THESIS

FOR THE DEGREE OF M.D.

That ACUTE ANTERIOR POLIOMYELITIS is a SPECIFIC INFECTIOUS DISEASE, with a DEFINITE INCUBATION PERIOD.

With a REPORT of an OUTBREAK at FORTH, LANARKSHIRE

by

JOHN REID, M.B., Ch.B.

FORTH,

LANARKSHIRE,

May, 1912.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

P R E F A C E.

In the Autumn of 1910 a number of cases of Acute Anterior Poliomyelitis occurred in the Upperward of Lanarkshire - the time that a wave of the disease passed over Great Britain. Four cases occurred in my own practice in the district of Forth. To my certain knowledge there were no cases of the disease in this district during the preceding eight years. I submit the history of the four cases and a critical study of our present knowledge of epidemic Poliomyelitis as my Thesis for the degree of M.D.

A B S T R A C T.

Section 1: Introductory.

2: Historical. -

Report of 4 cases illustrating incubation period, mode of transmission.

3: Aetiology. -

Infection of protozoan nature: possibly conveyed by biting insects.

4: Pathology.

5: Incubation Period.

6: Transmission.

7: Immunity.

8: Mortality.

9: Symptoms.

10: Differential Diagnosis.

11: Prognosis.

12: Treatment.

INTRODUCTORY.

Section I.

Anterior Poliomyelitis my be defined as an infective communicable disease which exercises a special selective action on the nervous system. The symptoms to which it gives rise depend on the portion of the nervous system which is affected. Until the present century sporadic cases only were reported, but of recent years the disease has assumed epidemic form, and very numerous epidemics of worldwide distribution have occurred. Although the mortality is not very great, the disease derives considerable importance from three of its features.

(a) Its incidence is mainly upon young children.

(b) In the majority of cases, permanent paralysis of one or more limbs results so that it cripples its victims.

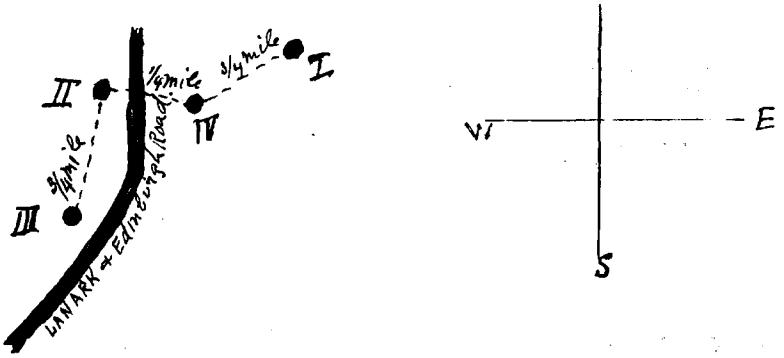
(c) It is definitely infective.

An outbreak of the disease occurred in the Autumn of 1910 in the Upper Ward of Lanarkshire - at the time that a wave of the disease passed over Great Britain. No cases of the disease had occurred in the district during the preceding eight years. Four cases were under my care.

The district is situated in Lanarkshire more than 900 feet above sea level; it is bleak moorland, and the soil is mossy and damp. The inhabitants are chiefly occupied in coal mining. They are fairly prosperous, and their houses are well built of stone. There are no local conditions or circumstances which require /

require description, but it may be stated that the old water supply which was very inadequate in 1910, consisting of one small spring in a glen fully a quarter of a mile away from the village, the water being carried in pitchers, has been replaced by a supply distributed by gravitation. There is no system of drainage, the sewage is discharged into an open gutter running along the fronts of the houses.

Three of the cases occurred in different rows of miners' houses, and one in a semi-detached cottage. The relative position of the affected houses is shown in the annexed sketch on which the distances are indicated.



I shall first of all relate the history of the four cases, pointing out the noteworthy features of each, and then I shall proceed to discuss the Aetiology, pathology, differential diagnosis of the disease, as well as the principles of its treatment.

Section II. HISTORICAL.

Case 1. History of Cases.

On Sunday, July 24, 1910, I was called to see a boy D. S. aged four and half years. He was on a visit to his grandmother. On Saturday July 16th he was taken by his parents ffom his home to a neighbouring village to visit an aunt who had a child ill with "Infantile Paralysis."

They returned home at night, and on the following Wednesday, the 20th, his mother and he came here to visit his grandmother. The boy was perfectly well on his arrival, but during the night he became restless, sick, and feverish. He vomited a little. His condition did not alter much during the next three days, Thursday, Friday, and Saturday, and as he was not improving I was sent for on Sunday.

On examination I found the boy was very pale, but well nourished. He was sweating profusely, and complained of pain in the joints when they were moved or touched. His temperature was 102° F, pulse 112, and respirations 28. He had not had diarrhoea. With the exception of some fine crepitations at the base of the left lung, physical signs were negative as regards the chest.

On the 25th and 26th his condition was practically unchanged. On the 27th the temperature fell to 99° F. and the boy said he was better. The pain in the joints was gone, but it was found that he could not move his legs. The knee jerks were absent, as were also the plantar reflexes. He did not /

not lose control of the sphincters. On this date he could move his arms, but on the 28th and 29th he complained of stiffness in the arms, and had difficulty in raising them. On the 30th the arms were almost powerless, and on the 31st were quite paralysed. To quote his mother's words on the 30th, "His arms are getting gradually weaker. At first he could put his arms round my neck and pull himself up, but now he can hardly lift his hands." He remained in this condition for two or three weeks, and at the end of that time, he began to move his arms, but had no power in his legs.

The muscles of the legs were flaccid, but apparent wasting was not great.

This child did not improve very much. He was able to raise his hands to his head and flex his toes, but he could not move his legs. He was in this condition till February of this year, when he contracted pneumonia of which he died on February 5th.

Case 2.

On September 5th 1910, I was asked to see W. McC., male, aged one year and nine months. I found the right lower limb completely paralysed and flaccid. The knee jerk and plantar reflex were absent.

The mother informed me that she first noticed the condition that morning when she found that the child could not stand. He had been"cross" for a few days, and had vomited, but she attributed /

attributed these symptoms to teething.

The muscles of this child's leg are still paralysed, and there is also considerable wasting of the muscles.

Case 3.

