

EPIDEMIC POLIOMYELITIS IN WESTMORLAND,*

SUMMER AND AUTUMN, 1911.

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AMID much that is uncertain as to the causal agent and the method of spread of poliomyelitis, there are certain undoubted facts confronting the health officer, calculated to make him face new problems and think in new categories.

In the matter of poliomyelitis most of us are at the stage when we are "marshalling our facts and court-martialing our fictions."

What are the facts?

1. Poliomyelitis is an acute, infective disease, which appears in epidemic form. There can, I think, be no doubt about this.

In his comprehensive and exceedingly able Paper on the Epidemiology of Poliomyelitis in the current number of "Brain," Batten† has collected the records of over 70 outbreaks of poliomyelitis, involving 17,500 cases. These records cover a period of 29 years, from 1881—1910, and embrace many lands. The first records of outbreaks come from Sweden and Norway, but every European country has suffered, while the United States of America have been visited by very severe epidemics indeed. Even Australia, Cuba, and the Island of Nauru in Melanesia have been invaded.

2. The clinical picture of poliomyelitis is changing. It is changing for the worse.

Until quite recently the text-books taught us that the characteristic victim of poliomyelitis was a child of two or three years of age who, put to bed, perhaps fretful and feverish, was found next morning with a flaccid paralysis of a lower limb. And so they called the disease "infantile paralysis." But this term must now be outlawed from medical nomenclature. Adolescents and adults are now being affected, and they do not always survive.

Still has shown that in some cases of poliomyelitis—

"there is a profound constitutional disturbance suggestive of some acute infective disorder. The facial palsy which some cases exhibit proves that parts at least as high as the pons may be affected, . . . and if the disease may produce inflammation of the grey matter at this level, there seems to be no reason why it should not produce it also in the grey matter of the cortex." ‡

* Read before the North Western Branch of the Society of Medical Officers of Health, December 15th, 1911.

† See also "Public Health" No. 2, vol. xxv, p. 51.

‡ Common Disorders and Diseases of Childhood. G. F. Still. Oxford Medical Publications, pp. 596-7.

Batten and John Thomson* share the same view. This being so, Batten prefers the term "polio-encephalo-myelitis," or as a neater and more portable term "epidemic paralysis."

To him we are indebted for a valuable analysis of the different types of polio-encephalo-myelitis. These are:—

1. A localized paralysis of one or more limbs.
2. An ascending paralysis, often rapidly fatal owing to involvement of the respiratory centres.
3. An acute ataxia.
4. A paralysis of one or more cranial nerves.
5. Hemiplegia.
6. Symptoms of meningitis.
7. An acute mental defect.
8. A type of case in which pain, especially on movement, is the most marked feature—so-called neuritis.

Most of these types are exemplified in the Westmorland epidemic.

3. The Virus. Landsteiner, Popper, Levaditi, Flexner, Lewis and Gordon have shown that the disease can be transmitted from man to monkeys by inoculation, and from monkey to monkey; that the virus is not killed by glycerination, and that it will pass the finest filter. The organism has not been cultivated in vitro, has not yet been stained, and has not come within the range of the most powerful microscope.

The virus can be obtained from the mucous membrane of the nose of a monkey which has suffered from the disease, and that even six months after the date of infection.

It is only possible to infect an animal by way of the nasal mucous membrane *after injury to this membrane*.

The virus is most readily obtained by emulsifying the brain and spinal cord of a human being or of a monkey dead of the disease.

Successful inoculation of the virus may be made into the brain, intraperitoneally, intravenously, or by application to the injured nasal mucous membrane.

THE WESTMORLAND EPIDEMIC.

(Cases arranged in chronological order).

In this epidemic the first case occurred on July 11th, 1911,—the last case on October 14th, 1911.

The disease first made its appearance in epidemic form in July, 1911, at Staveley, a village of some 650 inhabitants, situated on the main Kendal-to-Windermere Road.

* Clinical Examination and Treatment of Sick Children, John Thomson. Green and Sons, 1908.

