POLIOMYELITIS

SOME ASPECTS OF THE DISEASE IN ABERDEENSHIRE WITH SPECIAL REFERENCE TO THE OUTBREAK IN 1947

THESIS

FOR THE DEGREE OF

DOCTOR OF MEDICINE

PRESENTED BY

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C O N T E N T S.

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SCOPE OF THE THESIS.

In this Thesis I have made a study of Poliomyelitis in Aberdeenshire, where it first became a notifiable disease in 1913.

In 1947, there occurred in Great Britain an outbreak of the disease of greater proportions than had ever been seen before. In that year, a total of eighty-one persons in the City and in the County of Aberdeen contracted it; all were admitted to the City Hospital, Aberdeen, and all were there examined by me personally.

Firstly, in the Historical Section, I have tried to determine how long poliomyelitis has been present in Scotland generally, and in Aberdeenshire in particular.

The incidence of the disease in both the City and County of Aberdeen since 1913 has then been cited, and the 1947 Aberdeenshire cases have been classified.

Some Epidemiological Features of the disease, as they presented themselves in 1947 and in previous years, are then described, and a study made of its general behaviour - the places it visited, the people it attacked, and its effects upon them.

Differential Diagnosis is then discussed in relation to five conditions simulating poliomyelitis which have been seen in Aberdeen.

Finally, summaries of the case notes of all the 1947 Aberdeenshire cases of poliomyelitis have been set down in the Appendix, and two Maps have been added showing the distribution of cases in the City and County of Aberdeen.

HISTORICAL REVIEW OF POLIOMYELITIS IN SCOTLAND.

In tracing the history of poliomyelitis in Scotland it is not easy to determine when it first became recognised. References to the disease in the writings of early Scottish physicians are not numerous.

Several pieces of evidence suggest that the disease has a very long history in other parts of the world. The first is that there is, in the Museum of Pennsylvania University, a peculiar skeleton, dating from 3700 B.C., which was found in a village south of Cairo and in which one leg is shorter than the other. The International Committee (1932) states that the investigator who first noticed this - in 1900 A.D. - after careful consideration came to the conclusion that ".... nothing seems so probable as that the defect of growth is due to an attack of poliomyelitis....". The Committee also quotes another instance in the paleopathology of Egypt; an Egyptian stele, dating from between 1580 and 1350 B.C. depicts a man with a withered leg, with the foot in the "equinus" position. There was apparently no question that the drawing was inaccurate, and the possibility that the

deformity might be due to some other lesion of the nervous system or to disease of the hip joint, was recognised. The considered opinion of the investigator was that it was due to poliomyelitis.

In approximately 400 B.C. Hippocrates (Adams 1849) described a winter in the Island of Thasus - "....cold with great winds and snow...." - when "paraplegia set in and attacked many and some died speedily; and otherwise the disease prevailed much in an epidemic form, but persons remained free from all other diseases". It is difficult to think of any condition other than poliomyelitis that Hippocrates could have been describing, especially as he used the word "epidemic" in the same sense as that in which it is used to-day. Another comparatively ancient record is quoted by Aycock (1929). In Herjolfnes in South Greenland, twenty-five skeletons, dating from the beginning of the 15th century, were discovered in 1921. Aycock quotes Hansen's description of six of them, in which scoliotic pelves, kyphoscoliosis, atrophy of the shoulder and lower extremity and cubitus valgus deformity were noted. Aycock states that in his opinion the deformities of the bones very probably resulted from poliomyelitis in childhood.

Any positive proof that the disease did occur in olden times is unlikely to be found. It appears that it was not until 1784 that it began to be recognised by clinicians in this country. In that year Michael Underwood, a licenciate of the Royal College of Physicians of London, described it under the title of "Debility of the Lower Extremities". He states "....It is not a common disorder anywhere....nor am I enough acquainted with it to be fully satisfied either in regard to the true cause or seat of the disease". He also makes the point that, as far as he knew, no other medical writer had described it before him.

The disease is certainly not described as an epidemic one by the chroniclers whom Creighton quotes in his History of Epidemics from 664 till 1893 (1891 and 1894), but some reference to loss of power in the limbs is mentioned as for example in 1694, during the Fevers of the Seven Ill Years in Scotland when an illness affecting many people is described - " many contracting their deaths and losing the use of their feet and hands". But there was famine at the time and it appears more likely that the disabilities were caused by frost-bite from working in

the snow, in addition to debility engendered by lack of food and the effects of typhus fever.

A search through Hirch's Geographical and Historical Pathology (1883) has revealed no reference to the disease and one might certainly conclude that it can never have assumed the proportions that are known to-day. If it had occurred in such epidemic form as in 1916 when, in the registration area of the United States of America, there were 27,000 cases and 6,000 deaths (Department of Health, New York City, 1917), then the fact would doubtless have been mentioned by those who described the epidemics of other diseases in previous centuries.

Probably the first reference to poliomyelitis in Scotland occurs in non-medical literature - in Lockhart's Life of Sir Walter Scott and describes how Scott at the age of eighteen months, in 1772, was attacked by the disease. He was living in Edinburgh at the time. It reads as follows: "One night, I have often been told, I showed great reluctance to be caught and put to bed; and, after being chased about the room, was apprehended and consigned to my dormitory with some difficulty. It was the last time I was to show personal agility. In the morning I was discovered to be affected

with the fever which often accompanies the cutting of large teeth. It held me three days. On the fourth, when they went to bathe me as usual, they found that I had lost the power of my right leg. My grandfather, an excellent anatomist as well as physician, the late Alexander Wood, and many others of the most respectable of the faculty, were consulted. There appeared to be no dislocation or sprain. Blisters and other topical remedies were applied in vain. When the efforts of regular physicians had been exhausted, without the slightest success, my anxious parents, during the course of many years, eagerly grasped at every prospect of cure which was held out by the promise of empirics or of ancient ladies and gentlemen who conceived themselves entitled to recommend various remedies, some of which were of a nature sufficiently singular." A further indication of the nature of the disease may be inferred from the following sentence relating to Scott when he was four years old - "...although the limb affected was much shrunk and contracted, I, my lameness apart, was a sturdy child".

A paper entitled "Case of Inflammation of the Anterior Columns of the Spinal Cord. Recovery" was read in 1868 before the Dundee Medical Society by Crockatt. He

described the case of an adult male patient whom he saw in July, 1866. Three months previously the patient had become unwell with stiffness of the back, which developed into deep-seated pain in the lower spine and across the loins. Soon afterwards weakness of the legs was noticed, and this was followed a month later by stiffness and paresis of the left arm, and two months after that by a similar affection of the right arm. Crockatt noticed when first examining the patient that there was wasting in the affected groups of muscles and that there was no sensory loss. In discussing the etiology, he pointed out that there was paralysis and emaciation of the muscles of the limbs, with no sensory loss, and that the trunk muscles were unaffected. It seems unlikely, in view of the long history and the pain, that the condition was poliomyelitis, but it is interesting that the site of the lesion was considered to be in the anterior horn cells.

In 1881 Finlayson described a "Case of so-called Infantile Paralysis" in a twenty-three year old woman in Glasgow. He stated that the lesion was situated in the anterior cornua of the spinal cord, destroying large nerve cells there. He noted the history of acute onset with pain in the back, followed by paralysis of the right

upper limb. Later he observed wasting, but also signs of return of power in the affected muscles.

Byrom Bramwell (1881) showed a series of microscopical preparations to the Medico-Chirurgical Society of Edinburgh, illustrating the post mortem findings in a two-year old boy who died from diphtheria in 1879. He had developed poliomyelitis two months previously, with paralysis of the right lower extremity. Bramwell remarked that although infantile paralysis "is frequently met with in practice" it was rare for a post mortem examination to be made. He noted the disappearance of nearly all the large motor nerve cells in the anterior horns of the right lumbar spinal cord. The feature of interest from the historical aspect is his statement that the disease was often seen by practising physicians at the time.

