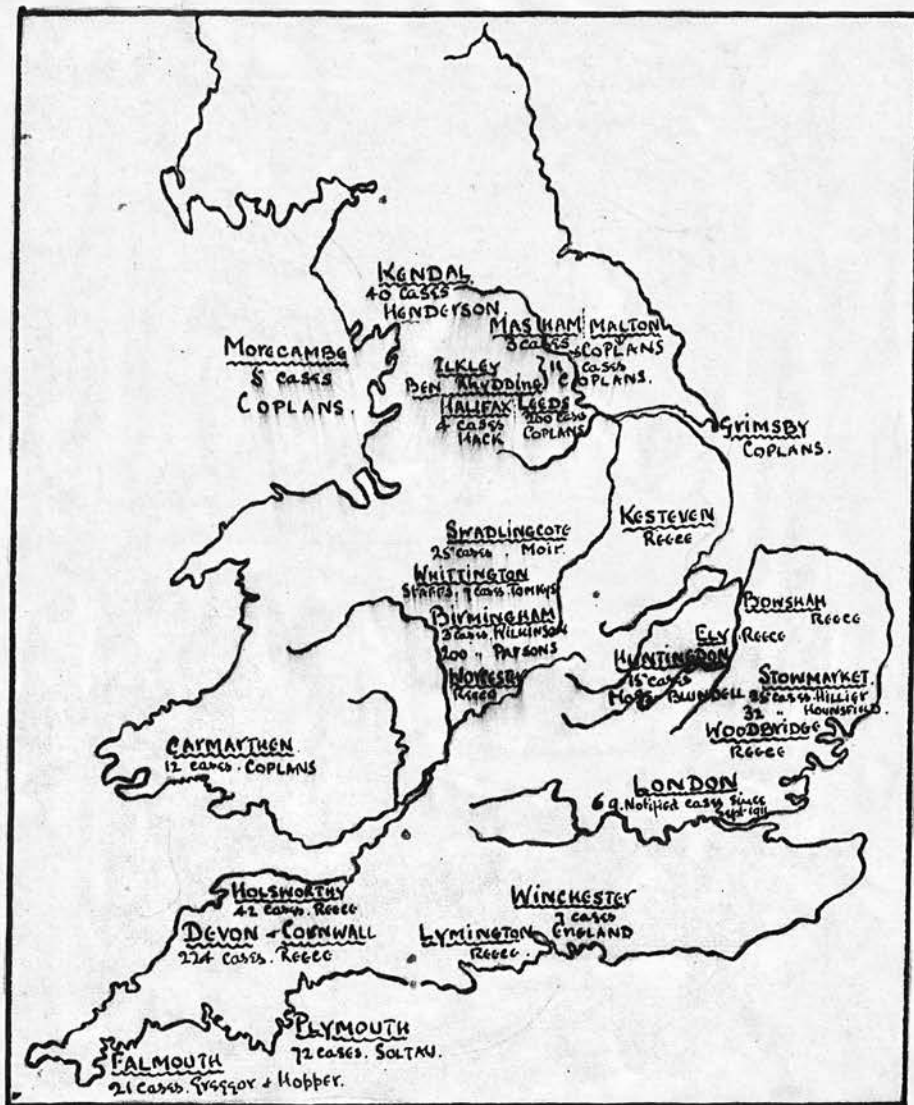
The earliest epidemic described in England was in 1896. Pasteur then recorded 7 cases which occurred at Much Hadham in one family. In 1908, a small outbreak of 8 cases was reported by Treeves from Upminster in Essex. In 1909, Parker reported 37 cases in Bristol, 2 of the cases dying. Dr. F. E. Batten has collected and described records of 26 epidemics from 1907 to 1910 in all parts of the world, including ten epidemics occurring in England and Scotland in 1910. These were in Melton Mowbray, 83 cases; Irthlingborough 4 cases; Cerne Abbas 16 cases; Weymouth 5 cases; South Shields 5 cases;

Barrow-in-Furness 37 cases; Maryport 13 cases, 2 deaths; Workington 5 cases; Carlisle 34 cases with 5 deaths and Tillicoultry 5 cases.

In the following year, 1911, which was one of the hottest and driest on record, there was a great increase in the number of cases. A glance at the accompanying map of England will show the distribution of the epidemics reported, and the numerical extent of the prevalence during that year. I do not propose to make a complete survey of all these, but some special features of the larger outbreaks, as described by the recorders are worthy of notice. A summary of these would seem to show that dust plays an important part in the spread of infection, though of course many other factors are undoubtedly concerned.

Gregor and Hopper reported 21 cases in Penryn in Cornwall. Their report states that this town has 3,000 inhabitants. The population is mainly of the working class type. The houses are old, and sanitation is very imperfect. The streets are badly looked after and the town is generally dirty. Five out of nine of the cases of Penryn occurred in the main road, along which there is a considerable amount of motor traffic, and this is the only playground of the children. All but four cases occurred in children under twelve.

In Huntingdon, 12 cases occurred in a small



Map of England, showing the places at which epidemics of Poliomyelitis occurred in 1911, the number of cases in the epidemics, & the name of the recorder.

village within an area of 200 yards, and 3 more cases in a village one mile away. In a report of these by Moss-Blundell, he states that the outbreak took place in the hot months of August and September. The school was closed for the holidays. The ages of the children were from $1\frac{1}{2}$ to 10 years. Constipation was a prominent feature in all except the last three, and the mothers noted that where one member of a family was attacked, some other member had malaise and diarrhoea, but developed no symptoms of the disease. There were no factors in the homes and surroundings to throw any light on the means of spread. All the cases, with one exception, occurred in houses off the main street where the traffic is very light, and the road treated with tar.

Hillier reported 25 cases from Stowmarket, Suffolk, all of which occurred between August 12th and September 18th. In three instances more than one case occurred in the same house. In one house there were 3 cases, and in two there were 2 cases. The majority of the remaining 18 cases occurred in groups in adjacent houses. Five were adults, the others children under 12 years. There were six fatalities, four from respiratory failure, and two from bronchitis and heart failure. At this time it was very hot weather, dust was very thick and swarms of flies were about. The measures adopted to cope

with the outbreak were, compulsory notification, isolation, destruction of flies, watering the streets with antiseptics, contacts were advised to spray nose and throat with permanganate of potash solution and householders were warned against the accumulation of refuse.

