An Address

ON

THE EPIDEMIOLOGY AND PATHOLOGY
OF POLIOMYELITIS.DELIVERED BEFORE THE PORTSMOUTH DIVISION OF THE
BRITISH MEDICAL ASSOCIATION,

BY

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The word "poliomyelitis" nowadays is restricted to the clinical and pathological results of a definite specific infection of the anatomy, though the part invaded may be the brain, the cerebellum, the spinal cord, or the peripheral nerves, or any combination of these structures. The term "polio-encephalitis" is no longer used, nor do we speak of "anterior poliomyelitis," nor of "acute poliomyelitis," nor of "chronic poliomyelitis," but simply of "poliomyelitis," to denote this peculiar specific fever which has the incidence of its effects chiefly upon the nervous system.

This malady has been incident upon humanity since very early times, as the references in classic literature to "he that hath a withered limb" will tell you, or the many portrayals of the deformities produced by the malady in early sculpture and mediæval paintings will show you, but never until our lifetime has it been known as a pestilence which, affecting one small district, in a few years claimed 50,000 victims; nor as a plague which in three short months could leave behind it 5,000 dead and 12,000 paralysed, as it did in New York State in 1916; nor as a swift scourge which, lighting upon the little village of Stoke Rivers in Devonshire, could lay low in a few days 45 persons out of a total population of 119.

These events are new to the world of medicine in which we have lived, for when I joined our profession poliomyelitis was not described as an infectious disease, though an epidemic of 11 cases in one village had been recorded as early as 1843 by Colmar. There were other small community outbreaks in my early years, but it was an outbreak of 1,500 cases in Sweden in 1805 which led to the most important discoveries upon the nature of poliomyelitis. On account of the geographical nature of Sweden and the distribution of its population in small village communities with few lines of communication, the spread of the infection was comparatively easy to trace, and Wickman, who investigated this epidemic, was able to prove that the disease only travelled along the lines of human communication, that it would not cross from village to village over a ravine, even though these villages were only a hundred yards apart, unless there was a road or a bridge or a boat by which human beings could pass. Moreover, he proved that whenever infection spread to a fresh village, someone had visited that village from a definitely infected village. That person was obviously one who was not sick with the disease, nor was he one who subsequently became affected.

Wickman therefore proved the very important facts that poliomyelitis is conveyed by human contact only, and usually by those who do not suffer with symptoms of the malady. He proved the importance of carrier infection. Wickman made very important clinical observations as well. He found eight different types of illness with nervous symptoms common during this epidemic, in addition to many cases of fever in which there were no nervous symptoms. Some of these were very unlike the usually accepted clinical picture of poliomyelitis. Many years before, however, Strumpell and others had stated their belief that acute encephalitis was really poliomyelitis affecting the brain, and Wickman boldly asserted that all the dissimilar illnesses observed by him in the Swedish epidemic were manifestations of one and the same infection; and his assertion was before very long a proved fact. He had able pathologists to help him, and with the advent of the American epidemics great advance was made by Landsteiner and Popper, by Flexner and Lewis, by Taylor and Amos, and by Levaditi.

These workers have succeeded in infecting monkeys with the disease by means of emulsions of the spinal cord from a fatal case, and by means of washings from the nasopharynx of any person recently ill with the disease. The infection can be attained by injecting the virus subcutaneously, or into any of the serous cavities, or into a nerve trunk, and most certainly of all into the central nervous system; but, curiously, intravenous injection will never produce the disease, for the blood seems to destroy the virus at once. They also communicated the disease by smearing the virus on any of the mucous surfaces, or rubbing it upon the fingers of the monkey or upon the bars of his cage so that he could contaminate his own mucous membranes. The malady can be propagated through an endless strain of monkeys, with the production in each of a fatal disease commencing with pyrexia and general illness and terminating with paralysis. Very nearly the same changes are found *post mortem* as are found in human cases, so that the clinical and histological picture in man and in the monkey may be said to be identical.