Stephen (1886) described in the Glasgow Medical Journal a case of poliomyelitis with unusual features, in a boy of five years. There was a febrile onset, with vomiting and convulsions, followed by paralysis of the neck muscles, so that "the occiput rested upon the dorsal region of the back". He quotes Hammond (1876) who stated "I have never seen a case in which any muscle of the head

or neck was involved", and Buzzard (1882) who stated "My own observations would lead me to think that the muscles of the trunk are not infrequently involved". One might well conclude that these statements were formulated following some considerable experience of the disease.

Three lectures were published in the early part of the present century by a Scottish physician, Byron Bramwell. In the first (Bramwell, 1905), he discussed the treatment of the disease, pointing out the usually rapid progression of the paralysis and the consequent difficulty of applying any treatment which might prevent its development. He advised the use of anti-pyretics in the pre-paralytic stage whenever the disease was suspected. On the occurrence of paralysis he thought that leeches and cold compresses should be applied over the spine in order to subdue inflammation in the anterior horn cells and that hyperaemia of the affected part of the spinal cord might be lessened by making the patient lie on the unaffected side. After the febrile stage he recommended the giving of quinine, potassium iodide or mercury in order to promote the absorption of inflammatory exudate in the cord. Bramwell insisted on complete rest for the affected limb for a period of four to six weeks, when active and

passive movements should be started and massage and electrotherapy brought into use as adjuncts to the restoration of power. He also realised that there was a place for surgery in the treatment of the residual paralyses and mentioned procedures such as division of the tendo Achilles and resection of the knee joint.

In his second lecture (Bramwell, 1908a) he described the results of lesions in the region of the anterior horn cells of the spinal cord. An acute lesion would lead to a flaccid paralysis of rapid onset with abolition of superficial and deep reflexes, and what he termed "trophic changes" - atrophy and reaction of degeneration in the paralysed muscles. There would be absence of sensory changes and bedsores, and in most cases, absence of paralysis of the bladder and rectum. Bramwell contrasted such an acute lesion with the chronic one of progressive muscular atrophy, and with lesions which did not exclusively involve the anterior horn cells, as, for example, amyotrophic lateral sclerosis, syringomyelia, disseminated sclerosis, combined sclerosis and tumours.

In a third lecture Bramwell (1908b) analysed, from the clinical aspect, 76 cases of poliomyelitis which had

occurred in or near Edinburgh; he pointed out that the majority - 80% - of the cases occurred within the first six years of life and described one case where the disease was said to be present at birth.

A fatal case of poliomyelitis in a woman of 41 was very fully described by Pirie (1910) who was then on the staff of the Department of Medical Jurisprudence at Edinburgh University. At the onset there was paralysis of the legs only, but the patient died within five days from what Pirie considered to be involvement of the vital centres. He thought that the affection was esentially an ascending myelitis - one of the eight types of poliomyelitis recognised by Wickman (1913). At post mortem examination the spinal cord was found to be soft and swollen and the pia mater congested. Microscopically there was engorgement of arteries and veins involving the whole length and breadth of the cord, with scattered petechial but no gross haemorrhages. Pirie found no indication of any thrombosis in the anterior spinal vessels - a theory which Batten in 1904 had suggested as explaining the symptoms of the disease; but he noticed the striking "collar" of cells round the vessels and the massive infiltration of the anterior horns by

round cells. He also observed that the lesion was not confined to the anterior horns but there was appreciable, though less intense, infiltration of the lateral and posterior horns and of the central region of the cord. He also observed greatly reduced numbers of nerve cells in the lumbar cord, and those that could be distinguished were swollen and rounded and had largely lost their Nissl's granules. The brain was not examined.

A case of acute ascending "Landry's" paralysis with recovery was presented by Monro (1911) to the Medico-Chirurgical Society of Glasgow. The patient, a joiner aged 52, was admitted to hospital in October 1910 suffering from progressive numbness and loss of power in the legs, of one week's duration. On admission he was afebrile and complained of pain in the calves; there was incomplete flaccid paralysis and slight impairment of sensation in the upper and lower extremities. The paralysis progressed to a maximum for a period of 17 days after admission and thereafter he gradually improved. When seen 3 months later he was easily tired and the left ankle jerk was absent, but otherwise he had fully recovered. There was no wasting and the normal

electrical reactions were preserved throughout. Monro compared his patient's illness with that which Landry had described in 1859 and pointed out that the differentiation between acute ascending myelitis, poliomyelitis and acute toxic neuritis was not always clear.

Landry's paralysis is further discussed in the section on Differential Diagnosis, but the observations of Pirie and Monro above suggest that in 1911 this diagnosis was not regarded as altogether satisfactory per se.

An important sequel to poliomyelitis was also described at this time by Ness (1911). A child of three years lost the power of his right leg and left arm. The leg remained paralysed but the arm recovered. Thereafter a swelling in the right flank was noticed and Ness was consulted because this showed no sign of disappearing. He found that the right lower limb was flail and that there was considerable weakness and wasting of the right external and internal oblique and transversalis muscles of the abdomen, resulting in a hernia and scoliosis of the spine. Mr. Alexander Rennie F.R.C.S., in a personal communication, states that he has found that scoliosis of

the spine with this etiology is not uncommon in orthopaedic practice in Aberdeen.

An epidemic of 62 cases of poliomyelitis occurred in 1910 in and near Edinburgh. The cases were presented and discussed before the Royal Society of Medicine by Lowe (1912). 47 of the cases came from the country - one from as far north as Sutherlandshire - and 12 others from north of the Forth, mostly in the Perth and Kirkcaldy regions.

In this section I have tried to set down the history of poliomyelitis in Scotland prior to 1913, but any opinion of its incidence in the North East of Scotland is impossible to formulate, since I have been unable to find any published work on the subject. Nevertheless one might assume from its known presence elsewhere in Scotland that the disease did occur in the North East. Two isolated instances might be offered in support of this idea. Firstly, Miss G.H., who lives at Pitfodels, Aberdeenshire, gave a history of having had the disease in 1891. She was 2 years of age at the time and was left with paralysis of her left leg. The limb has recently been amputated on account of severe intractable chilblains. She

stated that there were other cases at the time but was unable to give any names or figures. Secondly, A.S. - the father of one of the cases (No. 71) in the 1947 outbreak - developed the disease in 1905, and still shows evidence of some wasting of the left leg.

In May 1913 poliomyelitis was made a notifiable disease in Aberdeen by order of the Town Council and from then until now the notifications have been recorded in the Annual Reports of the Medical Officer of Health. Apart from these, no reference to the disease in the North East of Scotland has been found, although several accounts of its occurrence elsewhere in Scotland have been published. (Campbell 1913, Harrington and Teacher 1916, Bramwell 1919, Halliday 1930, Cappell 1930, Fleming 1931, Cruickshank 1933.)

INCIDENCE OF THE DISEASE SINCE 1913.

CITY of ABERDEEN. The disease first became notifiable in the City of Aberdeen in 1913 and the number of notifications have been obtained from the Annual Reports of the Medical Officer of Health for the years 1913 to 1947 inclusive. A number of errors have, however, been noted, the details of which are as follows.

In 1913, out of a total of 7 notifications, 3 were patients admitted to the Aberdeen Royal Infirmary, from Finzean, Peterculter and Kingussie, and have therefore been excluded.

In 1916,,2 of the notified cases were labelled "doubtful" by the Medical Officer of Health, but they have been included since they were notified by practitioners and the reason for so labelling them is not indicated.

In 1934, there were 2 notifications, but one of them was a very longstanding case who certainly did not contract the disease in 1934, and has therefore been omitted.

In 1937, there was one case notified which, however, did not appear in the Medical Officer's Report. It has been included.

In 1944, 6 of the 20 notified cases came from the County of Aberdeen, and have therefore been excluded.

