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RECENT EPIDEMIC OUTBREAKS OF ACUTE POLIOMYELITIS

THERE is, I fancy, always much difficulty in making a selection of a subject on which to address you on the opening night of the session by your President for the year; I know I have felt this difficulty. By usual custom the address from the chair must not be criticised, and the President has the right, for one night, of expressing any views he may think proper without fear of being shown to be altogether at fault by subsequent speakers.

Keeping this in mind I have decided to avoid all controversial subjects and to speak on some of the recorded facts, and generally accepted views, regarding Recent Epidemics of Acute Poliomyelitis, the most recent addition to the list of notifiable diseases.

This disease is known by many names, a fact which in itself goes to show that none yet suggested has been found altogether satisfactory. The authorities in these islands have decided that, for the purposes of notification, it shall be known as "Acute Poliomyelitis," which is at least not more unsatisfactory than others. In America it is usually referred to as "Epidemic Poliomyelitis," or "Epidemic Infantile Paralysis,"in some parts of the continent the term "Heine-Medin's Disease" is preferred, while some writers on the subject endeavour to avoid the main objections to any of these names by referring to it as Meningo-Myelo-Encephalitis Disseminata, which is at once condemned as too cumbersome for general use.

With us the disease is still perhaps most commonly called Infantile Paralysis, although it has long been recognised that many of those affected have long passed the stage of infancy, and that in many instances there is not any paralysis.

It seems to be clearly established that Acute Poliomyelitis has only in comparatively recent years appeared in epidemic form. The first definite and clear reference in the literature to an epidemic outbreak occurs in 1881, when Bergenholtz recorded the occurrence of eighteen cases in one district in Northern Sweden. Descriptions of sporadic cases of the disease are to be found in medical writings for at least a century before that time. After an exhaustive search into the literature, Professor Römer of Marburg comes to the conclusion that "the absence of reports of epidemics can be explained only by the fact that such epidemics did not occur before 1880."

It was not until the appearance of Medin's well-known record of forty-three cases in Stockholm in 1887 that attention was called to the subject. Since that time outbreaks have been more frequent and ever increasing in extent. In 1899 Wickman reported fifty-four cases in Stockholm, and in the same year Leegaard described an outbreak, also of fifty-four cases, which had occurred at Byatsburg in Norway, between July and October. Wickman's excellent record of his observations in over 1,100 cases in Sweden in 1905-6; his accurate description of the various clinical types met with in that epidemic, and of the post-mortem findings in the fatal cases, mark a great advance in our knowledge of the disease. In the same years – 1905-6 – Norway also suffered severely, about 1,000 cases being reported.

In Germany small outbreaks had been reported from time to time since 1886, the first large outbreak there occurred in Westphalia in 1909; 700 cases being reported, and there were at least 1,000 cases in Germany in that year.

About the same time outbreaks were reported from Austria, Holland, Switzerland, Russia and Australia.

In France reports of epidemic outbreaks began to appear in 1888 and gradually became more numerous. Netter reported 100 cases in Paris in 1909.

Italy had much the same experience, and small outbreaks also occurred in Spain. America came in with a record of thirty-eight cases in Massachusetts in 1892; since that time America has had annual visitations of ever increasing magnitude. In this, as in other things, she soon "licked creation," and in the Forty-First Annual Report of the State Board of Health for Massachusetts it is claimed that five-sevenths of all the cases of epidemic poliomyelitis reported from all over the world, up to that time, had occurred in America.

Epidemics increased in number and extent until 1910, when, all over Europe and in the United States and Canada, the disease was very prevalent.

According to Flexner America had between the years 1907 and 1910 23,000 reported cases, and it is estimated that at least 20 per cent. of the cases were not reported.

In the British Isles the first report of an epidemic outbreak was made in 1897 by Dr. Pasteur who met with seven cases in one family. Reports of small outbreaks then began to appear more frequently. The first large outbreak was reported by Parker of Bristol in 1909 – there were thirty-seven cases. In 1910 thirty-four cases were reported in Carlisle, thirty-seven in Barrow, and it is known that quite a large number of cases occurred in the towns and rural districts in the North of England. In that same year – 1910 – quite an extensive epidemic occurred in Leicestershire and Nottingham; Bingham and Melton Mowbray districts being most affected. The facts regarding this latter outbreak, which ran to seventy-four reported cases, were carefully enquired into by Dr. Farrar of the English Local Government Board, who presented a most interesting and detailed report on the cases. In this outbreak, as has so often been the case elsewhere – both in Europe and America – the true nature of the epidemic was not at first recognised; for a considerable time the cases were diagnosed as cerebrospinal fever, and it will be remembered that at that time our local papers here published almost daily reports of the progress of "the epidemic of spotted fever" in Melton Mowbray and Nottingham. This mistake was the more easily accounted for by the fact that the meningitic type was common, and that there occurred at the same time some cases of true meningococcal meningitis in the city of Nottingham.