Investigating all the members of a household in which there was a case of poliomyelitis in the acute stage, Kling, Petersen, and Wernstedt found that they could obtain the virus from the nasopharynx of 30 per cent. of the non-affected inmates, and therefore proved the important fact that there are always three other people who carry, and are therefore able to transmit the malady, for every one who suffers. As I shall try to prove, the disease is never transmitted by the one who is suffering from it, but only by the carriers; this has a very wide bearing when dealing with the malady from a public health point of view.

The virus of poliomyelitis thus obtained resists desiccation for twenty-four days, and a mixture of 50 per cent. glycerin, which kills most bacteria, has no effect whatever upon its potency, and advantage is taken of this fact to prepare the virus uncontaminated. Nor is it affected by 5 per cent. carbolic acid; but it is instantly rendered impotent by hydrogen peroxide or by a 1 per cent. solution of permanganate. The virus will pass through two inches of solid porcelain under pressure, which proves that, if particulate, the organism is very small. It is therefore known as a "filter passer." But if this virus is passed again and again through a fresh porcelain filter it becomes less and less potent with each passage, which suggests that the virus is particulate and only just small enough to pass with difficulty the pores of the filter. Actually the organism is said to be a very small round thing, which has been called a globoid, of which fifty would lie easily on the surface of a red blood corpuscle. It is claimed that this globoid virus can be cultivated upon Noguchi's medium—a mixture of peritoneal fluid and rabbit's kidney—and that the globoids can be seen *in situ* in the lesions in every case of poliomyelitis.

This virus is communicated from person to person by "droplet infection," which means that whenever I speak to one of you—say at a yard's distance—I spray you continuously with droplets from the surface of my respiratory passage; and if I am a carrier of poliomyelitis I spray you with the virus, some of which is likely to reach your respiratory mucous membrane when you are inspiring. In this untoward event one of three things must happen to you as the result of your receiving the virus: either (1) you develop the disease, or (2) you become a carrier and capable of handing the infection on to another person, or (3) you become immune relatively or absolutely. Whether you become a case, a carrier, or an immune person will depend, first, upon the dose of the virus which you receive from me per unit of time; secondly, upon your personal power of destroying that virus; and thirdly, upon how quickly the protein content of the virus stimulates or depresses your immunizing mechanism. These two last factors being of average intensity the result will depend upon how far I am from you when I spray you with the virus.

Now if a person develops the disease, the virus which is multiplying upon his respiratory mucous membrane seems at once to lose its virulence, so that a case of poliomyelitis does not seem able to convey the infection to another

person, though the virus may be still found upon the mucous surfaces for about a fortnight.

If a person becomes a carrier the virus is likely to become more virulent for a time, but soon it loses its potency and the person ceases to be a carrier in a fortnight or so. There are no chronic carriers as there are in connexion with diphtheria and epidemic meningitis.

It seems, then, to be a recent carrier who transmits and spreads the disease, and a chain of carriers who carry on the infection during those times when poliomyelitis is not about and give rise occasionally to those cases of poliomyelitis which are called the "sporadic cases," as distinguished from the "epidemic cases."

I want it very clearly to be apprehended that though the virus can be obtained from the mucous membrane of cases for some days after the disease has appeared, and monkeys can be inoculated with the disease from this virus, that does not mean for one moment that a patient even in the early days of his illness can deliver an infective dose of the virus to another person. There is strong evidence that a case of poliomyelitis is not infectious, and that a carrier, especially a recent carrier, may be very infectious. As this question as to whether poliomyelitis is infectious from case to case, or not, is one of paramount importance, I will state the evidence briefly. Let me in the first place give the story of a carrier. One day at the market town a farmer from a country village sold a horse to another farmer who lived some twenty miles away, and as part of his contract had to deliver the horse at the farm of the purchaser. He started from home leaving his child ill with poliomyelitis. He took the midday meal with the purchaser and sat between two of the children at table. Within forty-eight hours both of these children were ill with poliomyelitis and a few days later their mother and another child were stricken with the disease. Here were four members of one household stricken by one short visit of a carrier, with no possible source of infection from elsewhere. Probably, too, this father had infected his own child, for adults are often carriers.