The sudden cessation on September 18th was not attributed by Dr. Hillier to a change in the weather, as after that date 32 cases occurred in the districts around. He stated that the only measure thoroughly and efficiently performed was the watering of the streets.

Hounsfield reported the other 32 cases occurring in the same district in September. The houses attacked were off the main road. He noted that nearly all the cases started with typhoid symptoms.

From Swadlingcote in Derbyshire 25 cases were reported by Moir. They occurred from June to August, and all were in children under twelve. Five cases were of the encephaloid type and proved fatal. Two deaths occurred from the spinal type. The district was an industrial one. There was no evidence to show in which way the disease was spread. Twelve cases occurred in very poor and dirty houses, and there was practically no communication between these.

Soltau recorded 72 cases from Plymouth. He found no clue in faulty sanitation and over-crowding, as the

disease attacked mostly the better classes, with clean well-kept houses. Most of his rural cases were on the course of the main roads where there was motor traffic. He noted a large number of cases of dust catarrh and laryngitis, occurring at the same time as his epidemic of Poliomyelitis, and catarrh of throat and nasal passages was a constant prodromal symptom.

Reece in a report on Poliomyelitis in Devon and Cornwall, was unable to draw any definite conclusion as to dust being a factor in conveying the disease. He pointed out an interesting association of paralysis in various animals, occurring along with the epidemic.

SUMMARY.

To draw anything but the broadest inferences from the perusal of these reports as to the mode of spread of Poliomyelitic infection, would be very difficult and unsafe.

Direct Transmission.

That the disease is transmitted from case to case is evident, from the study of any epidemic.

Carriers.

Swedish investigations have demonstrated the presence of the virus in the nasal secretion of an abortive case, seven months after the onset of the illness. It has also been found in the nose and throat of healthy persons who had been in contact with others acutely ill. This strongly suggests the

existence of healthy carriers of the infection.

Domestic Animals.

The resemblance of Poliomyelitis to Rabies suggests the possibility of an animal infection, but it has yet to be proved whether the various types of paralysis which occur in horses, pigs, dogs and fowls, have any common source of infection with Poliomyelitis in man.

Flies.

The great prevalence of flies in the summer of 1911, remarked upon in several of these reports, suggests another mode of dissemination.

Flexner has shown that the house fly can harbour the virus in an active state for a period of 48 hours. This may well be a means of conveyance in the summer months, but cases do occur frequently in winter when no flies are about.

Dust.

With regard to dust infection, Thro and Neustaedter demonstrated the virus of Poliomyelitis in dust collected in a room occupied by an acute case. This proves that the virus exists in an active state in dust. The greatest incidence of the disease in this year of 1911 was in the small towns and rural districts, and it is here that dust is much more prevalent, since motor traffic - a potent means of stirring up dust from the roadways - has affected country districts much more

of late years. These places have not the same facilities for laying dust as the larger and wealthier towns, where the incidence is proportionately less.

Hillier in his report of the Stowmarket epidemic, drew attention to its sudden cessation following on the watering of the streets, and he was led to suspect thereby that dust was the mode of spread in his cases.

Soltau, also, in a study of the Plymouth epidemic, was inclined to be of the same opinion, as most of his rural cases occupied houses on the main road.

It is true that other recorders have noted cases persistently, far removed from main roads, but it is quite possible that the affected persons in these situations might have been in proximity to dusty roads, or become infected by fine dust carried in the air. Strong evidence in favour of this mode of infection comes from Gregor and Hopper's report of the Penryn epidemic, in which so many of the cases occurred along the dirty main street, which was the common playground of the children.

Though no definite deduction can be made from a study of these epidemics, as to Poliomyelitic infection being dust born, yet there is presumptive evidence that dust is an important vehicle in disseminating infection in a very considerable proportion of instances.

Prevention of Infection.

In the light of our knowledge concerning Poliomyelitis in general, which has been briefly reviewed in the foregoing pages, we have means at our disposal for controlling the spread of the disease when occurring in epidemic form.

Isolation.

In the first place, isolation of both patient and family should be insisted upon. The patient should be sent to a fever hospital for four weeks at least, and the family kept apart from others as far as possible, for two or three weeks.

All contacts should be instructed to spray the nose and throat with a solution of peroxide of hydrogen, or permanganate of potash. In addition

Urotropine: they should be given urotropine in frequently repeated, small doses. The use of this drug in the treatment of infections of the central nervous system, was recommended by Crowe of New York, on the grounds that it liberates formaldehyde during the process of disintegration within the body, and is found in the cerebro spinal fluid shortly after administration in sufficient amount to make this an unsuitable medium for the continued growth of organism.

Flexner and Lewis have proved its value in

Poliomyelitis. By injecting the virus into a monkey in whose cerebro spinal fluid urotropine was already present, and continuing the administration of the drug, they found that the incubation period was prolonged from 6 to 24 days, and that the onset of paralysis was entirely prevented. Therefore it should be found of use both in preventing and in treating this disease.

Sanitation.

Efficient sanitation must be maintained, and measures taken for laying dust in towns and villages.

Distribution of Poliomyelitis in London, 1911-1912.

Poliomyelitis has been notifiable in London since September 1st 1911. For the last four months of that year 69 cases were reported. In 1912 136 cases were notified. Probably the cases throughout 1911 if accurately known, would exceed those of last year, judging from the prevalence of the disease elsewhere in 1911.

I have indicated by a chart, the districts of London in which these cases occurred. The first figures show the number for the last four months of 1911, and the last figures the numbers for 1912. It will be seen that the disease was fairly evenly distributed in this city, and showed no marked prevalence in any one district. (Vide Chart No. I.)



Map of Londondistrict, showing the distribution of cases of Poliomyelitis reported during the last four months of 1911, and 1912.

The first number represents 1911, the one placed last, 1912.