In the following year – 1911 – there were also many cases in England, and quite an extensive epidemic was reported from Devon and Cornwall. The exact number of cases in this outbreak is not definitely known, but it was certainly over 200. This epidemic was very severe and the mortality in it high. A very valuable report on this epidemic in Devon and Cornwall was presented to the Local Government Board by Dr. Reece. This report dealt with 224 cases. In this outbreak also the cases were at first reported as cerebro-spinal meningitis.

In Scotland the records are few but outbreaks have occurred; Low reported sixty-two cases in, and near, Edinburgh in 1910.

In Ireland, at least since 1909, cases have been more common and many small outbreaks have been observed. In the autumn of 1911 there were many cases in Belfast and the neighbouring counties. It was again very prevalent in our own district in 1913. Severe localized outbreaks have occurred in several districts – mainly rural districts – throughout the country. The best known of these occurred at Coagh, Co. Tyrone, mainly in the practice of Dr. Burgess, commencing in October, 1913. Dr. Burgess had within a few weeks thirty-seven cases in a comparatively sparsely populated country district. A few months later Dr. Burgess had a single house outbreak, in this house there were six cases of very virulent type with four deaths. During the progress of this outbreak I had an opportunity of visiting the district with the late Dr. Brian O'Brien and Dr. Burgess, and of examining some seventeen or eighteen of these patients, then in all stages of the disease. Dr. Burgess's experience has been very exceptional and I hope he will let us have a record of this epidemic.

At the same time as the Coagh outbreak some fourteen cases occurred in quick succession at Irvinestown.

It is not possible to obtain anything like complete figures for Ireland. I am indebted to Dr. Bigger of the Local Government Board for a return of the cases reported to the Board since 1912. From that return it does not appear that, with the exception of these two instances, there has been any considerable localized outbreak.

I think it may be truly said that in almost every place where there has occurred for the first time a considerable outbreak of Acute Poliomyelitis the true nature of the disease has not been immediately recognised, and in many instances the earlier cases have been diagnosed and reported as cerebro-spinal meningitis. This occurred in the earlier outbreaks in Scandinavia and in the United States, and also, as I have already pointed out, in both the large epidemics in England. We are all quite familiar, and have been since our college days, with the clinical characteristics of the ordinary sporadic case of Infantile Paralysis, but with the very varied clinical types which appear to have been invariably met with in all these later epidemic visitations we are not familiar, and mistakes in diagnosis are likely to occur.

Seasonal Prevalence. – Practically all these outbreaks to which I have referred have occurred in the late summer and autumn. Some variation is shown in the times at which the period of maximum intensity has been reached. In many instances it has been in August, at other times as late as October. In some places cases have continued to crop up all throughout the winter months.

Symptomatology. – In all these epidemics the clinical types met with have been similar. Wickman gives a most exhaustive and masterly description of

the very varied clinical pictures presented by the cases in the epidemics in Sweden. He classifies the cases into eight divisions and his classification has been generally accepted.

Wickman's main types are –

I. The *Abortive* type in which the prodromal symptoms are present, but which clears up quickly without any paralysis.

II. The *Spinal* type. In this group is included a large percentage of the cases. It is characterised by the usual onset followed by paralysis, temporary or permanent, of the limbs or muscles of the trunk.

III. The *Ascending* or *Descending* type, fortunately a small group in which the paralysis generally begins in the lower limbs, less frequently in the arms, and gradually extends and involves the muscles of respiration, terminating in death after a few days' illness.

IV. The *Bulbar* or *Pontine* type in which some of the cranial nerves are affected frequently in conjunction with some involvement of the spinal centres.

V. The *Cerebral* type in which changes occur in the cortex of the brain, giving rise to spastic hemiplegia or monoplegia.

VI. The *Meningitic* type in which the symptoms closely resemble those of cerebro-spinal fever, the pia mater being much involved. Netter found that 29 per cent, of his cases were of this variety.

