ETIOLOGY OF POLIOMYELITIS.

BY

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DURING the past seven years poliomyelitis has been met with as an epidemic in many parts of Europe, America (including Canada and the United States), Australasia, etc. I need not refer to Wickman's classical description of this disease. New York in 1907 had a total of over 2,400 cases, and about one-half of this number in 1908. In 1909, during the months of July, August, September, October, and the first part of November, an epidemic of this terrible disease occurred at Montreal. It was widespread, most of the cases being from the poorer districts. Some, however, were found in the suburbs of the city and a few in the country districts, within a radius of fifty miles. Several hundred cases were treated here, between 50 and 60 of which were under my personal care.

During the autumn of 1910, while we have had only a few cases in Montreal, probably six to eight altogether, the epidemic has been present in many districts of Quebec and Ontario. In the Province of Quebec many cases have occurred at Sherbrooke and North Hatley; while in Ontario the disease has been prevalent in both Toronto and Hamilton, some one hundred cases having occurred at Hamilton alone.

The epidemics have differed in severity in different places. In Montreal we have not had many fatal cases. Among the number treated by me personally, the results were fatal in only three instances, or, in other words, a fatality of a little under 6 per cent. In many places, however, they have had a fatality of 12 per cent., and even as high as 17 per cent. In this respect it resembles typhoid fever.

Although the number of fatalities has not been great, the disease has added a great number of crippled little ones to our homes. It is, indeed, a sad sight, after an epidemic of this type, to see the number of children crippled and maimed for life.

During the past five years great progress has been made in the pathology of this peculiar disease. We have had opportunities to study it in recent cases which have ended fatally, and much of interest has been written on the subject.

What about the etiology?

In all the great medical centres distinguished pathologists and bacteriologists have devoted a great deal of

time, attention, and skill in an endeavour to ascertain the cause of poliomyelitis. Landsteiner and Popper have done the pioneer work. They inoculated two monkeys successfully with the spinal cord obtained from two fatal cases of poliomyelitis. The injections were made into the peritoneal cavity, and one of the monkeys died on the sixth day; it was paralysed in the lower extremities. The other was killed on the nineteenth day, and the pathological changes were the same as those found in fatal cases in the human beings.

It remained for Flexner and Paul A. Lewis, at the Rockefeller Institute, New York, to make another step in advance. They succeeded in producing the disease in six successive monkeys. They employed intracerebral and intraperitoneal injections, but at this time they failed to find or to cultivate any organism. They have pointed out that portions of a bacteria-free filtrate were inoculated into a bouillon, containing 10 per cent. of rabbit's serum, which had been rendered perfectly clear and sterile by being put through a Berkefeld filter. On the second day the fluid in the upper half of the tube was cloudy. On the fourth day the fluid was used for inoculation into a monkey, which developed paralysis on the thirteenth day. This is practically the same inoculation period for typhoid fever. Flexner has definitely established the infectious nature of the virus. However, they have made sections and film preparations without having found either bacterial or protozoal parasites that could account for the infection, so that they have concluded that the infecting agent of poliomyelitis belongs to the class of minute and filterable viruses which have not thus far been demonstrated with certainty under the microscope.

Landsteiner and Lavaditi showed that not only was the virus capable of being transmitted through a series of monkeys, but that it was so minute as to be able to pass through the finest filter.¹

Dr. Holt cites instances where 7 cases occurred in one family, 5 where 3 were affected, 5 where 4 had it, and 40 in which 2 were attacked. In 37 of the cases the interval between the first and second cases was ten days or less.² This certainly points to its contagious nature.

Several of my little patients who developed it lived in the same yard; and in other cases several lived in the same street and on the same side of the street. I think that it can rank with typhoid fever as far as contagion is concerned.

During the past few months I have been much impressed with the similarity between typhoid fever and poliomyelitis.

1. In the first place, both diseases occur in the late summer or fall months.

Table showing the Results of Examination of the Blood of Thirteen Cases of Poliomyelitis, and for Comparison the Blood of Three Normal Cases of Members of Infected Families to prove that the Widal Reaction was not a Family Peculiarity.