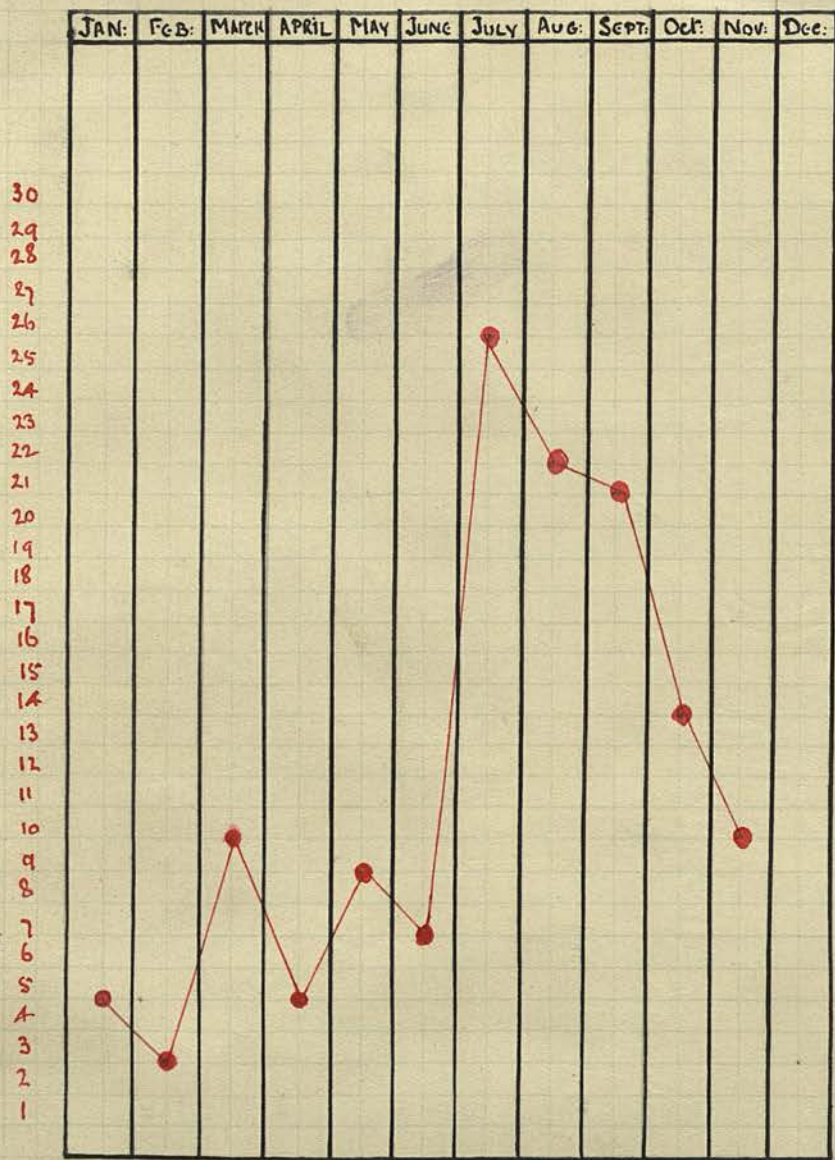


Chart showing the seasonal incidence of Poliomyelitis in London, in 1912.

The seasonal incidence of the disease in 1912, I have recorded graphically; it requires no further note. (Vide Chart No. 2)

IV.

DESCRIPTION OF CASES ADMITTED TO THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET, LONDON, IN 1912.

During last year, 60 patients, under the age of twelve, were treated in the wards of this hospital, for Poliomyelitis, and a synopsis of the cases is appended.

The youngest was 11 weeks old, and the eldest 11 years.

The children were admitted in varying periods after the onset of paralysis - as recently as the third day, and as late as five years. The majority were after the lapse of a few weeks, so that treatment was directed to the alleviation of paralysis, and the correction of deformities. Most of the children came from the districts in and around London, but a few were from more distant parts of the country, such as Glasgow, York, Exeter, etc.

35 were males, and 25 females.

No deaths occurred directly from the disease, but

one succumbed while in hospital from measles and broncho pneumonia, and the post mortem findings in this case will be described later.

Clinical Manifestations.

Onset.

In 8 of the cases, no premonitory signs or symptoms were observed by the parents, before the onset of paralysis.

General Symptoms.

A sudden illness with feverishness was the usual account obtained. Accompanying this were vomiting in 14 cases; headache - usually present only when paralysis was in the shoulder, thorax or upper limbs - in 8 cases; pain in the affected limbs in 27; and in the abdomen in only 2 cases. Pain did not appear at once, but, with one exception, it commenced before any evidence of paralysis, and usually in the muscles of the limb that became eventually most severely paralysed. It was generally acute pain, as the children would cry out if disturbed in any way. In several there was pain on passive movement noted weeks after the onset.

It has been stated that pain follows paralysis as a general rule, and appears more severely in the muscles which are partially, than those which are

completely paralysed, but in only one of these cases is it reported that the pain in the right leg followed the paralysis in that leg.

To cite one case - No. 9 in the synopsis - of a boy aged $7\frac{1}{2}$ years, - the illness commenced with headache and pain down the left leg. Twelve hours later he lost the power of the left lower limb, and though both lower limbs became affected, the paralysis was most marked in the left.

In 6 of the cases there was a history of absence of pain. This seems difficult to explain, except for the fact that the paralysis in these was not very extensive.

Constipation, and less frequently diarrhoea and retention of urine, were noted, principally where the paralysis affected the abdominal muscles. There was no history of the presence of any urticarial rash in any of the cases.

Paralysis.

Paralysis set in at varying times after the first signs of illness, sometimes coincidently, but more often later, and in no case after the 7th day. It is interesting to note here, that where the trunk and several limbs were affected, the paralysis as a rule first appeared in the lower limbs, next in the arms, and lastly in the back.

In one case - No. 6 - that of a boy aged 2 years, the legs became affected 12 hours after onset

of illness, the arms next in 24 hours and the back muscles not till 4 days later.

This would suggest that the virus had a special affinity for the lumbar and cervical enlargements of the spinal cord. But in contra-distinction to this, is the case - No. 16 - of a girl $2\frac{1}{2}$ years of age, whose back, intercostal and abdominal muscles were damaged while the limbs escaped. This was an epidemic case - one other member of the family being attacked.

With regard to the distribution of the ultimate paralysis, great variations were seen. The majority of the cases exhibited a flaccid paralysis of the limbs, with loss of the deep reflexes, and wasting of the muscles which showed the reaction of degeneration.

Examination of the Cerebro Spinal Fluid.

This was carried out in 16 recent cases. The net result showed the fluid to be clear and limpid, with no clot, and no bacteria visible in films, a slightly higher percentage than normal of albumen present, a definite reduction of Fehling's solution, and a considerable increase in the number of white cells - mostly lymphocytes. In other words, the cerebro spinal fluid in Poliomyelitis usually contains an increase in the number of leucocytes, and these are

mainly of the mononuclear type, a higher percentage of albumen and sugar, more constantly than normal.

Clinical Summary.