P. T., male, aged 1½ years, was brought by his mother to my house in September 1910. The mother said he could not move one of his legs - the right - which I found was paralysed. She had not noticed anything unusual about him previous to the paralysis, except that he was restless, had started during his sleep in the two or three days previous, was putting up his food, and was suffering from what she called "hives." The plantar and patellar reflexes were absent.

This child made an excellent recovery. There is no paralysis, and no wasting.

Case 4.

On Wednesday September 14th 1910, I was asked to see a little girl A.E.B., aged 2 years and 2 months. Her guardian said she had been feverish, restless, and excitable during the previous day and night. Her previous health was good.

On examination nothing abnormal was found in any of the organs. The temperature was 100.8° F. The child had vomited, but there was no diarrhoea. On Thursday and Friday her condition was practically as stated - restless and feverish at night. On Saturday morning the 17th, when her guardian awoke, she lifted the child and found the left arm and left

leg /

leg were powerless - "Just like a flail." She noticed also twitchings of the right side of the face. She said the spasm of the face occurred every few seconds for two or three minutes and then stopped. (When I saw her there was paralysis of the right side of the face and difficulty in swallowing).

The muscles of the left arm and leg were flaccid. On Sunday, the child had several convulsions. There was no retraction of the head and Kernig's sign was absent.

I performed a Lumbar puncture and drew off about an ounce of cerebro-spinal fluid under pressure. This fluid was clear, and no microbes were found on microscopic examination. The child's mind was quite clear on Monday and she had no convulsions that day, but on Tuesday they returned. Respiration became involved, and she died early on Wednesday morning from respiratory paralysis.

Historical Survey.

The disease is first mentioned by Underwood in 1784, but he did not separate it distinctly from other forms of paralysis to which children are liable. In 1840, Heine directed particular attention to the disease, and in 1851 it was well described by Bartliez and Rilliet. It was more thoroughly investigated about the same time by Duchenne, and in 1864 many clinical facts were published in a thesis by Duchenne the younger, and in a monograph by Laborde. In 1878 Barlow of Manchester published the most important monograph to appear in this country until comparatively recently. Further contributions

were made to our knowledge by Seelig Müller.

The first cases of the Spinal atrophic paralysis of adults were reported by Meyer in 1868, and other cases were reported by Kussmaul, Cuming, and Kallopean, but the true nature of the disease was not recognised until 1872, when Duchenne pointed out its identity with the atrophic spinal paralysis of infants.

In the Goulstonian Lectures 1907, Buzzard discussed the infective origin of certain nervous affections which are characterised by paralysis of acute onset. W. Pasteur had in 1897 reported the case of an entire family of seven children who were all attacked with a mild febrile disorder. The illness was followed by motor paralysis in three of the children, by general muscular tremors in a fourth, and by muscular tremors and strabismus in a fifth. Fifteen or more epidemics had been recorded in various parts of the world, and several instances were cited by Buzzard which afforded cumulative evidence of the infective nature of the disease.

In 1905-4-5-6 there were several local epidemics of Anterior Poliomyelitis in Norway and Sweden, and about this period, and during the following years numerous extensive epidemics were reported in Europe, America, and Australia. The transmissibility of the disease began to be generally recognised. It was exhaustively investigated and much experimental work was undertaken, notably by Landsteiner and Poper, by Flexner and Lewis, and by many other workers, with the result that the disease is now regarded as due to the infection of the system by a virus which /

which is filterable, and that it is in every sense a true specific disease.

Batten compiled a list of recorded epidemics, and the number up to the end of 1910 was 76.

1881 - 5 2	1896 - 1900 16
1886 -90 5	1901 - 1905 12
1891 -95 6	1906 - 1910 35

The following is a list of the outbreaks of the disease which have been recorded in Great Britain.

Epidemics of Anterior Poliomyelitis in Great Britain.

1908 Upminster Essex.....8 cases.

1909 Bristol.....37

1910 Maryport.... 13

Workington..... 5

Carlisle.....34

North West Lancashire..... 8

Barrow-in-Furness.....37

Melton Mowbray.....83

Irthlingborough..... 4

Cerne Abbas.....16

Harvieston..... 5

1911 Cornwall and Devon.....132

Huntingdonshire..... 15

South Derbyshire..... 25

Hampshire..... 8

Stowmarket..... 32

In 1911 epidemics were also reported from Plymouth, Stonehouse, and Devonport, and from Cambridgeshire.

Section III. AETIOLOGY.

The Aetiology of the disease is obscure - necessarily so because of the mystery which still attaches to its specific virus. It is therefore desirable to review certain conditions with which the prevalence of the disease may be associated.

(a) Relation to diseases of domestic animals.

Domestic animals are known to suffer from different forms of acute paralysis, and instances of paraplegia have been recorded in horses and colts, bulls, cows, and heifers, in poultry, in cats, in dogs and in pigs, but negative results have always attended the injection of the virus into those animals as well as into goats, guinea pigs, and mice. In America an illness among colts showing a series of symptoms corresponding very closely to those of Anterior Poliomyelitis has been recorded. Hounsfield reported that several cases of paralysis occurred in the Stowmarket district among horses, and the horses had to be shot. At Penryn a case of "poke neck" occurred in a horse belonging to the father of one of the patients suffering from acute poliomyelitis, but the existence of such a disease is not generally recognised, and I am informed by the Veterinary Surgeon that it is unkown in Lanarkshire. In the Massachusetts epidemic it was found that in 87 out of 142 families affected by the disease, animals of some sort were kept, and in 34 of these there was some illness or paralysis. The proportion is not sufficiently high to justify any inference that disease in animals is related /

related to the occurrence of Anterior Poliomyelitis in man.

(b) Relation to bathing.

That Anterior Poliomyelitis follows bathing has been noticed, and used to explain many sporadic cases. In the Massachusetts epidemic careful enquiry was made into 150 out of 923 cases occurring in 1909. Of these 62 had been swimming before the onset of the illness in water more or less contaminated with sewage. In a discussion at a meeting of the Ulster Medical Society, it was stated that many of the cases occurred at sea-side places where children were wading more than usual owing to a hot dry season. It has been thought that a possible connection exists between the disease and swimming in polluted bath waters - the source of infection residing in the slime which forms at the bottom of the bath. But at Penryn, though children bathed regularly in sewage polluted water, it was ascertained that not one single patient had bathed or waded in the polluted area. Besides, a large number of cases occurs among children too young either to bathe or to wade.

(c) Relation to food.