Through the kindness of Dr. Innes Dick, of Staveley, I am enabled to furnish the following account of the beginning of the epidemic, and as the first cases are typical of many more, I quote Dr. Dick's reports in full:—

CASE 1, Staveley, *July 12th*, 1911.—Dr. Dick, of Staveley, was called in to see F. B., male, aged 10, of Main Street, Staveley. The following history was elicited:—Child had measles in April, 1911, and never seemed to recover fully.

July 11th, 1911:—Went to bed with a severe headache; vomited same evening.

July 12th, 1911:—Vomited whenever he swallowed anything. The following symptoms were found:—Temperature 103; pulse 120. Great pain complained of in back of head and in neck. Child lying in bed on his left side with his neck markedly retracted, his limbs flexed at elbows, hips and knees. He screamed when he was moved about. Pupils normal. No ear trouble. During that night he was delirious, and on the following day the symptoms were greatly aggravated. There was a left-sided facial and upper arm paralysis, with great stiffness at the neck. Breathing was stertorous.

July 14th, 1911:—The child was better to some extent and the temperature was falling.

July 15th, 1911:—Temperature normal, and the condition slowly improved, save paralysis.

September 3rd, 1911:—At the kind invitation of Dr. Dick we examined the child together, and found left facial paralysis and a certain amount of weakness of the shoulder girdle muscles. Left arm raised with difficulty above the head.

(The mother states that the temperament of the child is quite altered. He is irritable and disobedient.)

December 13th, 1911:—Facial and upper arm palsy still present. Considerable improvement has taken place.

CASE 2, Staveley, *July 12th*, 1911:—A boy, I. L., aged 10, living some 20 yards away from Case 1, complained of pain in the back of the neck and down the spine and in the legs. Mother noticed that his face got very red. The following were the symptoms found: All spine tender; normal temperature; full bounding pulse. Symptoms looked like those of heat stroke—the weather at this time was excessively hot. The child was put to bed.

July 13th, 1911:—He could not use his right leg. There was complete motor paralysis of the right leg; loss of knee jerk. He had not emptied his bladder for 24 hours. The paralysis gradually improved.

September 3rd:—On examination we found that there was considerable disability in the right leg, the boy dragging the limb. There was no wasting of the muscles.

December 13th, 1911:—Certain amount of paralysis still persists.

CASE 3, Staveley, *July 11th*, 1911:—Dr. Dick called in to see an infant aged 3 months, male, residing in Staveley, because mother thought that its right arm was dislocated at the shoulder. On examination, complete motor paralysis of the right arm was found.

September 3rd, 1911:—No improvement.

CASE 4, Staveley, *August 13th*, 1911:—Dr. Dick was called in to see an infant A. B., male, aged 11½ months, residing at Kentmere, 4 miles from the other cases. The following history was elicited: Mother stated that child had been cross and irritable. She thought it was a sign of teething. Symptoms observed: Child fretful and irritable, gums inflamed, no fever. Continued so until

August 20th, 1911:—On this day it seemed to take little notice of anyone, though quite conscious and taking its food.

August 23rd, 1911:—Child unconscious. Retraction of head, stiffness of limbs, pupils widely dilated, non re-active to light.

August 24th, 1911:—Condition same, pupils non re-active to light. Breathing stertorous. Child unconscious. Pulse was ever slow. Head retraction marked. The left side of the body seemed to be more rigid than the right, though the mother stated that when lifting the child the whole body seemed more rigid than usual.

August 25th, 1911:—Same, but that night had a general convulsion. Temperature 104. No temperature until convulsion.

August 26th, 1911:—Much worse. Tempr. 102.

August 27th, 1911:—Child died. Certified posterior basal meningitis.

CASE 5, Kendal, *August 16th*, 1911:—Br. Brumwell, of Kendal, attended a male aged one year. Paralysis of lower limbs. Much constitutional disturbance.

CASE 6, Pooley Br., *August 17th*, 1911:—Dr. Inglis, of Penrith, attended male infant aged 9 months. Symptoms resembling Case 3. Died August 21st, 1911. Certified posterior basal meningitis.

CASE 7, Kendal, *August 22nd*, 1911:—Dr. McCallum, of Kendal, attended female aged 28. Type, symptoms of meningitis with acute ataxia. Partial and tedious recovery.