It is manifest from a survey of these records, that Poliomyelitis shows much resemblance, at the outset, to the other specific infections of childhood; so much so, indeed, that at times it may be a matter of great difficulty to differentiate it before the onset of the characteristic paralysis.

The paralysis which is present at first is almost invariably more generalised than that which exists permanently. In the course of perhaps a week to several months, certain muscles will begin to regain their tone and function, leaving others hopelessly wasted.

Recovery.

There is no means of knowing which muscles will eventually clear up, and which remain paralysed, in the early stages.

"Polioencephalomyelitis."

It is now recognised that a variety of types occur as a result of Poliomyelitic infection, hence the term "Anterior Poliomyelitis" is misleading, inasmuch as it describes the condition as affecting only the anterior

horns of the spinal cord. "Polioencephalomyelitis" is a more inclusive term suggested by Dr. F. E. Batten, and indicates that not only the spinal cord, but any part of the nervous system may be affected.

Types.

Under a variety of symptoms, depending on the part of the nervous system affected, the following types of the disease are included:-

1. The common spinal type with flaccid paralysis of one or more limbs.
2. An ascending form, resembling in many respects Landry's paralysis, and often fatal, owing to the respiratory centres becoming involved.
3. A cerebellar type, characterised by marked ataxia.
4. A cerebral type affecting the frontal, motor, or occipital regions.
5. A bulbar or pontine type in which the basal ganglia or nuclei of the cranial nerves may be hit.
6. A meningeal form simulating cerebro-spinal meningitis.
7. A form in which the peripheral nerves are affected, showing marked pain on movement - so-called "neuritic type."
8. Abortive forms, where the disease runs the usual course, but the paralysis rapidly clears up, and the patient recovers entirely.

FIVE ILLUSTRATIVE CASES.

The following two cases are of interest as illustrations of the bulbar or pontine type of the disease.

Case 22.

The patient was a girl of 3 years. She came from school on the 27th June at midday complaining of toothache and it was noticed that she was feverish and lay about all day. The next day she was not able to swallow the bread of her bread and milk meal, and was still feverish. The following day her speech had a peculiar nasal tone about it. On admission there was found to be some weakness of the masseter muscles, and of the face, neck and intercostals. The palate moved very little and swallowing was very difficult. The C.S.F. was in favour of Poliomyelitis. The patient was discharged 17 days after admission almost completely recovered.

Case 46:

Patient a boy aged $4\frac{1}{2}$ years. For 10 days previous to admission on 30th June he had been vomiting and having headaches on and off, and was very drowsy. On the day before admission he developed severe twitchings of the body, marked internal strabismus of right eye with nystagmus. There was then no pain, no

headache, and no vomiting, and the child was quite conscious. On admission the superficial reflexes were found to be markedly exaggerated - a condition resembling strychnine poisoning. The mental condition was unaffected. The deep reflexes were present. Some slight inco-ordination of the upper limbs existed. There was weakness of the right face, and the right pupil was contracted. The voice and deglutition were affected. Examination of the C.S.F. confirmed the diagnosis of Poliomyelitis. On discharge a month later, there was marked improvement in the conditions present - no strabismus, no nystagmus, and speech and swallowing had almost entirely returned.

Case 60 - Suggesting Intra-uterine Infection.

Patient (girl $4\frac{1}{2}$ years) was a full time child - normal labour and presentation. Child was always very healthy but has never been able to stand or walk. When the child was about 12 months old, the mother noticed that the limbs were wasted, and that the "joints were loose and seemed to drop from the knees". There had been no affection of the arms or sphincters at any time. There was no history of an acute onset. A flaccid paralysis of both legs was present and the limbs were wasted. Knee jerks and ankle jerks were absent. No reaction of the muscles to Faradism. The mother while carrying the



Case 60 - with Poliomyelitis of both p lower limbs.

This case suggested intra-uterine infection.



Case 60 - Seen wearing celloid splints three months
after discharge from hospital.

child had a "miscarriage" at the third month. Some weight extension was necessary to straighten the limbs, and celluloid splints were applied. Patient was seen 3 months after discharge and was walking quite well.

Case 39 - "Jump" Case. - 2nd attack 9 weeks after the 1st.

Boy aged 1 year 7 months was taken ill three days before admission on July 1st with vomiting. He lost the use of his left leg 2 days after onset of sickness. On admission the child was very fretful as if in pain. The legs only were found to be affected, but there was a lack of tone in all the muscles. Passive movements seemed painful. Knee jerks were not obtained. Sensation not affected. Abdominal muscles moved well and equally, the abdominal reflexes were present active and equal. On August 26th he developed a slight bronchial cough. On August 27th he was fretful and cried when disturbed. On examination the abdominal muscles were feeble and the cough was very weak. On August 28th there was marked alteration as compared with previous condition. The legs remained as before. There was great pain on movement on the trunk. The lower abdominal and cremasteric reflexes were not obtained, the lower recti were flaccid and the abdomen was distended. The upper abdominal reflexes were equal and active. There was

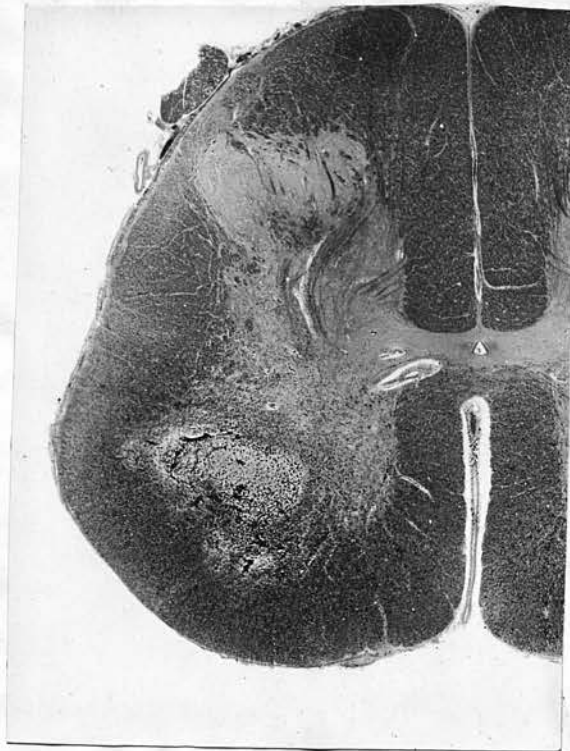
tugging of the navel upwards when he attempted to sit up. Lower intercostals were feeble. On October 17th he developed diphtheria and was sent away. He had improved considerably.