The symptoms of the disease do not suggest as a rule an infection by way of the alimentary canal. It is true that the disease has been communicated by the ingestion of infected material and at Stowmarket many of the cases began with typhoid-like symptoms. No relation has been shown to exist to the consumption of any particular article of food. The course of the /

the epidemics bears no relation to the fly curve.

(d) Relation to dust.

Inhaled dust is irritating and sets up a nasopharyngitis; and rhinitis or nasopharyngeal catarrh is a very constant symptom in the early stages of the disease. It has been shown that the infection is able to travel by this path. Cases are very often distributed along main roads - the disease is associated with hot and dry summers and subsides in the rainy season. The recent prevalence of Anterior Poliomyelitis has been explained by the more general adoption of the fresh air mode of life - windows are left open, allowing of the entrance, not only of fresh air but also of dust. It has been asserted that the increased prevalance of the disease has been coincident with the development of motor traffic which dissipates dust to great distances. A similar explanation was offered in regard to the increased prevalence of Tetanus, but careful enquiry showed that it was not supported by facts. And as regards Anterior Poliomyelitis there is no confirmation of the assumption, for the first case may occur in a cottage far removed from dusty or public roads.

(e) Relation to biting insects.

The theory that Anterior Poliomyelitis is conveyed by biting insects requires careful consideration. The causation of an increasing number of the infectious diseases, both of man and of /

of animals has recently been proved to be due to micro organisms which are filterable, and in many cases ultra microscopic. The passage of certain diseases e.g. Yellow Fever, Rabies, Foot and Mouth Disease, Anterior Poliomyelitis, and Measles by means of filtrates has been successfully accomplished through a series of animals, and it may therefore confidently be affirmed that the cause of these diseases is not a toxin. In Yellow Fever, the blood contains the infective agent which is conveyed solely by the mosquito Stegomyia Calopus. In Dengue it is conveyed by the Culex fatigans, and in Sandfly Fever by Phlebotomus pappatasii. These three diseases resemble malaria in being conveyed by biting insects which remain incapable of passing the infection for a definite period of several days. In malaria this period is occupied by the sexual life of the Haemamoeba. The infective agents of the three diseases may be allied to the parasites of malaria, and may therefore be of protozoan nature. Many of the filterable viruses (including that of Anterior Poliomyelitis) are resistant to the action of glycerine which is rapidly fatal to bacteria, and are readily destroyed by substances which do not injure bacteria or by a short exposure to a temperature which is only lethal to non-sporing bacteria after a prolonged period. The seasonal prevalence of Anterior Poliomyelitis is quite consistent with the view that the disease is protozoan and conveyed by a biting insect. The age incidence may be compared with that of smallpox in cattle which is only inoculable into young animals. And the fact that

like Typhus Fever the infection of Anterior Poliomyelitis tends to cling to houses - for second cases in the same house though separated by a long interval of time have been frequently recorded as well as the mediate transmission of the disease would thus be intelligibly explained. While it must be admitted that hitherto there has not any evidence been obtained of the conveyance of Anterior Poliomyelitis in this manner, certain data may be given which suggest insects as carriers.

1. The Sporadic occurrence of cases.
2. The seasonal distribution of the disease shows the largest incidence during the warmer months when insects of all sorts are most prevalent.
3. It has not been proved that the disease spreads most where many children are regularly in close contact. It is the exception for several in a family to contract the disease.
4. The disease is more common in rural districts than in large cities, where the proportion of biting insects to the human population must be less than in country districts.

Further when we consider that there may be a possible relationship between the paralysis of animals and acute poliomyelitis in man, the study of biting insects becomes more important.

AGE.

The disease is most prevalent amongst young children, and most commonly affects children during the second and third years of life. Babies in arms are rarely affected. In the Massachusetts epidemic, of 440 cases under six years 304 occurred in children between the ages of one and four years. With advancing age the liability to attack steadily decreases.

The following Table taken from the Report of the Collective Investigation Committee on the New York epidemic of 1907 shows the age incidence in the various epidemics.

	Wickman's 1905	New York	Rutland	Göteborg
Up to 3 years	169	463	90	11
From 3 to 6 years	181	197	90	5
From 6 to 9 years	154	40	15	2
From 9 to 15 years	165	21	15	0
15 years and over.	199	8	15	2

	Stockholm	Smedjebackener	Shelby	Gloucester
Up to 3 years	34	30	10	16
From 3 to 6 years.	12	30	0	10
From 6 to 9 years.	1	20	2	3
From 9 to 15 years.	1	20	5	2
15 years and over.	5	0	3	0

SEX.

Males are more often attacked than females. In Minnesota in 1909 the ratio was 193 males to 139 females (100 M. to 72 F.). In the Massachusetts epidemic of 1909 363 males and 263 females were attacked, showing the same proportion (100 M. to 72 F.). In Minnesota the proportion of males and females affected was more nearly equal in the first decade of life, but after ten years of age males were affected in greater proportion than females.

SEASON.

Gowers aptly summarises the relation of Season in the phrase "Two thirds of the cases occur in the hottest third of the year." Holt and Bartlett found in an analysis of 35 epidemics in 33 it was definitely stated that the epidemic occurred during the hot season only, the most frequent months being July, August, September and October. The month of August is nearly always that in which the greatest number of cases occur. In the Southern Hemisphere the disease has occurred during the Months of March and April - months corresponding to September and October in the Northern Hemisphere in atmosperhic conditions.

In the New York epidemic of 1907 the date of onset of the cases was.

Jan.	5	April	3	July	133	Oct.	71
Feby.	3	May	10	Augt.	188	Nov.	20
March	4	June	59	Sept.	218	Decr.	4

Seventy /

Seventy five per cent occurred in the third quarter - seventeen per cent. in July, twentysix per cent. in August and thirty per cent. in September.

Section IV. PATHOLOGY.

Buzzard summarised the theories which were extant in 1907 concerning the pathogenesis of Poliomyelitis. The first theory:- that it is a primary degeneration of the anterior horn cells and that an entirely secondary change occurs in the interstitial tissues, was in his opinion rendered quite untenable by the post-mortem appearances of acute cases; and the second theory:- the vascular or primary thrombosis theory, he likewise considered negatived. He inclined to a third theory, that the disease is due to a specific micro-organism or its toxin, the toxin exerting a special influence on the neuroglial tissue, but whether it was haematogenous or lymphogenous was very uncertain. He ventured to predict that in spite of the fact that its bacteriology was quite an unkown quantity acute Poliomyelitis would be included before many years in the category of specific fevers. And his prediction has been verified. Two papers read before the Pathological Society in 1907 and the Medical Society of London 1908 demonstrate the nebulous condition of the real pathology of the disease at that time.