CASE 8, Old Hall, *August 24th*, 1911:—Dr. Fuller, of Milnthorpe, attended male aged 11 years. Type, lower limb paralysis. Paralysis persists. Improvement.

CASE 9, Underbarrow, *August 26th*, 1911:—Dr. McCallum, of Kendal, sent for through the night to see a girl 8 years of age, who was screaming with pain in her head. Symptoms very like cerebro-spinal meningitis. There was a blotchy purpuric rash on trunk and arms.

August 30th, 1911:—I examined this case with Dr. McCallum. Child lying placidly in bed, quite conscious. On attempting passive movements of limbs child cried piteously. Head retraction: nystagmus. Motor paralysis of both lower limbs. Temperature 100. Lumbar puncture. No meningococci—L.G.B. Lab.

CASE 10, Underbarrow, *August 30th*, 1911:—In same house as Case 9, male (a twin), aged 10 months. developed an abortive attack.

CASES 11 and 12, Natland, *August 26th*, 1911:—Dr. Sturridge, of Kendal, attended two cases at Natland (2 miles south of Kendal), a male aged 9 and a female aged 12. Dr. Sturridge reports "both were taken ill within a few days of one another,

both with cerebral as well as spinal trouble ; both had sore throat and headache. One, the girl, is still paralysed."

CASE 13, Kendal, *August 26th*, 1911 :—Dr. McCallum attended male aged 5 years. Type, acute taxia ; fell during play ; had to be carried home. Parent thought due to wading in water. Flaccid paralysis of both lower limbs. Paralysis persists.

CASE 14, Kendal, *August 27th*, 1911 :—Dr. McCallum attended male aged 7 years. Type, lower limb paralysis. Recovery.

CASE 15, Crooklands, *August 27th*, 1911 :—Dr. Black (for Dr. Purves, Milnthorpe) attended male, aged 7 years, attending same school as Case 8. Type, lower limbs paralysed. Improvements marked, certain amount of paralysis persists.

CASE 16, Kendal, *August 28th*, 1911 :—Dr. Sturridge attended male aged 10. Type, lower limb paralysed. Complete recovery.

At this juncture, Dr. R. Musgrave Craven, the medical officer of health for the Westmorland Combined Districts, circularised all the doctors practising in Westmorland, seeking for information on past and present cases.

CASES 17-23, Kendal, *August 28th*, 1911, *et seq.* :—Dr. Manning about this time attended about seven cases in all. Four males, ages ranging from 3 years to 19 years, 3 females, ages from 2 to 10 years. Type, spinal. Three males and two female still paralysed.

CASE 24, Kendal, *August 29th*, 1911 :—Dr. McCallum attended male aged 1½ years. Type, meningitis. Recovery.

CASE 25, Kendal, *August 29th*, 1911 :—Dr. McCallum attended male aged 6 years. Type, lower limbs paralysed. Paralysis persists.

CASE 26, Kendal, *August 31st*, 1911 :—Dr. McCallum attended strong healthy male, labourer, aged 22. Returned from work because of violent headache, giddiness and unsteady gait. Patient removed to sanatorium. Lower limbs paralysed. Developed girdle pains. Very marked tympanites. Retention of urine for 14 days ; abdominal muscles paralysed. Rectal sphincter weak. Extremely ill. Kernig's sign marked. Tache cérébrale. Oculo-motor paresis. Repeated lumbar puncture. No meningococci—L.G.B. Lab.

CASE 27, Kendal, *August 31st*, 1911 :—Dr. Leeming, of Kendal, attended male aged 3 years. Child suddenly seized with very severe headache and vomiting. I saw this case with Dr. Leeming on September 2nd—the child was extremely ill. Unconscious ; head retracted ; paralysis of lower limbs ; nystagmus. Peculiar odour from body. Child died same night. Lumbar puncture. Clinical Research Lab.—meningococci found. Death certified cerebro-spinal meningitis.

CASE 28, Kendal, *September 3rd*, 1911 :—Saw with Dr. Cochrane (acting for Dr. Riddell, Kendal) male aged 4. Type : Twitchings of facial muscles, and of right arm. Pain on passive movements of limbs. Severe headache. Recovery.