Case 42 - Death from Broncho-Pneumonia, following
Measles and Poliomyelitis.

Boy aged 2 years, took ill 5 weeks previous to admission on October 17th with a cough, pain in the chest and running at the nose. He was kept in bed for two weeks and then was noticed to have lost power in both legs. On examination there was flaccid paralysis of both lower limbs, with loss of knee jerks. Three weeks after admission he developed measles, and died a week later of broncho-pneumonia.

Autopsy: Examination of the brain was refused. The spinal cord on section in the dorso lumbar region showed two small areas of degeneration, one in each anterior horn, and two less marked smaller areas in the posterior horns.

The microscopical sections of the spinal cord in this case are shown and described on the accompanying page:-



Section of lumbar region of spinal cord stained by the Marchi method, showing area of softening in the region of the anterior horn, and degeneration of the antero-lateral tract on the same side. The posterior horn and the posterior columns appear perfectly normal.



Section of lumbar region of the spinal cord of a child aged 2yrs who died 9 weeks after the onset of Poliomyelitis.

Section stained by the Weigert Pal method, shows an area of softening in both anterior horns, as evidenced by the absence of medulated fibres. The whole of the antero-lateral tracts are poorly stained, indicating a destruction of these fibres -in-these in these tracts.

TREATMENT OF THE ACUTE STAGES OF THE DISEASE.

During the very early stages of the disease, pain is the symptom which requires most attention, and it is this symptom which may lead to a mistaken diagnosis of Acute Rheumatism. The essential thing to do here is to put the patient at complete rest, on a water bed. The limbs are wrapped in cotton wool, and placed in any position that is most comfortable for the patient, and carefully supported in that position. Aspirin, or Salicin, given in doses of 3 to 5 grains, every four hours, will generally be found to give relief, but in some cases, morphia will be necessary. Great caution, however, must be observed in giving this drug, in cases where the respiratory muscles are affected, and it is well to combine it with Atropine, in these cases.

Urotropine should be given in 5 to 10 grain doses, every four hours, on account of the fact that the drug is excreted into the cerebro spinal fluid, and has been shown experimentally to prevent the onset of the disease in animals. Children may be given the above dose with safety, if it be well diluted with water.

When the muscles of deglutition, and respiration are involved, the child must be fed with great care, and, if it be thought that the glottis does not close on swallowing, resort should be had to nasal feeding.

It is advisable, in the acute stages, to perform Lumbar puncture, both to establish the diagnosis, and to relieve pain.

After the acutest stage of the disease is past, which usually lasts from five to ten days, it is important to put the paralysed limbs in a position of rest, so as to prevent contractions, and deformities from taking place. This is most effectively done by placing the limb in the celluloid splint, as carefully moulded to the leg, and worn both by day and night.

The use of celluloid splints has been in vogue for some time in the treatment of tubercular diseases of bones, and joints, but Dr. F. E. Batten is responsible for applying and developing their use in Polio-myelitis, with the result that we now have the means of relieving in a large measure the distressing effects produced by this disease.

I do not propose to detail the processes involved in making these splints, as this has been already described by Dr. Batten in the "Lancet" 1912, Vol. II, page 80. It will suffice to say that there are three stages in their manufacture:

1. that of taking the impression of the limb - the negative;
2. that of casting the positive from the negative, and
3. that of moulding the splint itself on the positive.

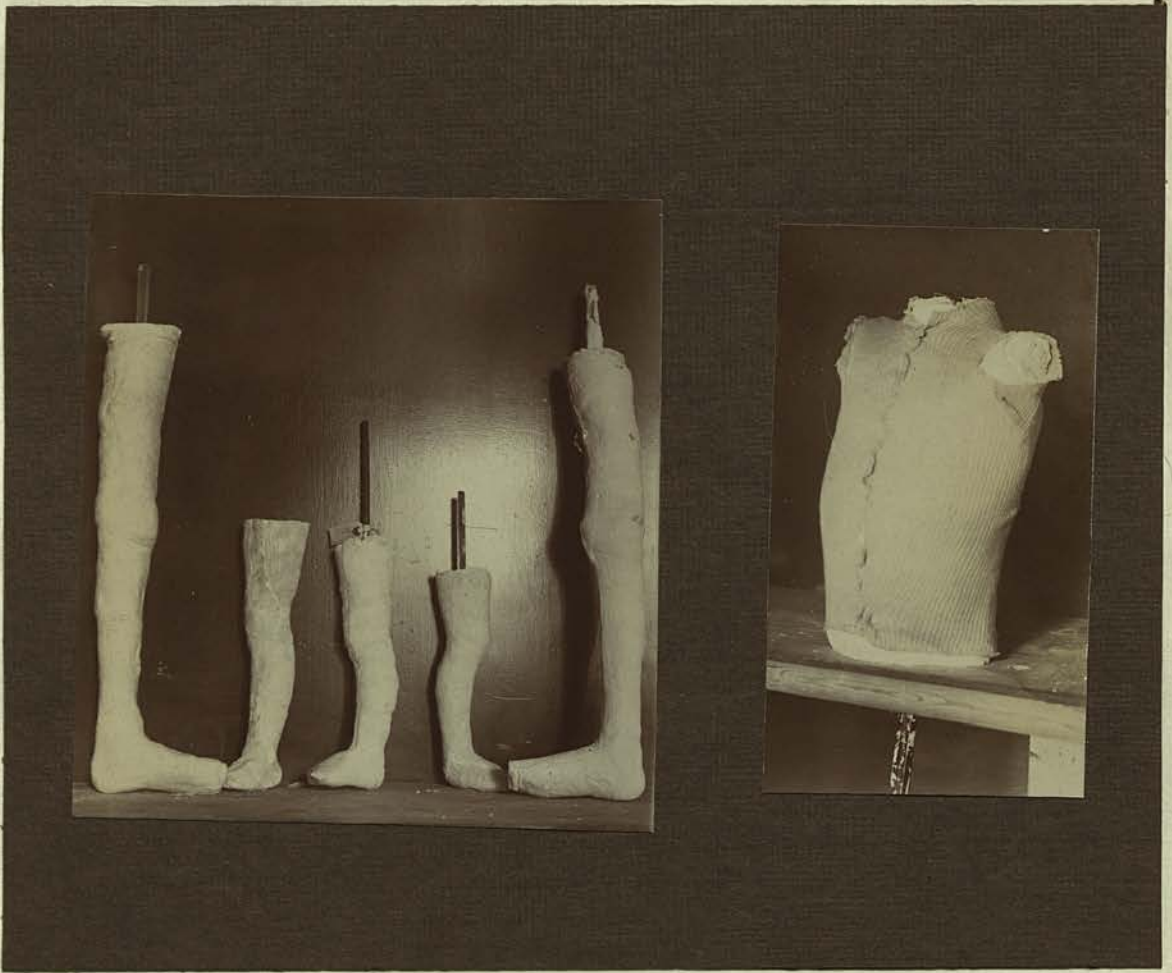


1. 2. 3.