Miller, describing the post mortem appearances in a fatal case, stated that he obtained a culture of Staphylococcus pyogenes aureus from the soft swelling which was found in the cervical region of the cord. He discussed various theories of the causation of the disease and favoured the view that it was inflammatory.

Pasteur, Foulerton and McCormac related a case of acute Poliomyelitis with diplococcal inflammation of the spinal sac.

A /

A micrococcus was found in the spinal fluid withdrawn during life, and the fluid on being inoculated into a rabbit produced ascending motor paralysis - after a prolonged period of incubation.

In 1903-4-5-6 there were several local epidemics of acute Poliomyelitis in Norway and Sweden. Geirsvold in 1905 collected statistics of 437 cases with 69 deaths and attributed the infection to a diplococcus which he had been able to isolate from about a dozen cases. The bacteriology of the disease at this time was very inconsistent, but the consensus of opinion was towards its inclusion in the group of specific fevers. Other epidemics of considerable magnitude began to be recorded and stimulated the investigation of the Aetiology and causation of the disease. Landsteiner and Poper in 1909 first showed that acute Poliomyelitis could be transmitted to monkeys by inoculating a fragment of the spinal cord from a fatal case. Leiner and Wiesner showed that the virus could be transmitted from monkey to monkey. Krause and Meinicke produced paralysis in rabbits by inoculating them with the spleen, brain, and cerebro-spinal fluid of persons who had died of acute Poliomyelitis. Landsteiner and Levaditi inoculated a chimpanzee with acute Poliomyelitis, and from this chimpanzee two macaque monkeys were inoculated. The virus was found to pass through a Berkefeld filter.

Flexner and Lewis inoculated a long series of monkeys from two cases and found that the virus was in no way diminished in potency/

potency. They found also that the virus was invisible even by special illumination, it was not arrested by the finest filter, and it was not stainable. They found that the virus was destroyed at 55° C., that it resists drying and glycerinisation, and that it survives a temperature of -8° C. When dried over $H_2 SO_4$ it remains virulent for at least 14 days, and when dried over KOH for at least 24 days. When dried in vacuo (away from light) it is potent for four weeks. The virus is destroyed by disinfection with menthol preparations, by potassium permanganate (1 in 1000 for one hour at 37° C), by peroxide of hydrogen 1 in 100, by formaldehyde vapour, by 1 per cent. thymol (one hour), and by .5 per cent. phenol in 3 days at a low temperature. The reactions suggest that the infective agent is of a protozoan nature.

Flexner and Lewis inoculated a filtrate of the cord emulsion into broth containing 10 - 25 per cent. of monkey's serum, and on incubation the inoculated medium became slightly turbid, but it is not proved that a real culture was obtained. They failed to prepare a vaccine. Kraus, however, claims to have produced immunity in the monkey by subcutaneous injection of a vaccine consisting of virus weakened by treatment with phenol.

Landsteiner and Levaditi and Römer and Joseph have succeeded in actively immunising monkeys by injecting sensitised vaccine which consists of a mixture of virus and immune serum.

The incubation period has been found to be longer when a filtered virus is used. Levaditi and Landsteiner having found that /

that the cords of monkeys infected with a small dose of virus that had been passed through a filter were exceedingly infectious for other animals, concluded that Poliomyelitis is due to a microbe which possesses the power of actively multiplying and not to toxic product elaborated by this microbe and accumulated in the cord.

The infection like that of epidemic cerebro-spinal meningitis is transmitted through the nares, but it is only possible to cause infection after injury to the mucous membrane according to Levaditi and Landsteiner, but Liener and Wiesner claim to have shown that the virus will pass the uninjured mucous membrane.

Monkeys living in the same cage with infected monkeys do not contract the disease if the nasal mucous membrane is intact. There is evidence to show that there are slight differences between the virus of Flexner and that of Landsteiner, the former being more virulent. There may be a variation in the virus similar to that in the Typhoid and para-typhoid organism.

The disease has also been experimentally produced by inhalation, by introduction of the virus by means of the stomach tube, and the fact that the virus can penetrate the mucous membrane of the digestive tract raises the possibility of the transmissibility of the disease by milk. The virus has also been successfully injected intracerebrally, subdurally, intraperitoneally and intravenously. The virus may also be introduced into a nerve trunk. It probably travels by the lymphatic spaces of the nerves and reaches the spinal segments at the entrance /

entrance of the nerve roots. The infection may be prevented after such introduction, if the nerve be severed above the point of inoculation. In whatever way infection is brought about it is the central nervous system and especially the gray matter of the cord that the virus selects. How long the virus remains in the body it is difficult to say. It has been found in the spinal cord 33 days after the onset of the disease. On the other hand Leiner and Wiesner found it had disappeared from the cord six days after the onset of paralysis. The virus may be obtained from the mucous membrane of the nose of a monkey which has suffered from the disease six months after the date of infection.

It has been found that one attack of the disease confers immunity. Netter found that the serum of patients who had suffered from infantile paralysis possessed neutralising properties with respect to the virus of poliomyelitis, and so furnished evidence of the identity of epidemic and sporadic poliomyelitis. Vipond (Montreal) obtained a positive Widal reaction in a large proportion of his cases and regards the disease as analogous to enteric fever. This observation has not been confirmed, but it may be mentioned that intestinal symptoms occur in 90 per cent. of all cases.

It has quite lately been considered whether acute Poliomyelitis is in reality a separate entity. It has been suggested that it may be related to cerebro-spinal meningitis in the same way as post-diphtheritic paralysis to diptheria, but that this view /

view is correct is exceedingly improbable.

As there is great similarity between the virus of acute Poliomyelitis and the virus of rabies as shown in (a) the progress of the virus along the nerve fibres, (b) affinity for the nerve centres, (c) reaction to chemical and physical agents, and (d) filterability, and as the character of the lesions found in the central nervous system is very similar in the two diseases, it has been suggested that the disease is of animal origin, i.e. casually related to disease in an animal. It is more likely however that the infective agent in both diseases is protozoal.

The macroscopic changes in the central nervous system are slight, and consist only of some hyperaemia and oedema of the meninges and grey matter. An inconstant feature is haemorrhage into the grey matter of the spinal cord. Hyperaemia is found at all levels of the cord. In the cord the grey matter appears either decidedly red or brownish red, and the line of demarcation between it and the white tracts has disappeared so that they fuse into each other.