Furthermore, a search of the records of the Aberdeen Royal Infirmary and the Royal Aberdeen Hospital for Sick Children has revealed case notes of 10 patients who were admitted there and for which no notifications were made - 1 in 1927, 2 in 1930, 2 in 1938, 2 in 1940, 1 in 1942, and 2 in 1944.

Allowing for these corrections it appears, therefore, that, so far as can be ascertained, 250 cases of poliomyelitis occurred in the City of Aberdeen from 1913 to 1947 inclusive. Of these, 240 were notified and 10 were not. Notes of 163 of the cases were found in the records of the Aberdeen City Hospital, of 13 in those of the Royal Aberdeen Hospital for Sick Children and of 4 in those of the Aberdeen Royal Infirmary. Case notes of the remaining 70 patients could not be traced.

COUNTY of ABERDEEN. Although poliomyelitis became notifiable in the County of Aberdeen also in 1913, unfortunately the records of the Public Health Department for the years 1913 to 1918 are very incomplete and it is therefore necessary to omit that period from this investigation.

During the years 1919 to 1947, 138 notifications were made, but again a number of corrections have been found necessary.

The case notes of one case which had been notified in 1939 were found in the records of the Aberdeen Royal Infirmary and showed that the acute episode of the disease had occurred many years previously; it has therefore been excluded.

In 1944, 4 notifications had been wrongly entered in the City lists - vide supra - and have therefore been added.

Lastly, case notes of 10 County patients who had not been notified were found - 8 in the records of the Aberdeen City Hospital and 2 in those of the Royal Aberdden Hospital for Sick Children.

There is thus a corrected total of 151 cases for the County of Aberdeen for the years 1919 to 1947 inclusive. Of these, 89 case notes were found in the records of the Aberdeen City Hospital and 8 in those of the Royal Aberdeen Hospital for Sick Children. The remaining 54 patients could not be traced.

The yearly incidence of the disease is set out, for City and County, in Table 1 following.

Table 1. Yearly Incidence of Poliomyelitis in Aberdeen.

Year	No. of Cases		
	City	County	Total
1913	4		
1914	2		
1915	8		
1916	81		
1917	10		
1918	2		
1919	1	1	2
1920	5	-	5
1921	8	4	12
1922	9	1	10
1923	-	-	-
1924	6	-	6
1925	5	2	7
1926	5	-	5
1927	4	1	5
1928	2	17	19
1929	2	3	5
1930	3	1	4
1931	1	2	3
1932	1	1	2
1933	10	11	21
1934	1	1	2
1935	1	1	2
1936	-	3	3
1937	1	3	4
1938	8	7	15
1939	1	4	5
1940	5	7	12
1941	2	8	10
1942	1	3	4
1943	1	3	4
1944	16	25	41
1945	-	3	3
1946	1	7	8
1947	49	32	81
Total	256	151	

CLASSIFICATION OF THE 1947 CASES.

Several different classifications of the various types of poliomyelitis have been suggested from time to time. Wickman (1913) divided the disease into eight types, namely

1. Spinal myelitic type.
2. That resembling Landry's paralysis.
3. Bulbar and pontine types.
4. The encephalitic type.
5. The ataxic type.
6. The polyneuritic type.
7. The meningitic type.
8. The abortive type.

The report of the 1916 New York Epidemic (1917) divided the cases in the following way:

1. Non-paralytic or abortive type.
2. Ataxic type.
3. Cortical type.
4. Ordinary spinal or subcortical type.

Over 700 Philadelphia cases were classified by Weisenberg (1917) into these groups:

1. Spinal form.

2. That resembling Landry's paralysis.

3. Pontine - bulbar.

   a. Bulbar.
   b. Pontine.
   c. Pontine-bulbar.
   d. Pontine-spinal.
   e. Bulbar-spinal.
   f. Pontine-bulbar-spinal.

4. Encephalitic.

5. Cerebellar.

6. Meningitic.

7. Abortive.

The 1947 Aberdeenshire cases have been divided on a relatively simple basis of clinical severity into four groups.

Group 1. Those who died - designated "D" in the tables and case summaries which follow.

Group 2. Those who developed paralysis which had not recovered after three weeks in hospital - designated "P".

Group 3. Those who had paralysis which recovered completely within three weeks - designated "R".

Group 4. Those without paralysis, but who showed meningeal signs - designated "M".

There are, of course, obvious fallacies in this form

of classification. The arbitrary period of three weeks for recovery or otherwise from paralysis was taken because after that period of time those patients who still showed paralysis were no longer retained in the City Hospital but were transferred elsewhere for orthopaedic treatment. In some of these complete recovery did, in fact, occur later. Moreover, Bodian (1947) has shown that all cases are encephalitic as well as myelitic, and it must therefore be purely fortuitous where the most intense lesions occur. Furthermore, a small lesion situated in the vital centres of the brain might well prove fatal, whereas a lesion of the same size in the lumbar cord might result in minimal disability. On the other hand, provided the patient survives, the site of the severest lesion, whether in the brain or cord, is of little importance in estimating the intensity of the infection in any individual patient. In other words, a patient with hemi-paresis of the face has been regarded, in the classification which I have used, as being as severely affected as one who had paralysis of all four limbs; and one who had ataxia which recovered rapidly as being comparable to one who had temporary weakness of any muscle or muscle-group.

In 31 of the 1947 Aberdeenshire cases no evidence of paralysis or paresis of muscles was found, but all showed meningeal signs and there were changes in the cerebro-spinal fluid. The differential diagnosis is discussed later, but it must be said that, without means for demonstrating the virus, it is impossible in these cases to be quite certain of the diagnosis of poliomyelitis. Nevertheless, the non-paralytic form of the disease is well recognised. Horstmann (1948) thinks that the number of these cases is increasing. In England and Wales in 1947, 23.3% of 4,717 cases were of this type (Bradley and Gale, 1948), and they are now regarded as indicating a very widespread distribution of the disease (Casey et al., 1945).

Then, in relation to the grouping outlined above, the question of bladder involvement is, I think, important. Whether retention of urine should be regarded as a paralytic phenomenon in poliomyelitis appears to be debatable. Retention of urine may occur in many conditions in which there is no apparent parenchymal damage in the central nervous system. Bodian (1947), however, has demonstrated lesions in the lateral horn of grey matter in poliomyelitis, and Fulton (1943) states that the

preganglionic parasympathetic fibres in the sacral region originate there, so that it seems reasonable to assume that retention may be a paralytic manifestation of the disease. Toomey (1933), on the other hand, thought that this was unlikely in view of the rarity with which he found somatic paralysis in the same segment as that from which the detrusor muscle of the bladder is supplied. If, however, as is the case, the disease can select very limited groups of muscles, or even single muscles, it seems reasonable tom assume that it might well cause paralysis of the detrusor muscle of the bladder alone. Again, Wright (1936) believed that bladder dysfunction in poliomyelitis was due to peripheral neuritis. In the present classification, retention of urine is assumed to be a paralytic manifestation of the disease. On this basis two patients (Nos. 9 and 52) have been included in Group 3 ("R") in whom it was the only sign of paralysis. The number of cases falling into each group is set out below.

Table 2 Clinical Classification of the 1947 Cases

	No. of Cases
Group 1 - "D"	6
Group 2 - "P"	32
Group 3 - "R"	12
Group 4 - "M"	31
	81

SOME EPIDEMIOLOGICAL FEATURES OF THE DISEASE.

Urban and Rural Incidence.

In Table 3 are set down the yearly Case Rates in City and County per 100,000 of population (figures taken from the Census of Scotland) and these are illustrated graphically in Figure 1. It is apparent that the County rate is slightly higher than that of the City - the mean rates being 3.5 per 100,000 and 3.1 per 100,000 respectively.