Three stages in the manufacture of the splint.

1. The negative.
2. The positive.
3. The finished splint.

The splints are made in the wards of the Hospital for Sick Children, Great Ormond Street, and with a little practice, the necessary skill is attained. It is of the greatest importance to place and maintain the limb in a good position, while the plaster impression is being taken in the first stage. This position is one in which the knee is slightly flexed, and the foot extended just beyond the right angle; the objects of the splint being, firstly, to maintain the affected muscles in a position of rest, secondly to enable the child to walk, and thirdly, to prevent any deformities from setting in. It is essential that the muscles should be relaxed so that the natural processes of repair may go on, and this position of the limb will be seen to adequately meet this necessity. At the same time it is found that when the child comes to walk, this amount of flexion of the knee, and extension of the foot, makes progress in the rigid splints most possible. Where any deformity is present, from the long continued over-action of the sound muscles - and deformity quickly takes place - it is essential to have it corrected before casts of the limbs are taken. Manipulation, hot baths, weight extension, and the use of various temporary appliances may suffice for this, in a short time, but in long standing cases, where firm contractures exist, it may be necessary



Positive casts of limbs and trunk.

to resort to tenotomy, before the desired position is attained.

It will be understood that, with these objects in view, the earlier after the onset of paralysis the splints are applied, the better. The only obstacle to this is the persistence of pain, as already mentioned, in some cases.

It is in those cases where the paralysis has affected principally the lower limbs that celluloid splints are especially suitable, and demonstrate their many and decided advantages over other forms of splints used for the same purpose.

When the paralysis is not very extensive, as a general rule, the results, in regard to power in walking, are most satisfactory. The cases in which the greatest difficulty is experienced in obtaining this desired result are those in which the psoas and iliacus, glutei, and lumbar muscles are involved in the paralysis. But even here, by fitting the patient with a celluloid or canvas jacket, as necessity arises, and connecting this with stout wire springs, or elastic bands, to the leg splints, it is possible to considerably improve the power in this direction. To correct any tendency to inversion or eversion on the part of the limb as a whole, a similar device may be employed.



Front and back views of finished leg splints.



Finished celluloid splints for power limbs & trunk.

For the arms, the splints are used with equally good results. Here the splint may be made in two sections - one for the upper arm and one for the fore-arm - and connected by two jointed plates of iron let into the celluloid material. Flexion at the elbow is aided by the use of the stout wire springs, already mentioned, and attached to each section of the splint by rivets.

The after-results of treatment by the use of celluloid splints are, in the large majority of cases, highly satisfactory. In a few cases the paralysis is too extensive to hope for any improvement.

Massage should be commenced as soon as the pain in the affected muscles has disappeared, and should be continued twice a day. The object aimed at is to endeavour to restore tone to the affected groups of muscles. Movements against resistance are to be recommended.

Electricity may be employed in the form of galvanism to affected muscles, or sinusoidal baths into which the patients are placed for fifteen minutes daily, but little use is derived from such treatment.



Boy in splints with walking machine.



Boy, aged three, in splints, standing in walking machine.

C O N C L U S I O N S .

Investigations involved in the writing of this thesis have led me to the following conclusions in regard to this disease:

1. That Poliomyelitis is an acute specific disease, not a primary degeneration of nerve cells, as was formerly believed:
2. That although the specific organism has not as yet been demonstrated, a virus has been isolated from infected individuals, and the disease produced in animals by inoculation therewith. That cultivation has recently been successful, and, no doubt, that the morphological characters of the virus will shortly be known. Further, that it has been found possible to detect a specific reaction in the serum of infected people:
3. That the organism gains access probably through the nasal mucous membrane; that it spreads to the central nervous system directly along nerve tissues by contiguity, and finally, that it leaves the body by the same route:
4. That the incubation period is variable, depending upon the virulence of the infection, and on the powers of resistance of the subject:
5. That the following anatomical changes are

met with:-

- (a) a mono-nuclear cell infiltration of the vessels of the pia - arachnoid membrane;
 - (b) degeneration of nerve cells.
6. A careful study of all the reported epidemics of recent years indicated that the incidence of the disease is most marked in the months of July, August and September, and
- That the following factors are chiefly concerned in its spread: human contacts, general sanitation, flies, possibly domestic animals, and more especially dust. I believe that the latter is a very important factor.
7. That clinically this disease has much wider limits than were formerly recognised, and
- That "Polioencephalomyelitis" is a more inclusive and accurate term, since a variety may be manifested clinically, apart from, and in addition to the common type of paralysis.
8. That examination of the cerebro-spinal fluid shows:-
- (a) Slight increase of albumen
 - (b) More active reduction of Fehling
 - (c) Increase of lymphocytes.
9. That in the treatment of the acute stages of the diseases, rest must be the object aimed at, for when the affected muscles are put into the position

of muscular relaxation, and kept there, no deformities should arise:

That an appliance which serves the double purpose of keeping the parts at rest, and, at the same time enabling the patient to walk, is much to be desired. Such an appliance we now have in celluloid splints.

That massage and movements against resistance should be commenced in the early stages of the disease, and be persevered with.

In the light of these conclusions it will be agreed that Poliomyelitis bears a strong likeness to the acute specific fevers of childhood, and it is to be hoped that an earlier recognition of this fact, by the general public, as well as by the medical profession, will do much to control the spread of infection. It is further to be hoped, and indeed anticipated, that as a result of investigations now proceeding, a prophylactic treatment for Poliomyelitis will come into practical use.

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S Y N O P S I S O F C A S E S

admitted to

HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET,
in 1912.