Microscopic appearances.

1. The vessels play a very important part. Arteries, veins, and capillaries are markedly congested, and in some cases excessively distended. Haemorrhages occur in some cases.

2. In addition to the hyperaemia there is a perivascular infiltration which is adventitial and composed of small lymphocytes derived from the fixed mesoblastic cells of the adventitia.

3. Dependent upon these changes interstitial changes occur in the /

the grey matter both diffuse and local, the diffusely infiltrated cells appearing to come from the vessels both peripheral and central. The intensity of infiltration varies in different cases.

In addition to this diffuse infiltration there are collections of cells in the grey matter. These collections of cells or foci always lie in proximity to the branches of some vessel, generally a vein or capillary. The majority of cells in the foci are polynuclear Leucocytes.

4. Changes in the Ganglion cells are of secondary importance, though an important factor. These cells show different grades of destruction e.g. granular degeneration and vacuolation up to complete disintegration of both cytoplasm and nucleus.

Section V. INCUBATION PERIOD.

What the incubation period is has not yet been definitely settled, but with regard to this, I think case No 1 is of exceptional interest. The boy is in contact with a case of acute Poliomyelitis on the Saturday, and on the Wednesday following he himself becomes ill and developes Poliomyelitis. These circumstances would lead one to put the incubation period down at four days. Wickman in an exhaustive report on the Swedish epidemic of 1905 showed that the incubation period lies between one and four days.

In investigating the Harvieston epidemic of 1910, Doctors Currie and Bramwell came to the conclusion that the incubation period is four days or less. On the Harvieston Estate are four houses A.B.C.D.

A family consists of		2	children	A.A., B.A.
B	" "	4	"	A.B., B.B., C.B., D.B.
C	" "	4	"	All over 8.
D	" "	2	"	A.D., B.D.

Patient.	Date of Attack.
B.B.	Sept. 12
A.B.	" 16
D.B.	" 18
B.A.	" 20
A.A.	" 24

The children of A. family did not visit the B. family after /

after the child B.B. was taken ill. The mother of the A. family nursed the B. children. The infection was therefore probably carried by her.

From a consideration of the epidemic in Westmoreland Dr. W.E. Henderson has come to the same conclusion that 4 days is the probable incubation period. Shorter and much longer periods have been recorded.

Infectivity.

The infectivity of the disease is not great, being far less than scarlet fever or diptheria. Of persons known to have been exposed to diptheria, scarlet fever, and poliomyelitis, the percentages contracting the disease were as follows:-

Scarlet Fever	22 per cent.
Diptheria	17 per cent.
Poliomyelitis.	6 per cent.

Both incubation period and infectivity depend upon the mode of transmission of infection. Scarlet Fever and Diptheria are diseases due to bacterial infection. But, as I have already pointed out, in Poliomyelitis the virus is probably of protozoan nature.

TRANSMISSION /

Section VI. TRANSMISSION.

The disease may be transmitted from person to person, and I think case I may be taken as an example of this.

The disease may also be carried from one child to another by persons not themselves suffering with the disease, as exemplified in the Harvieston epidemic already referred to, where the mother of the A family having nursed the B. children was the probable "carrier." Wickman having studied the abortive type of the disease is of opinion that the mild and abortive cases which are likely to be unrecognised are the most usual carriers. The Harvieston epidemic also illustrated the well-known fact that two or more members of a family may be affected. Very often the cases in one family are at the same stage - pointing to a common source of infection.

In the Swedish epidemic of 1905 the following table gives the number of multiple cases.

No. of cases in each house	1	2	3	4	5	6
No. of houses.	627	97	39	14	7	1

In the New York epidemic the figures were as follows:-

No. of cases in each house.	1	2	3
No. of houses.	100	18	5.

The degree of infectivity does not necessarily correspond to the degree of virulence. The virulence of the infection can be measured not only by the mortality of the disease, but also in many instances by the proportion of adults affected, and by the /

the period of incubation.

School Infection.

Wickman in the Swedish epidemic has shown that the disease tends to travel along the railways and great thoroughfares, and he brings forward evidence that the disease may be spread by schools. He cites four instances, the Trästena, the Gärdsby, the Sirko and the Tingsryd epidemics.

At Trästena there were 49 cases - 23 of the abortive type and 26 suffered from paralysis. Of those who took ill four children belonged to the school teacher, 25 to families of which the first infected child attended the same school, 12 to families who had healthy children attending school though the infected children were not at school, and four to families that had only indirect communication with the school. The remaining four cases had no ascertained connection with the school. There were 102 houses in the village of Trästena with 132 families and 19 houses were affected. There was:

1 case in 6 houses.
2 cases " 3 "
3 " " 5 "
4 " " 3 "
5 " " 2 "

With regard to an outbreak of Poliomyelitis at Créteil, Netter came to a similar conclusion. Seven children residing far apart, and whose parents did not visit each other, were attacked /

attacked. Three of the children went to the same school. Two of the others (not at school) had a brother and cousin respectively attending the school. Netter concluded that the infection was spread at the school by the three scholars who had contracted the disease, and by the two who without having the disease themselves infected their brothers and cousins.

In the Westmoreland epidemic already referred to, Dr. Henderson cites cases attending the same school. On the other hand there is evidence against the school being a factor in the spread of the infection. Many of the children who are attacked with the disease are not of school age, 75% being under four years. In institutions it is rare for the disease to become epidemic. Cases of Poliomyelitis can be and are treated without danger in general hospitals.

Nevertheless during an epidemic precautionary measures should be adopted. All contacts should be quarantined for a period of at least 14 days before returning to school, and no child who has had the disease should be allowed to return to school before a month has elapsed from the onset of the illness.

Notification.

The disease is not at present notifiable, though several Local Authorities made it so last year. It should be made notifiable generally. That it is an extremely serious disease - crippling for life many of its victims - no one will deny. Notification would lead the public to recognise that the disease /

disease is infective and would tend to lessen the spread by contact. Cases could also be investigated in a manner not possible at present.

Section VII. IMMUNITY.

Flexner and Lewis and other observers have found that animals which have had acute poliomyelitis and recovered are immune to another dose of the virus.

Clinically there is every reason to believe that persons who have had the disease and survived are also immune to a second attack.