This trend towards a greater rural incidence in poliomyelitis was first recorded by Wickman (1913) who noted it in relation to the Swedish epidemic of 1905. Lavinder et al. (1918) described it as a very constant feature of the disease and believed that there must be some fundamental law to account for it. This tendency, however, is generally regarded as a feature of most epidemic infectious diseases and Gill (1928) has noticed it in relation to malaria and plague in India. Sweetman (1948) found the very high attack rate of 109 per 100,000 in the rural town of Eccles in the 1947 epidemic of poliomyelitis. The reason usually postulated for a higher rural incidence is that in urban areas the

Table 3. **Yearly Case Rates in Relation to Population**

Year	Cases per 100,000 of population	
	City	County
1913	2.5	
1914	1.2	
1915	1.2	
1916	50.0	
1917	6.2	
1918	1.2	
1919	0.6	0.65
1920	3.1	0.0
1921	5.0	2.61
1922	5.6	0.65
1923	0.0	0.0
1924	3.8	0.0
1925	3.1	1.30
1926	3.1	0.0
1927	2.5	0.65
1928	1.3	11.11
1929	1.3	1.95
1930	1.9	0.65
1931	0.6	1.37
1932	0.6	0.68
1933	6.0	7.53
1934	0.6	0.68
1935	0.6	0.68
1936	0.0	2.05
1937	0.6	2.05
1938	4.8	4.79
1939	0.6	2.80
1940	3.0	4.79
1941	1.2	5.41
1942	0.6	2.05
1943	0.6	2.05
1944	9.6	17.12
1945	0.0	2.05
1946	0.6	4.79
1947	29.3	21.91
	152.9	102.37

Mean Rate 1913-47 = $\frac{152.9}{35}$ = 4.4

Mean Rate 1919-47 = $\frac{90.6}{29}$ = 3.1 $\qquad \frac{102.37}{29}$ = 3.5

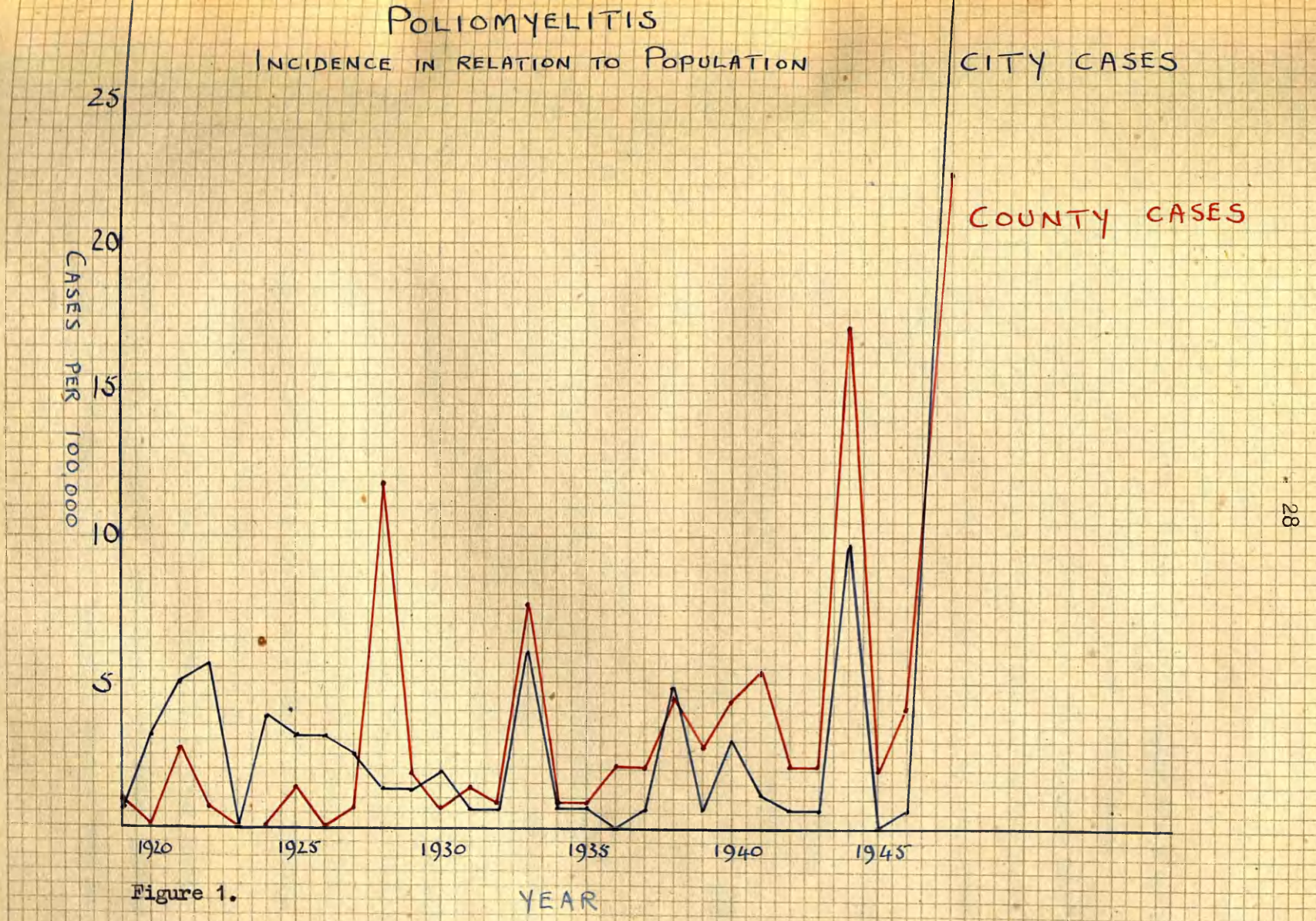

POLIOMYELITIS
INCIDENCE IN RELATION TO POPULATION
CITY CASES
COUNTY CASES
CASES PER 100,000
25
20
15
10
5
1920
1925
1930
1935
1940
1945
YEAR

Figure 1.

chances of exposure to the infective agent are greater and that therefore immunity through sub-clinical infection is more often obtained. The immunity of the rural population being lower, relatively more clinical cases occur there once the infective agent has become highly prevalent.

The difference in incidence between City and County areas in Aberdeen is, as noted, small and perhaps nowadays this difference may become less evident in view of the increasing facilities of modern modes of transport.

Epidemic and Endemic Incidence.

The yearly incidence of City and County cases together and in relation to population has been set down in Table 4. It was found that, although the County population had decreased slightly in the decade between the 1921 and the 1931 Censuses, the City population had at the same time increased by an almost equal amount, so that the total population of City and County together remained almost the same. (Table 5).

Table 4 indicates that, over the 29 year period 1919 to 1947 inclusive, the endemic or median rate is 1.60 per 100,000 per year, and that the mean rate is

Table 4.

Yearly Incidence of Poliomyelitis in Relation to Population.

Year	No. of Cases			Rate per 100,000 of population.
	City	County	Total	
1919	1	1	2	0.64
1920	5	-	5	1.60
1921	8	4	12	3.84
1922	9	1	10	3.20
1923	-	-	-	0.00
1924	6	-	6	0.64
1925	5	2	7	2.24
1926	5	-	5	1.60
1927	4	1	5	1.60
1928	2	17	19	6.09
1929	2	3	5	1.60
1930	3	1	4	1.25
1931	1	2	3	0.96
1932	1	1	2	0.64
1933	10	11	21	0.73
1934	1	1	2	0.64
1935	1	1	2	0.64
1936	-	3	3	0.96
1937	1	3	4	1.25
1938	8	7	15	4.80
1939	1	4	5	1.60
1940	5	7	12	3.84
1941	2	8	10	3.20
1942	1	3	4	1.25
1943	1	3	4	1.25
1944	16	25	41	13.17
1945	-	3	3	0.96
1946	1	7	8	2.56
1947	49	32	81	25.96
	149	151	300	94.71

Mean Rate $= \frac{94.71}{29} = 3.26$

Median Rate = 1.60

Index of Epidemicity = 2.04

Table 5.