No.	Age	Sex	Duration of Illness.	District	Symptoms	Muscles affected	Onset of Paralysis	Treatment	Result	Remarks
1	7 $\frac{9}{12}$	M	10 days	E. Dulwich.	Headache Pain in L. leg. Loss of power L. leg (12 hours later)	Both legs L. & R. Intercostals. Abdoms.	12 hours	Massage. Celluloid Splint for L. Leg.	In 3 months could walk without assistance	C.S.F. clear - Albumen .02, Lymphocytes. Pain lasted for 19 days. Sporadic case.
2	4	M	3 $\frac{1}{2}$ mos.	W. Ealing	Pain & stiff neck. Paralysis L. leg (6 hrs. later) Paralysis R. Arm (14 days later)	L. leg & R. upper arm and shoulder	6 hrs. 14 days	Massage. Celluloid Splint L. leg.	Able to walk with support in 2 months.	Brother affected 12 days before. No. 2 slept a night with him when ill.
3	10 $\frac{9}{12}$	M	3 $\frac{1}{2}$ yrs.	Millwall	Pain	Both lower limbs. Dorsal muscles.		None	I.S.Q.	Scoliosis - unable to stand.

No.	Age	Sex	Duration of illness.	District	Symptoms	Muscles affected	Onset of Paralysis	Treatment	Result	Remarks
4	7 yrs.	M	7 months	Hants.	Headache Vomiting No pain	L. Arm	3 days later	Sent to Block with K.L.B.	I.S.Q.	Sporadic.
5	7	F	1 month	Hants.	Pain in neck. Sickness	Both upper arms " thighs Back Abdomen	24 days	Massage.	I.S.Q.	C.S.F. - Clear - Alb. plus Lymphocytes. Incontinence & Malaena for a time.
6	2	M	2½ mos.	Amersham	Pain & Temp. Vomiting	Legs R. Arm Neck Back Speech	12 hrs 24 " 4 days	Massage - weight extension. Tenotomy, Splints for legs.	Able to walk in 5½ months.	Commenced to improve in 17 days.
7	7	M	9 months	Wealdstone	Temp. - no pain	Both legs R. & L.	7 days	Correcting back splint. Celluloid splints for legs.	Able to walk with support in 2 months.	Commenced to improve in 1 month. Diagnosed as T.B. Meningitis.
8	3½	F	1 year & 10 months	Glasgow	Pain	Back. Abdom. Legs Arms		Back Splints, Extension Plasters, Tenotomy. Cell. splints for legs & Cell. jacket.	I.S.Q.	Too extensive to hope for improvement.

III.

No.	Age	Sex	Duration	District	Symptoms	Muscles affected	Onset of Paralysis	Treatment	Result	Remarks
9	3	F	1 day	W.C.	Diarr. Vomiting. Temp. No pain.	Right Arm	2 days	U.D. sent home.		Faradism & Galvanism already diminished. C.S.F. - clear - Lymphocytes.
10	1½	M	2 mos.	Holloway	Nil	Both legs	?	Splints Cell.	Improved	Paralysis was first thing noticed.
11	8/12	F	7 wks.	S.Ealing	Nil	Arms, thorax, legs.	?	Nil	I.S.Q.	Paralysis was first thing noticed. C.S.F. clear - Lymphocytes.
12	4	M	? 12 days	Wealdstone	Bilious attack - 12 days ago Pain (head & back) Vomited - 8 days ago	Trunk & legs.	3 days	Urotropine Gr. V. 4 hourly.	Very much improved	C.S.F. Clear Lymphocytes.
13	7	M	15 mos.	Ashley, Leices- tershire	Sick head- ache. Pain.	Both legs Speech	6 hrs.	Nil	I.S.Q.	Epidemic case - Pain subsided in 14 days. K.J. + + Plantars extensor. Good Recovery. L. leg only affected now - no loss of power but deformity.

No.	Age	Sex	Duration	District	Symptoms	Muscles affected	Onset of Paralysis	Treatment	Result	Remarks.
14	3½	F	10 weeks	Forest Gate.	Headache	Thoracic muscles & limbs	2 days	Celluloid Jacket.	Helped.	Could walk before admission. Reflexes not affected.
15	2	M	15 days	Bognor	Nil.	Both legs L. & R.	?	Splints.	Improved	Can stand without splints.
16	2½	F	8 months	Huntingdon.	Vomiting Temp. Delirium	Back Intercostals Abdoms.	1 day	Galvanism, Massage, Jacket.	Improved	Limbs not affected, - paralysis immediately. Epidemic - one other in family. Can stand alone. Diaphragm not affected.
17	2½ 12	F	6 days	S. Lambeth	Fever Constipation.	L. Arm	4 days	Massage Galvanism	I.S.Q.	C.S.F. Clear Lymphos.
18	1 8 12	M	10 days	Regents Park	? Pain in head for 3 days. Nothing else	No definite paralysis. Weakness of Left thigh. L.K.J. absent.	24 days	No splint. Massage & movements.	Much improved	C.S.F. Clear. Lymphocytes. Unusual case - legs rigid. One case in same street developed 10 days later.

No.	Age	Sex	Duration	District	Symptoms	Muscles affected	Onset of Paralysis	Treatment	Result	Remarks.
19	3½	F	25 days	E. Finchley	Pain in legs for 2 days	Both lower limbs	1 day	Celluloid Splints.	Improved. Can walk with help.	
20	2	M	3½ mos.	Kent.	Pain Vomiting Fever.	R. Arm & R. Leg. & Flexors R. Hip	7 days	No splints - needs stronger iron one for outward rotation.	I.S.Q.	
21	3	M	9 days	Streat-ham	Drowsy Vomiting Fever. No pain.	L. leg.	? 2 days	Urotropine gr. X tds. Splint for L. leg.	Walks about improved.	C.S.F. clear lymphocytes.
22	3	F	3 days	London N.W.	Toothache Fever & inability to swallow Speech affected	Bulbar 5th 7th & Bulb.	? hours	Nil	Almost complete recovery.	C.S.F. clear lymphocytes.
23	3	M	2 years	South Acton	Pain for 2 weeks Fever Vomiting	Both arms & legs & mouth.	2 days	Celluloid splint for R. leg.	Improved.	
24	2	F	3 mos.	Bucks	No constitutional	Both legs -	?	Massage - Cell, splints	Improved.	