Initials.	Age.	Parts Paralysed.	Date of Onset.	Date of Examination.	Widal Reaction.		
					Positive.	Highly Suggestive.	Negative.
1. U. C. ...	3 years	Left and right legs	Nov. 2	Nov. 23	—	Yes	—
2. J. M. ...	4½ years	Right and left legs, left arm, thoracic muscles	Oct. 18	Nov. 23	—	Yes	—
3. B. M. ...	—	Right and left legs	Oct. 30	Nov. 18	Yes	—	—
4. B. L. ...	—	Both legs	Oct. 10	Nov. 14	—	Yes	—
5. M. L. ...	—	Normal case	—	Nov. 14	—	—	Yes
6. B. W. ...	19 months	Left leg	Sept. 25	Nov. 17	Yes	—	—
7. B. M. ...	14 months	Both legs and upper arms	Sept. 4	Nov. 4	—	Yes	—
8. E. S. ...	28 years	Right arm and leg	Sept. 17	Nov. 20	Yes	—	—
9. S. W. ...	1½ years	Both legs	Oct. 18	Nov. 20	Yes	—	—
10. H. F. ...	14 months	Both legs	Sept. 15	Nov. 20	Yes	—	—
11. F. J. ...	4 years	Both legs	Sept. 25	Nov. 20	Yes	—	—
12. J. K. ...	3 years	Right leg slightly	Aug. 20	Nov. 20	—	—	Yes
13. Mr. W. ...	28 years	Normal case	—	Nov. 20	—	—	Yes
14. P. C. ...	1½ years	Left leg	Sept. 10	Nov. 20	—	—	Yes
15. F. J. ...	2½ years	Left leg	Sept. 25	Nov. 20	—	—	Yes
16. J. M. ...	5 years	Normal case	—	Nov. 20	—	—	Yes

2. As soon as the cold weather commences both, to a great extent, become extinct. During the late summer and fall months of 1909, while we had a severe epidemic of typhoid fever in Montreal amongst the adults and young people, at the same time we had an epidemic of poliomyelitis amongst the young children and babies.

3. Diarrhoea is just as common a symptom in poliomyelitis as in typhoid fever.

4. The incubation period of one is practically the same as that of the other. This has been proved by Dr. Flexner to be thirteen days experimentally, and Dr. Holt found it to be ten days clinically.

5. Both diseases are prevalent in large cities and small towns at the same time where and when the cause of typhoid fever is traceable to the water and milk supply; whilst, on the other hand, where the supply of water and milk is pure, both of these diseases are less frequent. It is practically the same degree of contagion.

These observations led me to believe that the micro-organism of poliomyelitis might be of the same character as the bacillus of typhoid fever.

Upon examination of the blood in cases suffering from poliomyelitis, I have found the Widal reaction present as in typhoid fever, proving conclusively that the disease is from the same source and cause, and that the germ of the one is allied to that of the other; further, I do not hesitate to make the assertion that the precautions necessary to prevent typhoid fever will also have the same effect in arresting the spread of poliomyelitis.

It will be seen that I obtained the Widal reaction in 6 cases of the blood examination out of a total of 16 cases. In four instances the report is highly suggestive, while in 6 cases the result was negative. However, three of these were taken from individuals in good health to prove that the Widal reaction was not a family peculiarity. This only leaves 3 negative cases to deal with. Two of the negative results were obtained from cases of poliomyelitis about two and a half and three months from the time of onset of the disease, and both of the cases were mild in character, only one leg being partially paralysed.

It appears to me that these results correspond fairly well with the results which would be obtainable from the examination of a similar number of typhoid patients. I may add that the blood was examined in every case by physicians who make a speciality of this work.

As typhoid fever affects the most vulnerable part of the adult—the intestinal tract, so poliomyelitis attacks the weakest point of resistance in the child, which is the nervous system.

As to treatment, there is no doubt that within the near future a serum will be obtained; and, if so, in suspected cases, during an epidemic of poliomyelitis, it is well to look for the Widal reaction, and, if present, the serum should be injected, which should have the effect of either modifying or preventing the development of the disease. In cases in which the disease has already developed, to prevent further extension the serum might be injected into the spinal canal, and would be likely to have an effect similar to that obtained by the method of treatment in cerebro-spinal meningitis.

REFERENCES.

¹ Editorial note, *Archives of Pediatrics*, February, 1910. ² New York Academy of Medicine, Section on Poliomyelitis, January 20th, 1910.

THE late M. Auguste Loutreuil, a Frenchman who made a large fortune by industrial enterprise in Russia, has bequeathed £120,000 to the Académie des Sciences, £100,000 to the Sorbonne, £40,000 to the Fund for Scientific Research, and £4,000 to the Paris Pasteur Institute. The money is intended to be applied in furtherance of scientific research.