Levaditi and Landsteiner attempted to immunise monkeys against poliomyelitis by the same method as is used for producing immunity against rabies, but in this they were only partially successful. Flexner and Lewis by giving injections of diluted virus were able to immunise monkeys, and they further found the immunity principle to exist both in blood and in cerebro-spinal fluid soon after an attack of Poliomyelitis. After one or two months the cerebro spinal fluid lost its immunity principles, but it remained in the blood for a very much longer time. Attempts have been made, but without success, to immunize animals by vaccination with the virus inactivated by heat. Kraus has found that a saline emulsion of brain and spinal cord treated with 1 - 1½ % of Carbolic Acid is not infective when injected subcutaneously. Ten C. C. of virus thus sterilized and injected subcutaneously protects animals against a subsequent subdural injection. The serum of an animal immunized against Poliomyelitis possesses the capacity of inactivating the virus in vitro. This capacity has been applied as a test for the disease /

disease. If there has been an attack the serum of the patient possesses the capacity of neutralising the virus - in this way abortive cases may be identified.

As the epidemic in Aarau was normally in 1918 it was 28.1 per cent. As this is above the usual it is that mild cases were not recognised. In the Western epidemic the mortality was 15.3 per cent.

The average mortality by age in the Massachusetts 1910 for 601 cases was 10.31 per cent, as shown in the table.

	Cases	Deaths	%
1 year.....	117	13	
2 - 10 years.....	374	37	
Over 10 years.....	110	14	
Totals	601	62	
Average mortality			

Section VIII. MORTALITY.

Records of mortality in the various epidemics vary. It was 12 per cent. in Sweden, 14 per cent. in Norway, 5 per cent. in New York, 13.16 per cent in Upper Austria and 5 per cent. in the small epidemics observed by Netter in Paris and its suburbs. The mortality is higher for children of older age than it is for young children.

In the epidemic in Devon and Cornwall in 1911 the mortality was 22.1 per cent. As this is above the usual, it is probable that mild cases were not recognised. In the Westmoreland epidemic the mortality was 16.3 per cent.

The average mortality by age in the Massachusetts epidemic 1910 for 601 cases was 10.31 per cent. as shown in the following table.

	Cases	Deaths	Mortality (per cent)
1 year.....	117	11	9.40
2 - 10 years.....	372	37	9.94
Over 10 years.....	112	14	12.5
Totals	601	62	
Average mortality			10.31

Section IX. SYMPTOMATOLOGY.

The disease presents a great variety of symptoms, and as the paralysis is the most constant feature of the disease, diagnosis before that appears is attended with many difficulties, as none of the other symptoms are pathognomonic. The pre-paralytic stage may present a diversity of striking symptoms or may be almost devoid of these. Paralysis generally appears shortly after the preliminary manifestations, though cases are recorded of children going to bed apparently quite well and being found in the morning suffering from paralysis.

Lucas says noteworthy prodromal symptoms are irritability, restlessness, pain in the spine or extremities, apathy.

Important symptoms during the acute stage are, fever (100° to 106°) - duration 2 to 7 days, vomiting (25% in New York cases), restlessness, apathy, rigidity of neck, frontal headache, delirium, stupor, convulsions, photophobia, dysphagia, sluggish pupils, general pain, absence of deep reflexes.

Wickman believes the condition of the patellar reflex to be of great importance and states :

1. The patellar reflex disappears. That is the rule. Loss of the patellar reflex may be the only recognisable feature of the disease.

2. There may be a preliminary increase followed by loss of the patellar reflex.

3. When the crus or bulb is involved an increase of the patellar /

patellar reflex in the otherwise normal leg may be observed.

4. In connection with paralysis and loss of reflex in one leg there may be an exaggeration of the reflex in the other apparently normal leg.

5. Increase of the patellar reflex in a paretic and clearly atrophied leg.

Skin eruptions are sometimes met with. They occurred in 61 out of 742 cases in the New York epidemic. The eruption was papular. In 13 out of 153 cases in the epidemic in Devon and Cornwall there was a rash noticed. Some observers have drawn attention to the prevalence of herpes in epidemic Poliomyelitis, but this does not seem to be a constant feature.

Sweating is a characteristic early symptom, and this symptom was very prominent in case I. Professor Müller puts profuse sweating down as one of three cardinal symptoms, from which it is possible to make a correct diagnosis before the appearance of paralysis.

Pain and tenderness.

This is also a marked symptom of the disease. In some cases the pain is intense. In the New York epidemic in some cases it was said to be excruciating. It occurred most often in the lower extremities, next in frequency in the spine and trunk, and less frequently in the upper extremities and neck.

Meningeal Symptoms.

At the onset of acute Poliomyelitis it is sometimes difficult to /

to distinguish it from cerebro-spinal meningitis owing to the presence of pain, stiffness, and rigidity of the neck. In Poliomyelitis the head as a rule is not so markedly retracted as in cerebro spinal meningitis. Where there is a modified Kernig's sign the diagnosis is rendered more difficult

[Kernig's sign. The patient is placed so that the hip joint is semiflexed. Retaining the hip joint in that position, the knee joint is extended passively. If a contraction of the hamstrings results Kernig's sign has been obtained.]

As already stated paralysis is the most characteristic feature of the disease. The paralysis is associated at an early date with atrophy, with changes in the electrical reactions, and with a loss of reflex activity in the affected parts. As a rule the paralysis is of the flaccid type, but in the New York epidemic rigid paralysis was noted in 38 cases. The time that elapses before paralysis develops varies. In 462 cases paralysis was developed within a week of the onset, and in 518 cases within the first fortnight.

The left side is more often affected than the right, and both lower extremities and both upper are more frequently affected in combination than either one alone in combination.

Of the 462 cases referred to above:-

In 370 the lower extremities were affected.

" 83 the upper " " "

" 7 all " " "

" 2 the neck was affected.

The /

The bladder was affected 87 times and the rectum 52.

Both legs alone were affected 168 times.

The speech is sometimes involved. Such speech disturbances belong to the Dysarthrias and indicate bulbar localisation or cerebral involvment.

Regarding the order and time of recovery, if any, of paralysed parts, it was found in the New York epidemic:-

Improvement within the first month occurred in 137 cases

" " " two months " " 101 "

" " " 3 or more months " 119 "

There was a marked regression of symptoms in 47.6 per cent, and a complete disappearance of paralysis in 5.3 per cent.

Types of the Disease.