No.	Age	Sex	Duration	District	Symptoms	Muscles affected	Onset of Paralysis	Treatment	Result	Remarks.
25	6	F	4 years	West Ham	No constitutional	Left leg	1 day	Splint	Improved.	
26	2	M	8 days	Euston Rd.	Drowsy, nil else.	R. Arm, leg & face		Nil	Recovery	K.J's absent. C.S.F. clear, lymphocytes.
27	7	M	13 days	Swiss Cottage, N.W.	Drowsy Headache Constipation No pain.	R. Face Platysma R. Shoulder.		Nil	Improved	
28	9	F.	1 year	Burton-on-Trent	Headache Pain Stiff neck Constipation.	Arms & legs	48 hours	Tenotomy	Improved	Incubation ? 7 days - Sensory loss Bladder signs. 7 or 8 cases in district.
29	1 $\frac{9}{12}$	F	8 days	Hornsey	Pain, malaise	R. leg Back	3 days	Celluloid Splint.	Improved	
30	2 $\frac{4}{12}$	M		W.Green	Nil	L. leg		Massage	Improved	
31	4	F	4 days	Maida Vale	Feverish pain	Both legs.	6 hrs.	Massage Electric baths.	Improved	Two contacts died

VII.

No.	Age	Sex	Duration	District	Symptoms	Muscles affected	Onset of Paralysis	Treatment	Result	Remarks
32	3½	M	2 years	?	Cold Pneumonia Pain Stiff neck	Both legs	2 days	Massage Splints	Improved	
33	9	F	11 days	Bucks	Abdom. pain General pain Vomiting Constipation.	Both legs Back	12 hrs	Splints	Much improved: walking	C.S.F. Clear - Lymphocytes.
34	3	M	3 wks.	Dartford	Pain & stiff legs (nil else)	L. Leg		Massage	Much improved	C.S.F. lymphocytes clear.
35	4½	F	6 wks	Essex	Pain in R. foot, wrists & legs	R. leg		?	Improved	C.S.F. clear lymphocytes. Onset like acute rheumatism. Fever, pain & swelling of joints.
36	3½	M	3 wks.	Streat- ham.	Vomited, Pain	L. leg		Massage Sinusoidal baths.	Improved	
37	11½	M	6 mos.		Unconscious epistaxis	L. leg R. arm back & chest	2 hrs.	Massage - Galvanism	Improved	

No.	Age	Sex	Duration	District	Symptoms	Muscles affected	Onset of Paralysis	Treatment	Result	Remarks
38	2½	M	6 mos.	Berkhamsted.	Bronchitis, pain.	R. Leg	?	Massage & Galvanism	Improved	
39	1 $\frac{7}{12}$	M	3 days	?	Fever, Vomiting, No pain.	L. leg	hours	Massage & Galvanism	Improved	Supposed 2nd attack 8 weeks after admission. C.S.F. Clear lymphocytes.
40	1 yr	M	3 wks	Hounslow	Fever Pain	R. foot	1 wk.	Massage & Galvanism	Improved	
41	5	F	1 yr	Letton	Nil	L. leg		Massage	Improved	
42	2	M	5 wks	Kings X	Pain, Cough, Bronch.	Both legs	?			Measles & Bron. Pneu. Death. P.M.
43	5 yrs	M	12 days	Teddington.	Pain 1 wk. in L. thigh.	L. leg	1 wk.	Nasal douche formalin, massage.	Improved	
44	3½	F	3 yrs.	S.E.	Pain Bronchitis	Both legs Spine	Wks	Thomas's Hip Splint	Improved.	

No.	Age	Sex	Duration	District	Symptoms	Muscles affected	Onset of Paralysis	Treatment	Result	Remarks
45	5	M	2½ yrs.	N. Kensington	Pain after paralysis	R. leg	?	Massage	I.S.Q.	
46	4½	M	10 days	Holloway	Vomiting, Headache Drowsy	R. Face: Squint. Nystagmus	7 days		Recovery	Exaggerated superficial reflexes, like strychnine poisoning. Mental condition not affected. Slight inco-ordination of upper limbs. K.J.'s present. Rt. pupil smaller. Voice & deglutition affected. C.S.F. Clear lymphocytes and polys.
47	1 $\frac{9}{12}$	M	1 yr.	Suffolk	Fit	Complete deglutition R. leg	1 hr.	Massage	I.S.Q.	
48	4	M	1 yr	Kent.	Fever for a week	R. leg	7 days	Massage & electricity.	I.S.Q.	Mentally deficient.

No.	Age	Sex	Duration	District	Symptoms	Muscles affected	Onset of Paralysis	Treatment	Result	Remarks.
49	7	F	5 wks	Isleworth	Pain in legs, Back & Head.	L. leg	hrs	Massage & electricity	I.S.Q.	Simulating Rheum: Several cases in neighbourhood.
50	3	F	6 days	Bexley Heath	Cold, fever headache; fits of twitchings of eyes. No vomiting. Abdom. pain Pain L. leg 2 days ago	R. leg			Recovery in 3 wks	One other attack in adjacent house.
51	6½	M	1 year	Ealing	Fall, meningitis.	R. face	7 days	Electricity and splint	Improved	Brother affected at same time R. leg.
52	9/12	M	6 weeks	Folkstone.	None	All limbs	?	Massage	Recovered.	Spastic type. Reflexes increased.
53	6	F	2 yrs.	York	Meningitis with aphasia	Both legs Back		Jacket & splints.	Improved	Very severe case.
54	2	M	15 days	Southall Green	Acute illness Vomiting No pain	Both legs back & abdom.	24 hrs	Jacket & splints	Improved	C.S.F. Clear alb. 1% Reduction of Fehling - Lymphocytes.

No.	Age	Sex	Duration	District	Symptoms	Muscles affected	Onset of Paralysis	Treatment	Result	Remarks
55	1 $\frac{9}{12}$	M	6 months	Holloway	Sudden onset of paralysis	Both legs arms back & neck.		Splints	Improved	
56	2 $\frac{1}{12}$	M	4 months	Barnet	Sudden fever. Pain.	L. leg		Splint	Improved	
57	4 $\frac{4}{12}$	F	2 years	Gilling-ham.	Stomatitis Unconscious for a week.	General Both legs	7 days	Splints	Great improvement	
58	5 $\frac{1}{2}$	F	4 mos.	Camberwell	Frontal Headache: Bladder symptoms.	Both legs		Splints	Improved	
59	15 $\frac{5}{52}$	F	1 month	Willesden	Sudden onset at the breast	Very extensive General		Nil	I.S.Q.	
60	4 $\frac{7}{12}$	F	? From birth	Brixton	Nil	Both legs		Splints	Improved greatly.	? intra-uterine.