It was recently decided to form a Plymouth, West Devon, and Cornwall branch of the Research Defence Society, a Devon branch already existing under the presidency of Mr. Eden Phillpotts. The following are officers of the branch: President, Mr. W. Paul Swain; vice-presidents, Admiral Marrack, Colonel Giles, and the Rev. Dr. Trelawney Ross; honorary secretary, Dr. R. Jaques; honorary treasurer, Mr. Cyril A. Pode; committee, the president, honorary secretary, honorary treasurer, Miss Kirk, Colonel Giles, with two others yet to be appointed. A ladies' committee is to be formed.

THE GASTRIC ORIGIN OF ANGINA PECTORIS.

BY

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ON January 2nd, 1911, when I entered Dr. X.'s room he was suffering from an anginal seizure which had already lasted half an hour. He had been making ineffectual efforts to expel wind from his chest, and allowed me to introduce an oesophageal tube, a procedure which resulted in an exhaustive liberation of gastric flatus. The effects of the operation became instantly apparent in the altered aspect of the patient. His colour returned, and he seemed no longer in pain. The seizure was manifestly at an end. On the last day of January Dr. X. informed me that on two occasions he had again resorted to the operation of gastric deflation, and upon each he had brought the seizure to a sudden and abrupt close. Dr. X. is 63 years of age, and has suffered from anginal seizures from time to time for several years. His first severe attack occurred in 1908.

When a seizure has been brought to an abrupt and complete termination by the introduction of the oesophageal tube, as in the case of Dr. X., and when later seizures have shown themselves amenable to a similar operation, the fact that the seizures are the result of tension of the gastric atmosphere is demonstrated beyond question.

Admission of this fact involves the recognition of the theory of the gastric reflex in the case of Dr. X., and also having regard to all cases of the type to which Dr. X.'s seizures genetically belong.

This type appears to be a prevalent one, since in descriptions of anginal seizures gastric symptoms are often mentioned as holding a prominent place. Time after time in an account of this disease Dr. James Mackenzie refers to flatulent distension, and even propounds the theory of air-swallowing to account for it. Moreover, Heberden in his classic description states that the seizure comes on more particularly when the patient walks after eating.

Probably in the same category are to be classed cases dying in an anginal seizure when at the autopsy no traces of disease in the coronary arteries are found. These number nearly 50 per cent. in a long series of collected cases.

If this be so, the theory of the gastric reflex must be admitted to occupy at least an equal place in our regard with that of the cardiac reflex.

Recently a case came under my notice demonstrating the fact that under certain favouring circumstances tension of the gastric atmosphere is associated with painful attacks indistinguishable from those of angina pectoris.

Mr. M., aged 62, for many years a subject of flatulent dyspepsia, had lately lost flesh, and in the course of his wasting had developed seizures of a painful character. The pain affected him when he walked and on going upstairs. It was felt in the region of distribution of the upper intercostal nerves including the costo-humeral branch. The seizures came on earlier in the walk after he had taken food than when he fasted, they were associated with sensations of gastric distension and they subsided upon free eructation of flatus. Seizures of a similar character often awakened him from sleep, and alike were relieved by expulsion of gas from the stomach. Mr. M. had suffered for years from attacks of flatulence coming on when he walked and disturbing his rest at night, but never before had they been accompanied by pain. Since pain had grown severe morphine had been taken, but with no satisfactory result. In this case the exhibition of nitroglycerine* proved to be exceedingly effective in the relief of pain, seizures always subsiding immediately after its administration, and its efficiency remained unimpaired until the day of his death, which occurred about eight weeks afterwards. The *post-mortem* examination revealed the presence of carcinoma which had infiltrated the wall of the oesophagus in the lower third of its length. There were secondary growths in the coeliac glands and in the neighbourhood of the liver. The oesophagus was slightly dilated in the region corresponding with carcinomatous infiltration and the cardiac orifice was patent. The heart, coronary arteries, and systemic vessels were quite healthy.

The seizures of pain in this case might be explained as being the result of pressure from a tense gastro-oesophageal atmosphere transmitted through the oesophageal wall and impinging upon the fibres of the plexus gulæ. The plexus gulæ surrounds the oesophagus in the lower

* The action of the nitrites is upon every kind of plain muscle throughout the body (Dixon's *Pharmacology*, p. 194).