CASE 29, Kendal, *September 3rd*, 1911 :—Dr. Brumwell called in to see male aged 59. Had been ill for some days. Temperature 103, pulse good. Intense shooting pains down muscles of neck, back

and legs. Left leg and left arm paralysed. Could talk rationally. Tongue projected straight. Pupils contracted. Epileptiform convulsions lasted all night. Died 5 a.m.

CASE 30, Burneside, *September 4th*, 1911 :—(Burneside is near Kendal). Dr. Cochrane (for Dr. Riddell) attended male infant, aged 2 years. Type, right facial and right upper arm paralysis. Recovery.

CASE 31, Kendal, *September 6th*, 1911 :—Dr. McCallum attended female aged 1 year 4 months. Type, meningitis. Recovery.

CASE 32, Milnthorpe, *September 6th*, 1911 :—Dr. Black (for Dr. Purves, Milnthorpe) attended female aged 6 years, who died September 11th. Death certified posterior basal meningitis.

CASE 33, Gatebeck, *September 7th*, 1911 :—Dr. Fuller, Milnthorpe, attended male aged 4 years. Type, lower limb paralysis. Much improved. Still certain amount of paralysis present. (Dec., 1911.)

CASE 34, Crook, *September 7th*, 1911 :—Dr. Dick, Staveley, reports as follows :—male, aged 11 years, suddenly seized with headache, vomiting, fevered pains in back. Attends same school as Case 37. Great pain on passive movement of lower limbs. Decubitus flexed position. Difficulty with micturition, priapism marked. Temperature 100. Irritable, intelligence clear. Recovery.

CASE 35, Kendal, *September 9th*, 1911 :—Dr. Cockill attended a female aged 10 years. Type, lower limbs paralysed. Dropped foot. Complete recovery.

CASE 36, Kendal, *September 9th*, 1911 :—Dr. Cockill attended a male aged 3. Complete motor paralysis of lower limbs. Paralysis persists. Considerable improvement.