	1921 Census+	1931 Census+
Population of Aberdeen City	158,963	167,258
Population of Aberdeen County	153,392	145,601
Total	312,355	312,859

+ Census of Scotland 1921 and 1931

3.26 per 100,000 per year. For the whole of Scotland the median rate was 1.0 and the mean 1.8 for the years 1925 to 1946 (Health Bulletin, 1948), indicating that the rates are somewhat higher in the North East than over the whole of the country. The ratio between the median and the mean rates gives an index of epidemicity (International Committee, 1932). For the North East this is 2.0, which differs little from the figure for the whole of Scotland - 1.8.

The sharp rise in incidence in 1947 apparent from figure 1 may be partly explained by the inclusion of a considerable number of non-paralytic cases and this type of the disease is becoming increasingly recognised in the notification of poliomyelitis. In Aberdeen these cases were first included in 1928, when 4 of 19 cases notified had no paralysis. In 1944, 4 of 41 notified were non-paralytic. In 1947, out of a total of 81 cases notified, no less than 31 showed no sign of paralysis.

The epidemic rate for the North East of Scotland is difficult to estimate. Lavinder et al. (1918) state that "in a large aggregation of people, such as the

population of a city with over 100,000 inhabitants, a county or state, epidemics seldom attack more than one in a thousand of the population, often not more than one in two to four thousand". In the case of Scotland, one in every four thousand would mean that an outbreak would have to total 1,200 cases before it could be called an "epidemic", and by the same standard, 78 cases would be required in Aberdeenshire. On only two occasions in the North East of Scotland - in 1916 when there were 81 cases in the City, and in 1947 when there were 81 cases in the City and County of Aberdeen - has this minimum standard been attained. 1947 was regarded throughout Great Britain as an epidemic year and the incidence in England and Wales was only 18 per 100,000 (Lancet, 1948). The previous highest recorded figure for England and Wales was 3.8 per 100,000. It is interesting to note that, although 18 per 100,000 was only once previously exceeded in the North East of Scotland, on seven occasions there was a yearly rate of over 3.8 per 100,000 - in 1916, 1921, 1928, 1933, 1938, 1940 and 1944.

Although the epidemic and endemic incidences are

both rather low in the North East of Scotland, nevertheless the disease has the power to spread very widely. This may be seen from the maps of the City and County, on which the cases have been plotted. In Table 6 are given the names of the places in the County of Aberdeen where cases have occurred during the period under review. Thus, in 1944 for example, there were 16 cases in the City and 25 in the County; there was no obvious focus of intense infection in the City and the County cases were scattered in 17 separate localities. With the exception of 1928 and possibly of 1941, the geographic distribution of cases during the period under review suggests, I think, that the population at risk is small and widely spread and that the infective agent itself is either disseminated throughout the whole area or else is distributed in some very abstruse manner. The ability of poliomyelitis to spread in this way is a well recognised feature of the disease, and has been called the "creeping tendency" by by Dale (1928). It is, however, impossible to determine where it "creeps" from in any given year, and the impression to be gained from the two maps is, that cases of the disease appear in the different localities for some

Table 6.

Distribution of County Cases 1919 - 1947

Year	Place Name	No. of Cases
1919	Drumblade	1
1920	-	-
1921	Boyndlie	1
	Fraserburgh	3
1922	Bucksburn	1
1923	-	-
1924	-	-
1925	Cairnbulg	1
	Turriff	1
1926	-	-
1927	Dinnett	1
1928	Auchnagatt	1
	Cluny	1
	Fyvie	13
	Tillyfourie	1
	Rothienorman	1
1929	Auchterless	1
	Kemnay	1
	Turriff	1
1930	New Deer	1
1931	New Machar	1
	Kintore	1
1932	Dyce	1
1933	Fetterangus	3
	Huntly	1
	Insch	2
	Inverurie	1
	Old Deer	1
	Premnay	1
	Rhynie	1
	Tarland	1
1934	Pitfour	1
1935	Peterhead	1
1936	Ellon	1
	Peterculter	2
1937	Huntly	1
	St. Combs	1
	Maud	1

Table 6 (cont'd)

Year	Place Name	No. of Cases
1938	Ballogie	1
	Bankhead	1
	Ellon	1
	Forgue	1
	Kincardine O'Neil	1
	St. Combs	1
	Bucksburn	1
1939	Hatton	2
	Maud	1
	New Aberdour	1
1940	Alford	1
	Bridge of Don	2
	Fetterangus	1
	Pitfodels	1
	Udny	2
1941	Collieston	4
	Clatt	1
	Forgue	1
	Lumsden	1
	Peterhead	1
1942	Dyce	1
	Hatton	1
	Huntly	1
1943	Aboyne	1
	Auchnagatt	1
	Forgue	1
1944	Ellon	1
	Fraserburgh	1
	Glass	1
	Insch	3
	Inverurie	2
	Lyne of Skene	1
	Midmar	2
	New Deer	1
	Old Meldrum	2
	Peterhead	4
	Pitcaple	1
	Skene	1
	Tarland	1
	Tarves	1
	Torphins	1
	Ballater	1
	Pitfodels	1
1945	Crathie	1
	Methlick	1
	Old Meldrum	1

Year	Place Name	No. of Cases
1946	Fraserburgh	1
	Peterhead	1
	Udny	1
	New Deer	1
	Maud	2
	Mintlaw	1
1947	Alford	1
	Auchnagatt	1
	Bieldside	2
	Bucksburn	3
	Buchanhaven	1
	Corse of Monelli	1
	Cults	1
	Dyce	2
	Fetterangus	1
	Fraserburgh	2
	Fyvie	1
	Gartly	2
	Huntly	2
	Inverurie	2
	Maud	1
	Memsie	1
	Old Meldrum	1
	Peterhead	2
	St. Catherine's	2
	Stuartfield	2
	Udny	1

other reason than direct spread of infection from nearby.

From Table 6 and the County map it can be seen that the disease visited 18 localities on two or more occasions. For example, Peterhead had one case in 1938, one in 1941, four in 1944, one in 1946 and two in 1947; Auchnagatt had one case in 1928, one in 1943 and one in 1947. What is perhaps more interesting is that many other places of comparable size - New Pitsligo, Strichen, Cruden Bay and numerous others - have been free from the disease during the period under review, so far as can be seen from the information which has been collected. This probably does not mean that those who live in these localities have never been exposed to the disease, but rather that their immunity is such that no paralytic cases have appeared.

With regard to the City cases, the impression gained from plotting them on the map is that there were more cases in the more densely populated parts of the City.

An attempt was made to calculate the population densities of the municipal wards and relate them to the incidence of cases of poliomyelitis occurring within the corresponding ward boundaries. It was found, however, that

it was not possible to do this with any degree of accuracy because between 1913 and 1947, two municipal wards were added in the City and a further seven had their boundaries extended. Moreover, the population varied to some extent in the different wards from year to year. Nevertheless, in order to compare the case incidence and population density for the two epidemic years 1916 and 1947, ward densities were calculated from data supplied by the City Engineer's Department and the results have been set down in Table 7. Thereafter the number of cases of poliomyelitis per 10,000 of population for the two epidemic years 1916 and 1947 was calculated for four ranges of ward density and the resulting figures have been set down in Tables 8 and 9.

For reasons already mentioned, the results of this investigation are probably not accurate, but taken as they are they suggest that there is no correlation between population density and case incidence in the City of Aberdeen in the two epidemic years 1916 and 1947.