Wickman also describes (*a*) an Ataxic type, but it seems doubtful if this variety should not be classed with the "Bulbar and Pontine" cases; and (*b*) a *Polyneuritic* type which many writers think should be included in the "Spinal" class.

The incubation period is said to be generally five to ten days, but many observers believe they have met with instances of much longer periods, and Wickman reports one case in which he was satisfied it could not have been longer than one day.

Whatever the type the onset is always much the same, varying only in severity. An initial rise of temperature, generally digestive disturbances with vomiting and occasionally diarrhoea, drowsiness, profuse sweating, irritability, marked hyperaesthesia, with pain and general tenderness.

Wickman lays stress on the diagnostic value of (1) drowsiness, (2) pain and tenderness, (3) stiffness of the neck, and (4) profuse sweating. According to Müller the outstanding signs are (1) the profuse sweating, (2) the hyperaesthesia, and (3) leucopenia.

In the cases which occurred in our own neighbourhood in the last few years all Wickman's types were met with and I have seen examples of them all.

The Spinal type was common, several showed involvement of the cranial nerves.

In 1909 I saw with Dr. Gaussen two cases in one family at Lambeg, children of ten and twelve years; both had taken ill on the same day. When I saw them on the morning of the fourth day of illness one showed complete hemiplegia, was unconscious, with rapid breathing and a temperature of 104° with drenching sweating. This child died a day or two later; this was a typical example of the cerebral type. The other had a comparatively mild attack of the *Spinal* type with partial paralysis of both legs.

In the autumn of 1913 there were many cases in the city. At that time I saw with Dr. M'Lorinan four cases in one family, all had taken ill within a couple of days. When I first saw them two had comparatively mild attacks of *Spinal* type one with partial paralysis in both legs, the other with partial paralysis in one arm. Another was still very acute, showed profuse sweating, with high temperature and very great tenderness all over, especially in the legs. The dread of being touched was so great that the child when anyone approached the bed held itself quite rigid and gripped the mattress with a hand on each side; the sweating was so free as to drench the pillow and bed. There was no mental confusion.

We enquired from the mother if the other members of family were quite well; she told us that the youngest child, an infant of ten months, seemed dull but not really ill. On examining this infant we found it had a temperature of 101°, was rather apathetic and had complete absence of knee jerks. This child did not develop any further signs. The temperature next morning was normal, but the absence of the patellar reflex remained for some time. This I take to be a typical example of the *Abortive* type. At this time when there were many cases of poliomyelitis occurring in the city, I saw two cases of "Landry's paralysis" within a fortnight. One was a powerfully-built man of twenty-one years who had taken ill with headache, general tenderness and mild fever with some vomiting. The vomiting and headache cleared off but the temperature remained elevated without obvious cause. I saw him with Dr. Martin on the fifth day of his illness, he had that morning shown some weakness in his legs and had complete loss of patellar reflex. He was removed to Purdysburn Hospital. Next day there was complete paralysis of the legs; the paralysis quickly extended to the arms and later to the respiratory muscles, and he died from the respiratory paralysis on the ninth day. The other was a case of Dr. Burnside's, a boy of fifteen years. In this

case the paralysis had extended much more slowly. When I saw him the legs were completely paralysed and the arms almost so. He was unable to swallow and there was great difficulty in respiration. He died the following day.

Of the *Ataxic* type I have only met with one case. This case I saw with Dr. Rentoul in Lisburn. Two children in one family had been taken ill on the same day. One developed a very severe attack of the combined meningitic and spinal type with complete paralysis of both legs, which was permanent. The other, a boy of seven years, after a short feverish attack with gastric disturbance lasting a couple of days, developed a very ataxic gait. When I saw him the fever had cleared up. In bed he appeared quite well and very bright. He showed complete loss of knee jerks, and when stood upon the floor he was able to walk without assistance but with a *very* staggering gait. There was then no pain or tenderness. He was most anxious to give exhibitions of this staggering which he created as a great joke. He was kept at rest in bed and the ataxia rapidly disappeared. The knee jerks remained absent for a considerable time.

The position of "Landry's paralysis" in relation to acute poliomyelitis has given rise to much writing. Strümpell had insisted from clinical observation that they were identical. Wickman satisfied himself from histological findings that this view is correct. Römer states that "Landry's paralysis is only infantile paralysis with a fatal issue." Zimmerman as early as 1885 from post-mortem evidences came to the conclusion that "Landry's paralysis and infantile paralysis formed only different degrees of one and the same disease."