Such instances as the story of the farmer just recited could be multiplied indefinitely—the Swedish epidemics teemed with them. But when we come to seek for evidence of case-to-case infection we have to search the literature carefully for any event put forward as an example. And when the few recorded instances are examined it is found in every case that it is one child of a household who is said to have infected other members of the household or other contacts, and we know full well that whenever a case of poliomyelitis appears, and as soon as it appears, 30 per cent. of the members of that household are already carriers and provedly capable of infecting; and in any case it is at least three chances to one against the sufferer from poliomyelitis being the infector of others, even if he were capable of infecting, which I submit he is not.

The only real test that would prove a developed case to be capable of infecting others would be to remove the patient in the early pyrexial stage and place him in a community of susceptibles. Put him in the children's ward of a hospital and see what happens. I have seen this done, as all of you have seen it done, on scores of occasions since we first entered hospital, yet there is not one single record in the whole of medical literature of such a patient transmitting the disease to another child in hospital. Dr. Batten, who had thirty years' experience of the Great Ormond Street Hospital for Sick Children, went into this point very carefully in the Lumleian Lectures for 1916, and vouched that no case had ever occurred in that hospital, where acute cases of poliomyelitis were frequently admitted into the general wards. I think that this evidence is very conclusive, but I know how difficult it is to get the public to accept it. In the lay mind one case of infectious disease must always arise from another similar case, and in deference to such an opinion it is wiser to isolate patients.

I would like to submit to you my opinion that when an epidemic of poliomyelitis occurs in a community it is preceded by a carrier epidemic in which for a time no cases occur but many carriers are made, and that, with the increasing advent of carrier infection to susceptibles, the virulence of the infection is raised and at last definite

cases occur. By this time the infection is all through the community, and doubtless much immunization has already occurred. This is probably the reason why poliomyelitis seems in its epidemic form to avoid schools, institutions, and towns, and densely populated districts where there is much daily mixing of the community, and to prefer sparsely populated country districts, where communication between house and house and from village to village is more restricted. It seems to me to be the only explanation of the occurrence of the first fifteen cases in an epidemic on the same day in fifteen widely separated places of one country-side, as happened in one of the English epidemics. Such a preceding carrier epidemic best explains the curve of incidence in an epidemic, with its rapid blaze, high peak, and rapid fall, all the cases occurring within a few days and then no more cases. All the cases at Broadstairs, for example, occurred between October 13th and November 9th, and there were many more cases among the general population than in the schools.

Now if this description which I have put forward is just it follows that when a case of poliomyelitis occurs in a semi-isolated community such as a school which has been assembled, say, for several weeks, there has already been a carrier outbreak going on for some time, and all the boys are already infected as cases or carriers or they are immune. The actual cases are not infectious, and all the damage that can be done has been done so far as the boys are concerned. Therefore keep them together, and the epidemic will die out with no further trouble than would have occurred in any case. But before the epidemic has died out do not disband the carriers, by whom alone infection can be spread, and scatter them over the country for the continuance of the disease.

The virus of poliomyelitis has never been found except upon the mucous surfaces or within the nervous system. It has been proved to be capable of passing through the whole length of the intestine with undiminished potency, and therefore any part of the respiratory and alimentary mucosa may be the point of advent of the poison to the nervous system, and it seems likely that the point of advent may determine which part of the nervous system is chiefly attacked. When once within the nervous system the virus seems to travel fairly readily.

It follows from these facts, and from the fact that the blood stream will not convey the virus but seems to destroy it at once, that the general swelling and parenchymatous degeneration which is found in the lymphoid tissue of the body generally, and in the liver and spleen, must be the result of some toxin produced by the virus.