Table 7. Municipal Ward Densities - City of Aberdeen

Municipal Ward	Ward Density based on population in 1911 & Ward area in 1913 (persons per acre)	Ward Density based on population in 1931 & Ward area in 1928 (persons per acre)
Torry	23.8	21.4
St. Clements	22.7	51.1
Greyfriars	153.1	104.9
St. Machar	11.7	14.2
Woodside	6.0	9.9
St. Nicholas	122.5	61.8
Rosemount	73.0	22.8
Rubislaw	21.2	13.8
Gilcomston		87.1
Holburn		67.0
Ruthrieston	19.6	24.3
Ferryhill	32.3	22.6
St. Andrews	59.2	

Table 8.

Relationship between Case Incidence in 1916 and population density			
Ward Density based on population in 1911 & Ward area in 1913 (persons per acre)	Total Cases	Total Population	Cases per 10,000 of Population
0 - 20	23	40,636	5.66
20 - 40	13	56,558	2.30
40 - 60	17	18,293	9.29
over 60	28	46,700	6.00

Table 9.

Relationship between Case Incidence in 1947 and population density			
Ward Density based on population in 1931 & Ward area in 1928 (persons per acre)	Total Cases	Total Population	Cases per 10,000 of Population
0 - 20	21	44,937	4.67
20 - 40	20	48,759	4.10
40 - 60	4	19,065	2.10
over 60	4	54,497	0.73

Age Incidence.

Bramwell (1908) has described a case of poliomyelitis in which the disease is said to have been present at birth, while Fox (1938) published the post mortem findings in a man of 68 years who died of the disease. In 1916, a boy of five weeks from the City of Aberdeen was notified as a case of poliomyelitis, and in 1934 a man aged 60 from Aberdeenshire was reported to have developed it. In the 1947 outbreak in Aberdeenshire, the youngest patient was a child of 11 weeks (Case 70) from the County of Aberdeen, and the oldest a woman of 40 (Case 75) from the City. Thus although the majority of cases belong to the lower age groups, the very young and the elderly cannot be regarded as being immune. The age incidence of the 401 cases occurring in the City and County of Aberdeen from 1913 to 1947 is seen from the following table.

Table 10.

Age in years	No. of cases	%
Under 1	47	11.72
1 - 4	189	47.13
5 -14	119	29.67
Over 14	46	11.48
	401	100.00

These figures make no allowance for the fact that all the cases notified in earlier years (including the epidemic one 1916) were paralytic, whereas of the 81 cases occurring during 1947, 31 were not paralysed. If the 257 paralytic cases which have occurred in the North East of Scotland since 1919 are separated into two groups comprising those from the town and those from the country, a distinct difference in age incidence becomes evident.(Table 11).

Table 11.

Age in years	City No. of cases	County No. of cases
0 - 4	81 (62.31%)	55 (43.31%)
5 - 14	38 (29.23%)	47 (37.01%)
over 15	11 (8.46%)	25 (19.68%)
	130(100.00%)	127(100.00%)

The figures show a higher percentage of children amongst those affected in the City than in the County. This is in accordance with a well recognised feature of the disease, which is that the age incidence of poliomyelitis varies inversely with the density of the population, being lower in more densely, higher in more sparsely populated

communities. The theory of latent immunisation explains this by assuming that in densely populated communities, which are in frequent communication with other communities, a continuous reservoir of infection is maintained. In more dispersed and isolated populations, the virus is less constantly present, being probably introduced from time to time, but not maintaining itself continually. Immunisation proceeds more rapidly and more regularly in the former, more slowly and more variably in the latter. The rate of immunisation, therefore, which depends upon the rate of spread of the virus through a population, appears then to be governed by the general principle of mass action; the more concentrated the population the faster is immunisation accomplished, the younger will be the average age of the susceptible population, and the earlier the age incidence of the clinical disease.

Sex Incidence.

The ratio between the sexes has varied somewhat in different outbreaks of poliomyelitis, but "the excess of males over females is one of the most constant epidemiological features of the disease".(International Committee, 1932). Forsbeck and Luther (1930) in the series of more than 7,000 cases occurring in Massachusetts from 1908 to 1929, with the exception of 1911 and 1917,

noted that in the epidemic years, the variation from the mean ratio of the sexes was very small. Only twice in the 19 years covered had the females outnumbered the males, and these two years had fewer total cases than any other year reported upon.

The sex incidence of the North East of Scotland cases for the years under review has been set out in Table 12 and expressed in graphic form in Figure 2. From these it may be seen that, with few exceptions, there are more males affected than females, and that this is true for division of the disease into urban and rural incidence, age incidence, and paralytic and non-paralytic forms. The overall total is 228 males and 173 females, giving a ratio of 1.32 to 1. The International Committee (1932) quote ratios, from 21 investigators, varying between 1.75 and 1.05 to 1 - the totals being 20,410 males and 15,591 females, a ratio of 1.30 to 1, which corresponds closely to that found in Aberdeenshire.

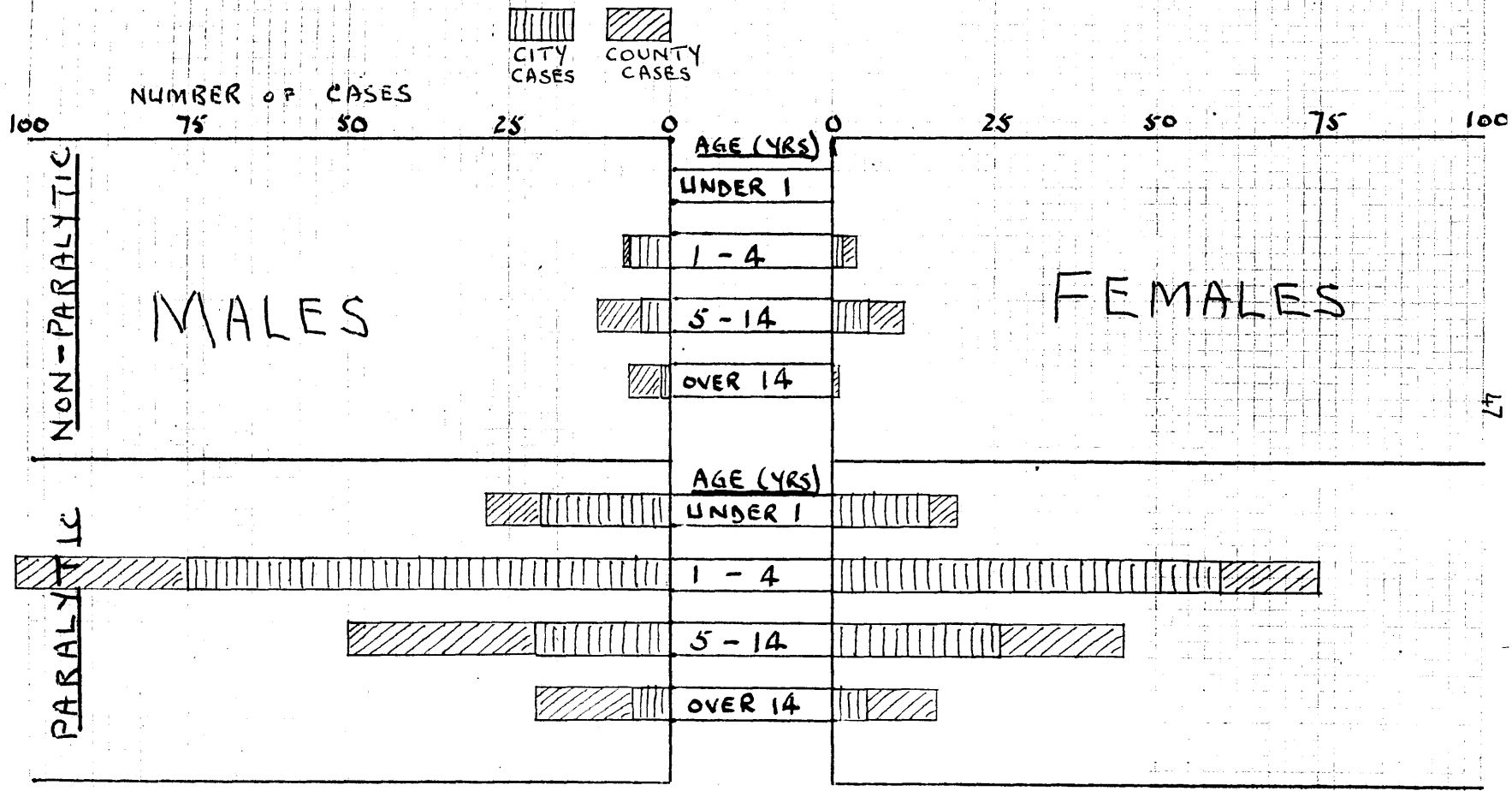
Sex Incidence of Poliomyelitis.