Römer refers to Landry's paralysis as "poliomyelitis acutissma." According to him it was the custom until a few years ago to allow the diagnosis in many cases (which we now know to be poliomyelitis) to depend upon the ultimate result, viz.: If the case recovered with temporary or permanent paralysis the diagnosis was "poliomyelitis," if having shown all the initial symptoms the case cleared up without any evidence of paralysis the diagnosis was "polyneuritis;" while if the paralysis extended and involved the respiratory muscles and the heart and the case ended fatally the diagnosis was "Landry's paralysis."

Second Attacks. – Second attacks have been reported by many observers but appear to be very rare. They have also been experimentally produced in the monkey.

The Case Mortality. – The case mortality in these epidemics seems to have varied within wide limits. In examining the records it is impossible to avoid the conclusion that the figures are not always closely comparable.

Author	Place	Year	Cases	% Mort
Wickman	Sweden	1905	868	16.7
Leegaard	Norway	1905	577	14.5
Krause	Westphalia	1909	633	12.3
Müller	Hesse-Nassau	1909	100	16.0
Frost	Cincinnati	1911	150	30.7
Frost	New York	1912	1,108	16.5
England.				
Reece	Cornwall & Devon	1911	154	22.1
Farrar	The Midlands	1910	74	10.8
Ireland.				
Burgess	Coagh (Tyrone)	1913-14	37	27.0

In some outbreaks the abortive cases appear to have been carefully looked for, in others they seem to have been generally overlooked; and when it is remembered that so accurate an observer as Wickman found that the abortive cases in some outbreaks accounted for over fifty per cent. of the total cases it will be apparent how their inclusion or exclusion must modify the statistics. In most of the mortality tables the abortive cases are excluded, only those in which there was definite evidence of paralysis being counted. Then the case mortality has generally been between ten per cent, and thirty per cent., though in some outbreaks it appears to have been considerably higher.

The Causative Agent. – In 1909 Landsteiner succeeded in producing acute poliomyelitis in the monkey by injecting into the peritoneum an emulsion of the spinal cord of a fatal case in a child. He further attempted to infect other monkeys by injecting emulsions of the cord from this monkey into the peritoneum, but with negative results.

Landsteiner's experiment opened up a new field of research into which many investigators entered with keen zest. Landsteiner published his results in the month of April, 1909, and in the following November, within a few days of each other, Flexner and Lewis, Leiner and Wiesner, Römer, and Landsteiner and Levaditi, working independently in widely separated places, all came in with successful experiments by which they had succeeded in transmitting the disease from one monkey to another. Success had been obtained by employing intracerebral injections instead of intraperitoneal. Since that time a great amount of very valuable experimental research has been done.

In 1913, Flexner and Noguchi of the Rockefeller Institute reported that they had isolated

and cultivated from the central nervous organs of human beings and monkeys that had succumbed to acute poliomyelitis "an exceedingly minute globoid body" which they found to be the causative micro-organism of poliomyelitis. They obtained this micro-organism in pure culture and subcultures. They confirmed their results by successful inoculative experiments. Noguchi perfected a method of demonstrating this microorganism in stained films and sections. It is exceedingly minute and its demonstration is a matter of great difficulty. I recently had an opportunity of seeing this micro-organism in Dr. Flexner's laboratory in New York.

The virus of poliomyelitis will pass through the finest earthenware filter, and filtrates of the infected brain and nervous tissues readily produce the disease in apes and monkeys.

All attempts to produce poliomyelitis in the horse, sheep, dog, goat, guinea pig and mouse have failed. Apes and monkeys are the only animals available for experimental research.

It was soon found that specific antibodies were present in the serum of humans and monkeys who had recovered from the disease. The blood serum from such cases when mixed with the virus destroys the infective power of the virus. The serum retains this power for a very long period. Netter records a case in which the blood serum was found to be still active after thirty-two years.

This "serum test " is of great value; by it it has been possible to prove (1) that epidemics in different countries are due to the same infecting agent; (2) that sporadic cases and epidemic cases are due to the same cause; (3) that the "abortive" cases are true cases of poliomyelitis, and it is also of great value in settling the diagnosis in doubtful cases. The test has the disadvantage of being very expensive as two monkeys are required.

Immunization. – All attempts to find a method of immunizing monkeys which should be reliable and free from danger have so far failed.

The following methods have been tried: –

1. Inoculations with active virus in minute doses.
2. ,, ,, dried virus.
3. ,, ,, virus attenuated or killed by chemical agents.
4. ,, ,, heated virus.
5. ,, ,, mixtures of virus and serum containing antibodies.