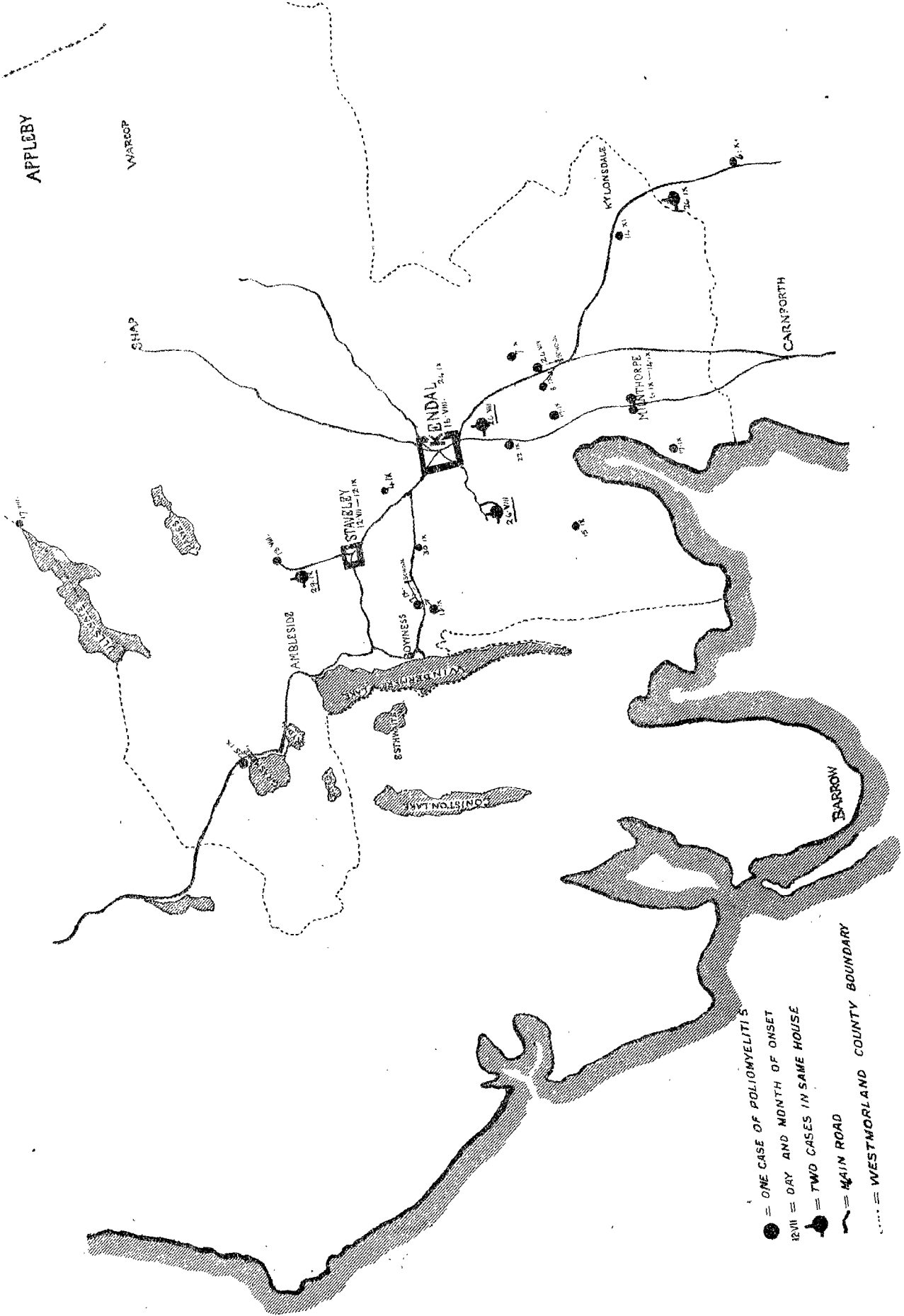
CASE 37, Crook, *September 11th*, 1911 :—Dr. J. Cochrane Henderson, Windermere, reports as follows :—male aged 9 years, complained of acute headache and pain in back, of nausea and high fever—this on September 11th.

Dr. J. C. H. called to see the boy on Sept. 13th. Temperature 103, pulse 120. Paralysis of both lower limbs. Kernig's sign present in both limbs. Retraction of head marked. Patellar reflexes abolished.

September 14th, 1911 :—Temperature 101, pulse 140. Marked prostration. *September 18th*, 5.15 p.m.—I saw this child with Dr. J. C. H. Temperature sub-normal, pulse 120, soft and unequal. Intelligence clear. A little stiffness of neck. Pupils equal, dilated slightly, reactive. Prostration extreme. Breathing shallow and laboured. Respiratory muscles seem to be involved, abdominal reflexes abolished. Complete motor paralysis of lower limbs, save for slight movements of toes of right foot and faintly of left. Sensation unimpaired. Grave prognosis given. Child slowly recovered, both limbs still partially paralysed. (December, 1911.)

CASE 38, Staveley, *September 12th*, 1911 :—After an interval of two months another case occurred in Staveley, which I saw along with Dr. Dick. Male, aged 4 years. Type, both lower limbs paralysed. No improvement. (Dec., 1911.)

CASE 39, Milnthorpe, *September 14th*, 1911 :—Dr. Fuller attended female aged 14 months. Type, lower limb paralysis. Recovery.



CASE 40, Witherslack, *September 15th*, 1911:—Dr. Black (for Dr. Purves, Milnthorpe) attended female aged 6 years. Type, lower limbs paralysed. Recovery.

CASE 41, Storth, *September 17th*, 1911:—Dr. Black attended female aged 4 years. Type, lower limb paralysis. Recovery.

CASE 42, Stainton, *September 17th*, 1911:—Dr. Black attended male aged 8 years. Lower limb paralysis. Complete recovery.

CASE 43, Kendal, *September 24th*, 1911:—Dr. Cockill reports that he attended a male aged 17. Date of onset of disease September 24th, 1911. Temperature 103, slight headache, vomiting, hyperaesthesia about neck. Kernig's sign well marked. Chief symptom great difficulty and terror in deglutition. (Clinical picture very like rabies). No paralysis of limbs. Died September 26th, 1911. Except for drowsiness 3 hours before death, the patient's intelligence was quite clear.