City and County of Aberdeen.

1919 to 1947.

Table 12.

Age in years	Non-paralytic				Paralytic				All cases				Grand Total		
	City		County		City		County		City		County				
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	Total
Under 1	-	-	-	-	20	15	8	4	20	15	8	4	28	19	47
1 - 4	6	2	1	2	75	60	28	15	81	62	29	17	110	79	189
5 - 14	4	6	8	6	21	27	29	18	25	33	37	24	62	57	119
over 14	1	-	6	1	6	7	15	10	7	7	21	11	28	18	46
Total	11	8	15	9	122	109	80	47	133	117	95	56	228	173	401



SEX INCIDENCE OF POLIOMYELITIS
CITY AND COUNTY OF ABERDEEN
1919 - 1947

Fig. 2.

Case Fatality.

One might expect that in years of epidemic prevalence, the case fatality in poliomyelitis would be high, but all the evidence in published work indicates that this is not so and that, in fact, the case fatality is, in general, inversely proportional to the number of cases. This has been attributed to differences in the reporting of non-paralytic cases in epidemic and inter-epidemic years. During an epidemic, the attention of both physicians and public is directed to the disease, which results in more complete recognition of its mild forms. In years of few cases, the milder forms are less often diagnosed, and the case fatality represents more nearly the ratio of deaths to paralytic cases. Furthermore, in recent years, the non-paralytic form of the disease has become increasingly recognised as such. For example, in the epidemic year 1916, all the cases notified in the City of Aberdeen were paralytic; in 1947, in 31 of the 81 cases notified in the City and County of Aberdeen, no evidence of paralysis was found.

Thus the question of the inclusion of the non-paralytic cases assumes importance in estimating mortality rates. In 1947, out of the 81 Aberdeenshire cases, there were six

deaths, giving a mortality rate of 7.4%. But when the non-paralytic cases are excluded, the mortality rate becomes 6 out of 50, or 12%. It has been impossible to ascertain exactly how many non-paralytic cases were included in the figures for past years in Aberdeen, as out of 320 cases, only 195 clinical notes have been found. From these, however, it has been discovered that in the County list, 4 non-paralytic cases were included in 1928, 1 in 1933 and 4 in 1944; in the City list 1 non-paralytic case was included in 1922, 1 in 1940 and 1 in 1941. In this discussion all these non-paralytic cases have been excluded.

The total yearly incidence of paralytic poliomyelitis in the City and County of Aberdeen, together with yearly deaths, are set out in Table 13. It shows that for the City (1913 - 1947) and the County (1919 - 1947) together the death rate is 32 out of 358 cases or 8.9%. When the City cases of 1913 - 1918 are excluded (there being, as explained on page 18 no figures obtainable for the County in these years) it becomes 27 out of 257 cases or 10.5%.

It is of interest to note that the death rate during the 1947 outbreak in North East Scotland - (12%) -

Paralytic poliomyelitis.

Table 13. Yearly Incidence and Deaths.

Year	Aberdeen City No. of cases	Aberdeen City No. of deaths	Aberdeen County No. of cases	Aberdeen County No. of deaths	Total no. of cases	Total no. of deaths
1913	4	-			4	-
1914	2	-			2	-
1915	2	-			2	-
1916	81	4			81	4
1917	10	-			10	-
1918	2	1			2	1
1919	1	1	1	-	2	1
1920	5	-	-	-	5	-
1921	8	2	4	-	12	2
1922	8	-	1	-	9	-
1923	-	-	-	-	-	-
1924	6	-	-	-	6	-
1925	5	-	2	-	7	-
1926	5	-	-	-	5	-
1927	4	-	1	-	5	-
1928	2	-	13	1	15	1
1929	2	1	3	-	5	1
1930	3	1	1	-	4	1
1931	1	-	2	1	3	1
1932	1	-	1	-	2	-
1933	10	1	10	-	20	1
1934	1	-	1	-	2	-
1935	1	-	1	-	2	-
1936	-	-	3	-	3	-
1937	1	1	3	1	4	2
1938	8	-	7	1	15	1
1939	1	-	4	-	5	-
1940	4	-	7	-	11	-
1941	1	-	8	1	9	1
1942	1	-	3	-	4	-
1943	1	-	3	-	4	-
1944	16	3	21	3	37	6
1945	-	-	3	2	3	2
1946	1	-	7	1	8	1
1947	33	6	17	-	50	6
Totals	231	21	127	11	358	32

is higher than (1) the overall death rate - (10.5%) - for the years 1913 - 1947, (2) the death rate in the 1916 City outbreak - (4.9%) - and (3) the death rate for England and Wales in 1947, which was 333 of 3,461 paralysed cases - (9.6%).(Bradley and Gale, 1948). This fact would appear to contradict the general finding, mentioned on page 48 that the case fatality is usually inversely proportional to the case incidence of the disease. The case fatality was therefore calculated for varying yearly incidences of the disease during the period 1913 to 1947, and the results have been tabulated. (Table 14) This shows that, in the years of low incidence, the mortality rate was in fact higher than in the years of high incidence.

Case Fatality in relation to Yearly Incidences.

1913 - 1947

Table 14.

Yearly Incidence (Cases per year)	No. of years involved	No. of cases	No. of deaths	Per cent. deaths
0 - 4	16	43	8	18.6
5 - 10	11	74	3	4.0
11 and over	8	241	21	8.7

Case Fatality in Urban and Rural Areas.

In a number of epidemics of poliomyelitis it has been stated that the case fatality was higher in the urban than in the rural cases. Frost (1913) reported that in Ohio, the case fatality in the City of Cincinnati was 38.9% while in the smaller towns and rural areas it varied from 9% to 18.2%. This aspect of the disease was closely examined by the International Committee (1932) which came to the conclusion that no satisfactory reason for it had been expounded in the literature, but suggested that one explanation was probably that in urban outbreaks the cases are more massed in the younger ages where the case fatality is highest.

In the 1947 outbreak under review, there were six deaths from poliomyelitis, and all were from the City of Aberdeen; none of the 32 County patients died. Taking the years 1919 to 1947 inclusive, an analysis shows that among 130 City patients there were 16 deaths (12.3%), and among 127 County cases, 11 deaths (8.6%). During this 29 year period, although the percentage of those affected under the age of 1 year is comparable in both City and County (10.9% and 9.4% respectively), there were 5 deaths amongst the City cases, and none in the County.

On the other hand, the percentage of children under 4 amongst those affected was 73.2% in the City whereas in the County it was only 38.4% - and 10 of the 21 City deaths and 2 of the 11 County deaths occurred in this age group. The higher death rate and the higher proportion of cases in this lowest age group in the City might therefore account for the fact that the total death rate is not higher in the County than in the City.

Case Fatality in relation to Age.

Age exerts a marked effect upon the outcome of cases of poliomyelitis. Van Rooyen (1940) states: " The (death) rate is considerably higher in the case of children under one year and in elderly persons". The high fatality rate in infants under one year was noted by Weisenberg (1917) who recorded figures from the Municipal Hospital, Philadelphia. These showed that, of 175 cases occurring there in this age group in 1916, 74 (42.2%) died.