The virus gains access to the nervous system by one path only, and that is by travelling along the nerves, either along the perineural lymphatics or along the axon, just as the virus of rabies does or as do the toxins of tetanus and diphtheria. It has recently been proved that the path is by the anterior roots only—there seems to be a trap at the posterior root ganglia which prevents access by the posterior roots; for if the anterior roots be divided injection of any of these poisons into the corresponding nerve is not followed by infection, whereas if the posterior root be divided infection takes place as in the intact animal. This may be the explanation why the damage in poliomyelitis falls most heavily upon the ventral horns of the spinal cord, which are in close connexion with the anterior roots.

The effect of the virus upon the nervous system is twofold. First there is a poisoning of the nerve elements which destroys their function. This poisoning may be recovered from, or it may end in degeneration of the nerve element. It is the recovery from such a poisoning effect which is responsible for the rapid clearing up of the paralysis, or of part of the paralysis, which one so often sees in the first week or two of the illness. Secondly, there is a local inflammatory reaction, often so intense as to lead to actual necrosis of the affected region and frequently associated with many small haemorrhages. The actual inflammatory process is the outpouring of lymphocytes in very large numbers round the blood vessels with marked oedema of the tissues, and, so far, it is exactly like the lesion of lethargic encephalitis and the lesion of syphilis, but in poliomyelitis there is always a considerable number

of polymorph cells mixed with the lymphocytes, which will at once distinguish this lesion from that of syphilis and lethargic encephalitis.

Whenever the inflammatory lesion reaches the surface of the nervous system and the meninges there will be outpouring of cells into the cerebro-spinal fluid and a recognizable change in this fluid, immensely important in diagnosis, together with signs of meningitis. Now as meningitic symptoms, such as headache, head retraction, diplopia, nystagmus, vomiting, and pains in the back and limbs with pyrexia, are among the commonest of symptoms of all cases of poliomyelitis, especially when this disease is epidemic, it will interest you to know that from the first symptom of the disease the cerebro-spinal fluid of poliomyelitis is highly characteristic, and that a lumbar puncture will not only relieve the headache and pains and possibly lessen the severity of the disease, but it will help you to a diagnosis in at least 98 per cent. of the cases. There is an increase in the protein content, and often a fine spider-web clot forms on standing for a few moments. The chlorides are normal: this will at once distinguish it from tuberculous meningitis, in which the chlorides are characteristically subnormal. The sugar is normal: this will at once distinguish it from all forms of bacterial meningitis, for in every one of these the sugar is below normal. There is an increase in the cells, which may be lymphocytes or polymorphs or both. The culture is sterile.

There is only one other acute pyrexial disease which gives a fluid sometimes like this, and that is lethargic encephalitis, but I wish you especially to remember that the polymorph cells are never found in the cerebro-spinal fluid of lethargic encephalitis, and when these are present the diagnosis must be that of poliomyelitis.

CONGENITAL IDIOPATHIC DILATATION OF THE COLON TREATED BY STRETCHING OF THE PELVI-RECTAL SPHINCTER.

BY

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CONGENITAL idiopathic dilatation of the colon (Hirschsprung's disease or megalocolon) is described in textbooks as an enormous dilatation of the colon with hypertrophy of its walls, affecting all or part of the large intestine. The parts most frequently affected are the descending, iliac, and pelvic colons.

The object of the present note is to advocate the trial of a method of treatment which aims at dealing with the cause. L. E. Barrington-Ward¹ in 1914 gave a full account of the disease and supplied a list of thirteen different views as to its causation, under the headings of "Mechanical," "Neuropathic," "Inflammatory," and "Congenital." He referred to *post-mortem* examinations made at Great Ormond Street Children's Hospital on nineteen undoubted cases, in all of which the sigmoid colon was hypertrophied. In ten of these cases the hypertrophy also involved the rectum as far down as the anal canal. In no case was any mechanical obstruction to be found, though in one the bowel was said to be kinked as it turned into the pelvis. After discussing the various theories he states:

"The theory that best explains this series of cases is as follows: There is a congenital abnormality in the lower end of the hind gut which takes the form of a muscular hyperplasia. It usually involves the rectum and extends for a varying distance up the pelvic and iliac colon. . . . In some cases there is definite localization of the change (as in three cases described) where the hyperplasia has affected a segment of the intestine chiefly. . . . The physiological functions of the intestine are in abeyance, a block is produced, and the bowel behind, for a varying distance . . . dilates and hypertrophies."