CASE 44, Grasmere, *September 25th*, 1911:—Dr. Hough, Ambleside, kindly furnishes the following record:

A young woman of gentle birth and comfortable circumstances, living at Grasmere, aged 24, one of a strong, active, athletic family, herself robust and never a day's illness of any consequence,

"On Friday, September 22nd, 1911, went a long walk over the fells, got wet through, came home, had a hot bath and change. Felt none the worse.

"Saturday, September 23rd. Felt as usual. A pouring wet day, so stayed in doors till evening, when she went a walk and again got wet through, again a hot bath and change. Played bridge all evening, her sister *thought she looked tired*.

"Sunday, September 24th. Went to church and spent the day as usual, not feeling ill.

"Monday, September 25th. Came down to breakfast, but felt and looked bad, so went back to bed. Complained of aching pains all over. Some headache and backache. A doctor staying in the house saw her and thought she was in for influenza, and gave her some aspirin and phenacetine, and I think a subcutaneous injection of morphia. She had a bad, painful and restless night, and seemed worse on

"Tuesday morning, September 26th. At 12 noon I saw her for the first time. Found her in bed. Looked extremely ill. Far too bad for a thorough examination. At 2 p.m. Dr. Brooksbank of Windermere kindly saw the case with me. She was propped up in bed with a most anxious expression, face dusky, lips blue, skin generally dusky in patches showing venous congestion. Using all her extraordinary muscles of respiration and alae nasi. Extremities cold and clammy. No rash. Tache cérébrale present.

Eyes.—No ophthalmoplegia, but pupils unequal. The right, though turned to the light, dilated.

Ears.—Hearing normal.

Nose.—Slight epistaxis yesterday.

Temperature.—100.9.

Respiration.—Irregular—32-58.

Pulse.—120. Soft, compressible, regular. Heart sounds feeble, no murmur, not a bad impulse.

Tongue.—Dirty patchy fur, not dry. Sordes on teeth. Swallows jerkily.

Bladder and Rectum normal.

No spasm of neck or face muscles. No rigidity of neck. No retraction of ribs. Diaphragm acting. Arms move all right voluntarily, some curious spasms of fingers.

Legs paralysed, except for power of rolling out. ? psoas action.

Patella and planters reflexes absent. No numbness or anaesthesia or hyperaesthesia.

Kernig's sign?—Leaning forward produced cramp in the thighs.

Pain. Some headache, not severe. Some pain in back.

Her fear of suffocation owing to difficulty of breathing masked all other symptoms.

She died at 5 p.m. same day, less than 5 hours from the time that I first saw her.

Examination of cerebro-spinal fluid by Dr. Mervyn Gordon confirmed diagnosis of poliomyelitis."

CASE 45, Crook Road, near Kendal.—Dr. Cockill attended male aged 8 years. Child complained of severe headache and pains in limbs. I saw this case with Dr. Cockill on September 30th—condition as follows:—Extreme irritability. Intense photo-phobia. Lying curled up in bed. Complaining of headache. Temperature 101. Flexion extreme at knees. Screams on any attempt at examination being made. Intellect clear. No head retraction. Marked myo-tatic irritability. Type, neuritis. Complete recovery.

CASES 46 and 47, Kentmere, *September 29th*, 1911:—Dr. Dick attended male aged 8 years. Transient symptoms of poliomyelitis. Left leg weak for several days. Complete recovery. Brother of case 44 (living in same house), aged 10 years, had similar symptoms.

CASE 48, Hollin Hall, near Ky. Lonsdale, *October 14th*, 1911:—Dr. Mathews, of Kirkby Lonsdale, attended a male aged 10. Type, facial and arm paralysis. Paralysis persists.