So far no method which is effective and free from danger has been found.

Serum Therapy. – At present there does not seem to be much prospect of the successful employment of serum therapy in poliomyelitis. Where monkeys are infected by intracerebral injection of the active virus, the giving at the same time of large doses of serum containing antibodies either by the intravenous, intraspinal or intraperitoneal route produces no results. Even if such a method of treatment were efficacious the fact that the ordinary "serum animals" could not be made use of must prove a great difficulty.

Attempts have been made to treat acute cases of the disease with the serum from human cases which have recovered from an attack. There is a note in the current "British Medical Journal" of the employment of this method by Netter in thirty-two cases. The results are still doubtful.

Method of Spread. – Two main views on the method of transmission of the disease were for a time held. (1) That the disease is communicated by personal contact; and (2) that it is conveyed by insects, the stable fly being held mainly responsible.

Rosenau of Harvard in 1912 reported that he had successfully transmitted the disease by allowing numbers of the stable fly (Stomoxys Calcitrans) first to feed on an infected monkey and by then transferring them to a healthy monkey. Anderson and Frost of Washington also reported that they had conveyed the disease in this way; but many other observers after careful and elaborate experiments obtained completely negative results, and Anderson and Frost failed in all their attempts to repeat their experiment. The fact that only after the injection of *very large* doses of the active virus can the disease be produced when the intravenous route is employed also goes far to establish the improbability of biting insects being the means of spread. After an exhaustive enquiry the stable fly has now left the court without this stain upon its character.

It is now generally accepted that the disease is spread by personal contact by (1) acute cases; (2) by abortive cases; (3) by chronic carriers; and (4) by healthy carriers.

It has been proved that the active microbic agent is at times to be found in the nasal washings of all these classes, and it is generally believed that the disease is conveyed by the passage of this active agent from the upper respiratory tract of one individual to the upper respiratory tract of another. It is possible readily to infect monkeys by rubbing in the active virus to the *abraded* nasal mucous membrane.

It has been long known that the disease spreads along routes of traffic, and that schools are often important factors in spreading the infection,

and there is no doubt that should the disease appear in epidemic form in Belfast the picture palaces would be accused of helping in its dissemination as they have been accused, perhaps with truth, of helping to keep alive our scarlatina.

General Measures for the Control of Epidemics. – The attack rate in epidemic outbreaks of poliomyelitis is as a rule a low one, to this rule it is true there have been exceptions; for instance in the outbreak in Nauru there were 700 cases in an island population of 2,500.

The susceptibility of any community does not seem to be great. Public Health Authorities are correspondingly slow in taking active steps to control epidemics. In England a step in the right direction was taken at the beginning of 1913, when the disease was made compulsorily notifiable. Scotland and Ireland followed but with a shorter step, for in Scotland and Ireland it is left to the option of each sanitary area to adopt notification, or not to do so, as they may think advisable. This is much to be regretted, for only universal notification is of much value. With us in Belfast the disease has been notifiable for a couple of years.

In America where they have had much larger experience, isolation of the cases, and in some places also of the contacts is the rule. At present there is an extensive outbreak in Vermont. In Vermont they are isolating the acute cases for a minimum period of six weeks, and all known contacts for two weeks.

Drug Treatment for Contacts. – It had been found by Flexner that the administration of large doses of urotropin to monkeys before giving to them an infective dose of the active virus modified the results. In some cases the attack was prevented altogether, in others the incubation period was increased from the normal, of five to eight days, up to twenty-four days, and the attack, if it occurred, was generally mild. Urotropin has been much prescribed for known contacts.

From this, I fear, disjointed and necessarily sketchy account of some of the recent epidemic outbreaks of this disease, I hope it will appear that Acute Poliomyelitis is becoming a much more serious medical problem than formerly. Although the attack rates so far recorded have been low, such epidemic outbreaks are a grave danger to any community. How grave is shown not only by the serious case mortality but, perhaps more so, by the fact that epidemic waves of this dread disease leave behind them a trail of maimed and crippled individuals. In some rural districts where severe outbreaks have been experienced scarcely a house is left which does not contain some member severely and permanently handicapped in the struggle for existence. I feel sure that you will agree with me that it is the clear duty of our profession to put forth every effort to prevent such outbreaks, or if that is not found practicable, to perfect our means of dealing with the sufferers.