Wickman has described eight types of the disease.

1. Spinal Poliomyelitic type. In it there is a localised paralysis of one or more limbs. It is the most common type.

2. An ascending paralysis, often rapidly fatal owing to the involvment of the respiratory centres. The paralysis usually begins in the lower extremities and gradually ascends. Occasionally it happens that the paralysis is descending instead of ascending.

3. An acute ataxia. In this type the symptoms are due to lesions of the cerebellum.

4. Bulbar type. Here there is paralysis of the parts supplied by the cranial nerves which have their nuclei in the medulla or pons.

5. Hemiplegia. This type is due to the changes in the cortex of the brain.

6. An acute mental defect.

7. Symptoms of meningitis.

8. Polyneuritic type. In this type pain , especially on movement, is the most marked feature.

In addition there are abortive cases with or without paralysis in which the symptoms are only transient. These can only be identified when they occur in direct association with a marked case of the disease.

Section X. DIFFERENTIAL DIAGNOSIS.

The characteristic flaccid paralysis differentiates the disease from all common nerve affections of childhood (except perhaps myelitis), but the occurrence of Poliomyelitis in adults may occasionally require some care in making the diagnosis.

The characteristic symptoms on which Wickman lays stress are: (1) Drowsiness, (2) Pain and tenderness, (3) Stiffness of the neck (4) Profuse perspiration.

The following are some of the diseases from which acute Poliomyelitis may be differentiated.

1. Haemato-myelia.

Haemorrhage into the grey substance of the cord may produce a sudden paralysis followed by muscular atrophy, loss of reflex excitability, with reaction of degeneration in the related parts. The initial fever is absent, the invasion is more sudden, disturbances of sensibility, paralysis of the sphincters and bed sores occur.

2. Cerebral Hemiplegia.

In the Cerebral Hemiplegia occurring in children there is a different commencement, the facial nerve is implicated, there is no muscular atrophy, there is increased irritability of tendons, and the electrical irritability is preserved.

3. Amyotrophic Lateral Sclerosis.

This disease begins in the upper extremities which become more /

more or less paralysed and wasted, while the antagonists of the paralysed muscles become rigid and contracted. The arm is held tightly to the body, the forearm is flexed and pronated, and the hands and fingers are strongly flexed, while the lower extremities are affected with spasmodic paralysis. The initial fever is absent.

4. Landry's Paralysis.

In Landry's paralysis there is sudden onset, but the progress is less rapid, the constitutional disturbance is not so severe, and there is little or no febrile disturbance. The flaccid paralysis is of more uniform distribution. The muscles do not waste and do not show electrical changes, The cranial nerves are rarely affected. Severe pains are rare, but paraesthesiae are not uncommon. There is no loss of cutaneous sensibility. Deep reflexes are absent. The sphincters are unaffected. The prognosis as to life is not so good, but muscular recovery is likely to be more complete.

5. Acute Toxic Polyneuritis.

The onset and progress is more gradual , often without any constitutional disturbance or feverishness. The peripheral muscles are more affected than the proximal groups. The cranial nerves are not commonly affected. Paraesthesiae are very common, and there is tenderness on pressure. Cutaneous sensibility is affected generally (to) some degree. All the reflexes are abolished./

abolished. The sphincters are unaffected. The prognosis is good as to life, and as to recovery of muscular power.

6. Acute Ascending Myelitis.

In this disease the onset is sudden and attended with very severe constitutional disturbance. Ascending paralysis involving successive segments of the spinal cord develops. Pain in the back is common, and there is marked loss of cutaneous sensibility ascending with the paralysis. The reflexes are lost as the disease progresses. The sphincters are affected early. The prognosis in this affection is exceedingly bad.

7. Progressive Muscular Atrophy.

This disease usually begins in one or both of the upper extremities with atrophy of the thenar and hypothenar eminences, the interossei, the forearm muscles, and those of the shoulder. Shortening of the extensors of the wrist produces the claw-like hand. Extension is extremely gradual, and years may pass before both arms, both legs, intercostals or diaphragm are attacked. Twitching of the muscles (fibrillation) is a common symptom. In cases of rapid wasting and paralysis there may be reaction of degeneration.

8. Paralysis following obstetric operations.

This is generally the result of pressure. The paralysis is similar to that occurring in the upper extremity of a person who falls asleep with the arm hanging over the back of the chair. In most cases the paralysis passes off in a few days.

9. Beri-beri.

In this disease there are usually catarrhal symptoms to begin with, followed by pains and weakness in the limbs, diminished sensibility in the legs, and paraesthesiae. Loss of power in the limbs may progress so that the patient cannot walk or move the arm. Atrophy of muscles is present and may extend to the muscles of the face. Associated with the atrophy is a good deal of pain. Heart troubles and oedema are conspicuous.

10. Epidemic Cerebro-spinal Meningitis.

Here there is rigidity of the limbs, Kernig's sign is present and extensor plantar reflex. There is early optic neuritis. On lumbar puncture an excess of fluid is found, and in it there is an excess of albumen and it is turbid. Paralysis is rare, with the exception of transitory paralysis of eye muscles. When the patient survives more than a few days, the fever becomes intermittent or remittent. Headache is so severe that patients often scream. A rash is common, hence the name "Spotted Fever." In a blood count the number of leucocytes is high, and there is a relative increase of polymorphonuclear leucocytes. (In Poliomyelitis there is leucopenia). Epidemic cerebro-spinal meningitis generally occurs in winter and spring. Bacteriological investigation demonstrates the specific organism - meningococcus. In addition there is an absence of dextrose - the normal 0.05 % being found in acute Poliomyelitis.

Graham /

Graham Forbes gives an analysis of 33 specimens of cerebro-spinal fluid obtained from 30 cases of acute Poliomyelitis. The time at which lumbar puncture was performed varied from 3 days to 6 weeks after the onset of illness.

In 24 specimens the fluid was clear and free from clot.

In 8 specimens there were traces of blood and clot, the amount depending on blood contamination.

In 1 specimen the fluid was clear on withdrawal, but a fine web of clot appeared on standing.

On boiling with a few drops of Acetic Acid:-

7 specimens gave "normal faint haze."

3 specimens gave definite cloud.

5 specimens gave fine precipitate.

6 specimens gave heavy precipitate.