Here the epidemic ended, so far as Westmorland was concerned, but Dr. Mathews, M.O.H. for Kirkby Lonsdale, informs me that he attended three cases in Lancashire, just over the Westmorland border, at Sillet Hall. Of these, two cases occurred in the same house—a female aged 12, where the cranial as well as the spinal nerves were involved; and a boy aged 5 with lower limb paralysis. These cases were first seen on September 26th. Both are still paralysed. Again on October 6th he saw (at Docker Park, Arkholme) a boy aged 6. Type, lower limb paralysis.

Dr. Mathews makes a very interesting statement with regard to his cases. He says "In at least two of the houses (farms) I have been struck by the fact that there have been many unaccountable cases of loss among the stock."

DOUBTFUL CASE.

I have said the epidemic began on July 12th, 1911, but there died at Garlands Asylum, Carlisle, on July 6th, 1911, a labourer of Kendal, aged 45 years. Death was certified

as due to cerebral meningitis. The history of this case is very remarkable. On July 2nd, 1911, he was transferred from the County Hospital, Kendal (general hospital), to the Kendal workhouse, as he showed signs of mental disorder. When at the poorhouse he developed acute mania, was transferred to Garlands Asylum, where he died in two days' time.

At the post-mortem examination death was found to be due to meningitis, death being certified as being due to cerebral meningitis.

SUMMARY.

Total number of cases (including doubtful case, 3 cases certified as posterior basal meningitis, and one as cerebro-spinal meningitis 49

Total number of deaths 8

Mortality per cent. 16.3

Total number of males 36

Total number of females 13

Ages:—

Under 2 years ...	8	10 years,	...	7
2 years ...	2	11 years	...	3
3 years ...	4	12 years	...	2
4 years ...	4	17 years	...	1
5 years ...	1	19 years	...	2
6 years ...	2	22 years	...	1
7 years ...	3	24 years	...	1
8 years ...	3	28 years	...	1
9 years ...	2	45 years	...	1
		59 years	...	1

No. with one or more limbs paralysed. ... 37

No. with ascending paralysis ... 3

No. with acute ataxia symptoms ... 3

No. with paralysis of one or more cranial nerves 6

No. with symptoms of meningitis .. 10

No. with symptoms of acute mania ... 1

No. of cases still paralysed (Dec. 15th, 1911) 19

Two cases occurred in the same house in four instances.

Two scholars affected in the same school in two instances.

Practically all who had paralysis of one or more limbs complained bitterly of pain on passive movement of the affected limbs. Viewing the epidemic as a whole, this symptom was the most universal and striking feature in the clinical picture.

SOME CONJECTURES AS TO METHODS OF SPREAD.

Theories as to the way this disease spreads are legion. They range from French mariners hawking Spanish onions, to the house fly.

The following are some of the theories:—

1. Bathing in running water, pure or impure.
2. Consumption of foreign flour.
3. Consumption of sweetmeats infected by the house fly.
4. Exposure to heat.
5. Exposure to damp and cold.
6. Over-exertion.
7. Schools.
8. Vermin-borne.
9. Caught from lower animals.
10. Dust-borne.

Are there amongst these ten conjectures any which will fit the facts?

1. *Schools.*—In some of the epidemics recorded by Batten the school has undoubtedly been one of the contributing causes.

In the Westmorland epidemic in only two schools were two scholars affected, besides the epidemic was in full swing during the summer holidays.

2. *Vermin.*—Our answer is in the negative. The disease invaded many homes where vermin are never seen.

3. *Caught from the lower animals.*—Save for Dr. Mathews' interesting observations at the Kirkby Lonsdale farms, I am not aware of any undue mortality among lower animals in the affected areas. And yet how very like rabies is the virus itself and, in fatal cases, the symptom complex.

4. *Bathing and wading in running water.*—Hitherto in sporadic cases this has been a well-known theory. Dr. Dick, of Staveley, tells me most of his cases were bathing and wading in the icy waters of the Kent as it flows through Kentmere.

5. *Dust-borne.*—This theory has some stout defenders. In the Westmorland epidemic I must confess that when one considers the geographical distribution of the cases one is struck with their close proximity to much frequented main roads. The vast majority occurred in a narrow belt of country lying to the west and south of the county, a tract which embraces the main roads from the south to the Lakes.