A. F. Hurst² believes that this affection is due to achalasia, or absence of the normal relaxation of the circular muscular fibres found at the pelvi-rectal flexure, or of the anal sphincter. He holds that there is a close similarity between what is known as cardiospasm (or, as he terms it, achalasia of the cardia) and megalocolon.

A. J. Walton,³ in discussing the surgical treatment of cardiospasm, describes fourteen cases in which he adopted the method suggested by Mikulicz—that is, opening the stomach and digitally stretching the lower constricted portion of the oesophagus. The result in these fourteen cases was entirely satisfactory.

THE PELVI-RECTAL SPHINCTER.

The lower part of the large bowel consists functionally of two parts, a colic part and a rectal part, and between these there is a functional junction. In the dead body, hardened with carbolic acid and formalin, the functional tract shows considerable variety in form and appearance. As a rule, however, it is a definitely contracted region of the bowel, always empty, and in strong contrast with the part above, and, to a less degree, with the part below.

Under normal conditions the faeces do not at once pass through this tract, but collect in the movable pelvic colon above until just before defaecation, when they are discharged into the fixed rectum below. Between the two parts there is an angulation, especially in the erect position. W. J. Mayo⁴ has shown that the mucous membrane of the last two inches of the colon is disposed in longitudinal folds, contrasting with the circular arrangement in the rest of the colon. This favours the suggestion that the segment referred to is normally obliterated by a sphincteric action of its circular muscular fibres. A similar arrangement is seen in the abdominal portion of the oesophagus, and in the pyloric end of the stomach.

It has not yet been shown that there is developed in the pelvi-rectal tract a sphincter comparable with that found at the pylorus, but over its whole length, which varies from half an inch to two inches, it is believed that there is a sphincteric action of the circular muscular fibres sufficient to obliterate the lumen and to withstand the weight of the contents above. The lumen becomes patent when the circular muscular fibres of this tract relax. Interest is being taken in this matter at Queen's University, and Professor Walmsley believes it is possible to demonstrate a more definite sphincter in the foetus.

Recently I had the opportunity of examining the rectum of a woman, aged 28, in whom I had, six months previously, excised the entire colon with a few inches of the terminal ileum, for multiple papillomata of the former. The junction of the ileum with the lower bowel was made about eight inches from the anus—that is, well above the pelvi-rectal tract. I expected in this patient that the rectum would be full of liquid faeces, but it was empty, and it was only on passing a sigmoidoscope some distance up that semifluid faeces began to escape. The presence of an efficient pelvi-rectal sphincter may be assumed in this case. The patient can retain faeces for many hours at a time, the bowels acting from two to five times in the twenty-four hours.

TREATMENT OF MEGALOCOLON.

Medical.

By means of enemata, massage of the colon, liquid paraffin, and aperients, a certain proportion of cases may be benefited, but the mortality is very large, some authors placing it as high as 74 per cent.

Surgical.

Barrington-Ward, in the paper referred to, thinks that a fair estimate of the surgical mortality would be about 36 per cent. He considers that the ideal form of treatment is the removal of the whole of the affected bowel down to the anus. He advocates, in young children, operation in two stages. At the first operation the ileum is divided and implanted as low down in the rectum as possible. Some months later, when the child's strength has increased, the large bowel above the anastomosis is excised. Treatment on these lines has been carried out by Arbuthnot Lane and others with success.

An operation of the magnitude of colectomy in a debilitated child, even if performed in two stages, is a terribly severe measure, and would undoubtedly, if universally adopted, be attended with a high rate of mortality. Moreover, it seems unscientific and incorrect to remove a hypertrophied organ striving to overcome an obstruction, instead of seeking out and dealing, if possible, with the cause.