Albumen was present in 24 cases. The amount varied usually with the duration of illness, being more marked during the first three weeks. All specimens yielded the normal amount of sugar 0.05 %. Slight excess of lymphocytes (1 - 5 per c.m.m.) was noted, but in 6 specimens a definite increase, and in 6 specimens very few cells or none, were seen. There were very few polymorphonuclears.

On bacteriological examination no organisms were found in stained films. 23 cultures were sterile and 7 showed obvious contamination.

The following table shows the difference in the spinal fluid /

fluid in acute Poliomyelitis and cerebro spinal meningitis.

Spinal Fluid.

Anterior Poliomyelitis:	Cerebro-spinal Memingitis:
Clear.	Marked turbidity.
Free from clot.	Coarse purulent clot.
Albumen small in quantity.	Great excess albumen.
Dextrose in normal amount.	Absence of dextrose.
Lymphocytes slightly in excess.	
Very few polymorphonuclears.	Copious deposit of polymorphonuclears.
Sterile.	Diplococcus Meningitidis.

11. Influenza.

Catarrhal conditions of the respiratory tract are common in Influenza, and rare in Poliomyelitis. Influenza is also more common in winter.

12. Tubercular Meningitis.

This disease as a rule has a more gradual onset. Headache is less violent, and the course of the disease more regular. It is practically speaking always fatal.

13. Acute Meningitis.

Here there is generally a known cause - suppurative disease of ear or nose, injury or acute illness. Spinal sumptoms are less common.

PROGNOSIS.

Section XI.

As regards life, the prognosis is good, but as regards ultimate recovery it is bad. Many of the individuals attacked are permanently crippled.

Still it is not invariably such a grave disease as is generally supposed. Many cases recover completely. In the New York epidemic the paralysis disappeared in 40 cases or 5.3 per cent., while there was almost complete disappearance of the paralysis in 13 cases or 1.8 per cent., making a satisfactory recovery in 7.1 per cent. of all cases.

In the Massachusetts epidemic of 1910 it is stated that complete recovery occurred in something over 25 per cent. of cases examined at the end of four years.

TREATMENT.

Section XII.

This may be divided into three stages. -

1. Acute.

The patient should be kept at rest in bed in the most comfortable position. Theoretically the prone position is the best.

The bowels should be opened by calomel or castor oil followed by enemata.

If fever is present, Pot Citrat gr. V., Sp. Aeth. Nit mx Aq. Chlorof. ad3ʃ may be given every 4 or every 6 hours.

If the temperature rises above 103°F., sponging with tepid water and alcohol may be tried. Antipyretics such as aspirin, phenacetin, antipyrin may be given in doses varying with the age of the patient.

If there is great pain the limbs should be carefully supported - the patient lying on a water bed. Hot applications, mustard poultices or leaves may be applied to the spine. Leeches also may be applied or dry cupping may be tried. Lumbar puncture is said to relieve pain, and may be performed especially in cases where symptoms of cortical pressure are evident. Morphia may have to be used. The diet should be fluid for the first few days. Care must be taken in the feeding, and if swallowing is difficult, a nasal tube should be employed.

Extensive paralysis as a rule passes off in a few days leaving generally a residual permanent paralysis. From the time paralysis /

paralysis occurs a prolonged hot bath proves a most successful element in the treatment.

It consists of laying the patient in a bath of water at least twelve inches deep, heated to 100° F., and letting him lie in it from 10 to 15 minutes every 4 hours. The temperature of the water in the bath should be increased by the addition of still hotter water till it stands at from 102° to 104° F. It should not be allowed to cool off several degrees, else a feeling of chill will be produced. The patient on being lifted out of the bath should be rolled in a blanket and dried in bed.

Urotropin in large doses is said by certain observers to have a beneficial effect. Netter, Gendron, and Touraine report four cases of acute poliomyelitis treated by lumbar injections of serum obtained from patients recovered from the disease. In all the cases paralysis had appeared before the treatment was commenced. One case died and the other three improved - two of them very markedly. It is suggested that this treatment should be started early and persevered with.

2. Stage of Recovery.

The patient should be carefully guarded against chill. Care must be taken to have the affected limbs kept warm. Unless contra-indicated by pain or fever, massage may be begun 10 or 14 days after the onset, then passive and resisted movements should be prescribed. Electricity may also be employed. Galvanism of the affected /

affected muscles may be tried for a few minutes daily, and later the faradic current may be employed. In suitable cases electric treatment may be continued for two years. In this stage the best drug is Strychnine. It should be given at first in doses of 1/100 grain, but this dose should gradually be increased.

3. Later Stage.

To prevent deformity, mechanical supports may now be employed. Continuous stretching of the paralysed muscles must be avoided as nothing injures them so much. Surgical treatment should be employed to correct deformities. They may be removed except in cases of very long standing by simple tenotomies and fasciotomies, and the use of moderate corrective force under ether.

In cases of loose or wobbly joints, the required stiffness may be furnished by surgical means such as tendon shortening and tendon transference, silk ligaments or arthrodesis; of these arthrodesis at the ankle and tarsus are the most useful. Tendon transference gives better results if deformities are first corrected, and both this operation and arthrodesis are best postponed until the age of nine or ten years.

BIBLIOGRAPHY.

American Journal of the Medical Sciences, May, December 1908.

British Medical Journal, February 5th 1910, March 18th 1911, November 4th, December 2nd, 9th, 30th 1911.

Therap. Gazet., September 1909.

Brain, Vol. 32, p. 285, Vol. 34, pt. 1.

Boston Medical & Surgical Journal, August 11th 1910.

Journal American Medical Association November 1909, December 4th and 18th 1909, January 1st, February 12th, April 2nd, May 28th 1910, February 18th and 25th, March 4th, November 18th 1911.

Edinburgh Medical Journal, October 1911.

Bulletins of the Massachusetts State Board of Health 1911.

Report of the Collective Investigation Committee on the New York Epidemic of 1907 - Nervous & Mental Disease Monograph Series No. 6.

Local Government Board Reports on Public Health and medical subjects, New Series No. 61.

Medical Officers of Schools Association - Poliomyelitis in relation to the spread of infection by schools, 1911.

Wickman, - Die Akute Poliomyelitis.

Ross, - Diseases of the Nervous System.

Lancet, September 6th, November 18th, 1911; February 17th 1912.

International Clinics Series XXI., Vol. 1, p. 55.

Public Health, March 1912.

The abstracts contained in the Lancet, British Medical Journal, and Brain, also the Review of Neurology and Psychiatry 1907-1911 have likewise been consulted.