Are there carrier cases?—This epidemic left Staveley free for two months and re-commenced there. As touching this fascinating subject of carrier cases we can but vaguely surmise. The more one investigates this strange and very tragic disease the more perplexing seems the solution of its epidemiology. It is all so intangible—as yet. For instance, while conducting

medical inspection at Warcop—a little village tucked away in deep pastoral solitudes between Appleby and Kirkby Stephen, I came across a boy aged 13 who ten years before was stricken with a severe type of poliomyelitis. He was quite crippled in both lower limbs. After inquiry in the village and elsewhere, this strange story emerged :—

Forty-two years ago 3 boys, aged about 3 years, had poliomyelitis within ten days of each other: one was the vicar's son, the other the blacksmith's son, and the other a farmer's son—the two latter were playfellows and were the first to be affected; the vicarage was invaded about a week after this. All were paralysed. In each case the paralysis persisted. The blacksmith's son lived in the village till about 9 years ago. Thirteen years ago a village boy suffered from poliomyelitis and still has one arm paralysed, at the same time a girl aged 4 years had an attack. Ten years ago my school-boy passed through his attack. Three years ago a boy aged 4 years had an attack of poliomyelitis of the facial palsy type. All this happened among a little community of some 200 people. Is there a carrier case here?

Or again, why do cerebro-spinal meningitis, posterior basal meningitis, and poliomyelitis exist cheek by jowl in the same epidemic?

What is the incubation period?

Batten suggests within 6 days.

Bramwell and Currie, investigating the Tilli-coultry outbreak, think 4 days.

Our experience in Westmorland would seem to point to a similar conclusion. See cases 9 and 10; 11 and 12; 8 and 15; 34 and 37.

The late Professor William James used to say that "the field for new discoveries is always the unclassified residuum." That the unclassified residuum is a fine field for new discoveries in the domain of poliomyelitis, we must all admit.

I desire most cordially to thank the Doctors practising in Westmorland for the help they so readily afforded me, and that too in the days before poliomyelitis was made a notifiable disease. I am deeply indebted also to Dr. Reginald Farrar of the Local Government Board, to Dr. Craven, M.O.H. for the Westmorland combined districts, to Dr. Mathews, M.O.H. for the Kirkby Lonsdale Urban District, and to Drs. Batten, Edwin Bramwell, and Mervyn Gordon for much valued counsel and guidance during my investigations.

SOME EXPERIENCES IN CONNECTION WITH EPIDEMIC DIPHTHERIA.*

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AT a period when there is an increasing demand for the inclusion within the sphere of public health activity of non-communicable diseases, both medical and surgical, an apology seems to be almost indicated for introducing so hackneyed and threadbare a subject as diphtheria.

Nevertheless, in spite of an apparent decline in the general mortality from this disease in recent years, and especially since 1902, the country is in about the same position as it was between 1880 and 1890, diphtheria still accounting for between 5,000 and 6,000 deaths annually in England and Wales. And, since the fatality has certainly been reduced during the last ten years or so by the use of antitoxin and the extension of hospital treatment, it is reasonable to assume that the incidence is not falling so rapidly as might be desired, diphtheria being of all the common infectious diseases, after perhaps smallpox, the most easily controlled by direct attack.

The well informed critic might even be disposed to suggest that, before the field of work of the medical officer of health is enlarged in accordance with the desire of many, a more determined effort should be made to deal with diphtheria, and, one might add in parenthesis, scarlet fever as well.

Diphtheria has been a subject of especial interest to myself since the time that I first commenced practical work in our particular branch of medicine. Whilst I was assisting Dr. Thresh in Essex, I had charge of the hospital attached to one of his rural districts, and entered upon my duties in the middle of a big village epidemic. Nothing could be much more distressing than the arrival of child after child, bearing evidence of country vigour and parental care, who nevertheless died within the first fortnight of the disease with that almost hopeless combination of vomiting and cardiac failure that is so characteristic of certain types of epidemics. No one who has had an experience of this kind, and who has personally visited the stricken village time after time, can fail to realise the necessity for taking far more energetic steps for controlling the disease than

* Read before the Home Counties Branch of the Society of Medical Officers of Health, on December 16th, 1911.