In the North East of Scotland in 1947, 5 of the 6 deaths occurred in children under the age of 15, the other being a woman of 40. These numbers are too small for useful comment, but the death rates in relation to age groups for all the cases notified from 1913 to 1947

have been arranged in the following Table (15)

Table 15. Deaths from poliomyelitis in relation to Age Groups.

Age in years	No. of cases	No. of deaths
Under 1	47	5 (10.6%)
1 - 4	178	7 (3.9%)
5 - 14	95	12 (12.6%)
Over 14	38	8 (21.0%)
Total	358	32 (8.9%)

These figures illustrate the general principle for poliomyelitis that the case fatality is highest in infants undera year and in the older age groups, where the incidence is low, and lowest in the ages where the incidence is high. Nevertheless it seems surprising that even more infants do not die, in view of their proved high susceptibility to the disease. Perhaps this may be due to the rapid development of antibodies, or 'boosting' of inherited immunity once the virus enters the tissues. Moreover, the lower death rate in the 1 - 4 years age group - that in which the morbidity

figures are highest - might be explained by the fact that the individuals had amassed sufficient immunity through sub-clinical infection, in their earlier months or years, to prevent the disease from killing them, but insufficient to avert a full clinical attack. And yet it is difficult to understand why persons in the older age groups - particularly those over the age of 15 - who are relatively resistant to the disease, should succumb so readily once attacked. Possibly the reason is not that the virus acquires new powers of invasion, as may happen (Burnet, 1945), otherwise many other people in the same age group would be expected to be attacked also. Furthermore, it probably could not be due to an immunologically distinct strain of virus; this again would surely mean that many more people would succumb than actually do. It has been demonstrated experimentally that the development of immunity to the poliomyelitis virus is highly strain specific (Melnick and Horstmann, 1947) although this has not yet been proved to be the case for human beings. If, however, it is presumed that such an event does occur, then it might be argued that the immunity present in the members of a large community - such as Aberdeenshire - would

be against the same strain of virus in each individual. If, then, an immunologically different strain appeared, it is difficult to understand why more people in the higher age groups should not be attacked. A possible explanation may be that there occurs, in the individual himself, some circumstance which allows the virus to reach the central nervous system, and that this circumstance is present only on very rare occasions in the higher age groups.

SEASONAL INCIDENCE.

The seasonal prevalence of most infectious diseases is a very general epidemiological principle and the seasonal incidence of poliomyelitis has been recognised for many years. It appears to have been first noted by Sinkler in 1875. In an article on "Palsies of Children" he then wrote: " I observed two or three years ago that many of our cases of infantile palsy were said to have been attacked in the summer months, and since then I have carefully noted the time of year when the paralysis came on in each patient.... .... forty of fifty-seven cases were affected in the summer months. This fact has not, to my knowledge, been remarked before, and seems to me to have much bearing upon the causation of the disease".

Barlowe (1878) made an analysis of 63 cases of infantile paralysis observed in the course of seven years. In this he states: " of the influence of season we have strong evidence; of 53 cases, in which the date of attack could be fixed with accuracy, 27 occurred in the months of July and August, a fact worthy of notice as having a bearing on the primary causation of this affection".

In a general way the incidence of poliomyelitis, like that of diphtheria, decreases as warmer climates are approached. Northern Europe and the Northern United States of America, according to available reports, comprise the belt of greatest prevalence in the northern hemisphere. The countries of Southern Europe and the Southern United States have much less of the disease, and as the tropics are reached, poliomyelitis occurs more rarely.

In a study of the seasonal prevalence of infantile paralysis in the United States of America over a period of 11 years (1912 - 1922), Aycock et al. (1924a) found that there was a marked regularity in the summer prevalence of the disease and a definite secondary increase in its occurrence, usually in March and April. They considered that the regular occurrence of this increase from year to year, and with a certain degree of uniformity in all parts of the country, was suggestive of the possibility of two modes of transmission. These workers also reported (1924b) a marked seasonal variation in the ratio of the reported morbidity to the reported mortality of poliomyelitis and believed this to be due largely to failure to recognise the milder forms of the disease in its inter-epidemic

periods, and to a lesser extent, to delayed reporting of cases as compared with prompt reporting of deaths.

Although poliomyelitis has a markedly lower incidence in the southern part of the United States than in the northern, the age distribution of the disease is of the same order (Aycock, 1929). This might suggest that its comparatively rare occurrence in southern climates is not due to a correspondingly sparse distribution of the virus, but rather to a variation in the frequency with which the virus produces disease on the one hand or sub-clinical immunity on the other. Under such circumstances, the frequency with which disease or immunity without disease results from initial exposure to the virus may be due to variations in (1) dose of virus; (2) some inherent quality in the virus; or (3) some inherent quality in the host.

(1) Variations in the dose of virus transmitted in various climates is probably not a major factor. If warmer climates tended to diminish this dose, it is not possible to explain the increased prevalence of the disease in the warmer season of the year both in cooler and in warmer climates.

(2) No direct evidence is available concerning the virulence of the virus of poliomyelitis in different climates. When the clinical disease does occur in warm climates it compares in severity with the clinical disease in cooler regions.

Doull, Ferreiri and Perreiras (1927) have published data which indicate that the morbidity of measles, whooping cough, mumps and chicken pox in the tropics is equal to that in cooler climates, while scarlet fever and diphtheria both show a markedly diminished occurrence in the tropics, and their observations are in agreement with those of Rogers (1919) in India. Doull et al. present evidence that in diphtheria, carriers of virulent organisms are as common in the tropics as in cooler climates.

(3) Aycock (1929) suggests that seasonal and climatic fluctuations in the occurrence of poliomyelitis may have a relationship to corresponding effects of climate and season on the host, and that there may be some variation in the physiological activity of the body which influences resistance to poliomyelitis. For this inherent resistance he suggests the term "autarcesis" in contra-distinction to resistance provoked by the virus of the disease (immunity).

While the seasonal incidence of poliomyelitis is thus well recognised, there is very little evidence in the literature of attempts to relate this incidence to statistical data of weather conditions.

Temperature and precipitation in relation to the morbidity rates of infantile paralysis in 15 cities were studied by Toomey and August (1932). They found, for example, that in the town of Buffalo, New York, 1910, 1912, 1915, 1924, 1926, 1929 and 1930 were epidemic years; that 1912, 1915, 1926 and 1930 were years of low precipitation and nearly normal temperature; that 1924 had an increase in precipitation and a temperature slightly less than normal; and that the year 1929 was one of approximately average precipitation and temperature while 1910 showed clearly increased precipitation and normal temperature.

On the other hand the years 1911, 1913, 1914, 1918, 1919, 1921, 1922 and 1923 were all years of low precipitation and approximately normal temperature, with no increase in the number of cases.

Toomey and August concluded that there is no connection between changes of temperature and precipitation and the increase or decrease in the morbidity rate of poliomyelitis in the cities studied.

Nevertheless, in a statistical survey of poliomyelitis outbreaks in the city of New York, Bowerman (1945) showed that dry warm summers seemed frequently to be associated with outbreaks of the disease, but that the absence of rain seemed more significant than changes in mean temperature.

The unusual weather conditions during the epidemic period of poliomyelitis in 1947 prompted an investigation into a possible relationship between the incidence of the disease in the North East of Scotland during the period 1913 to 1947, and the weather conditions then prevailing. Statistical meteorological data were obtained from the records at the Air Ministry Meteorological Station at King's College, Aberdeen relative to the period under review.

The rainfall for each month was obtained along with the deviation which this showed from the average rainfall in the corresponding month of each of the previous fifty years, and is expressed in millimetres.

With regard to temperature, daily maximum and minimum readings are recorded at the Meteorological Office. The mean for each month of the daily maximum temperatures is calculated, with a similar mean of the minimum temperatures. The mean of these two figures is then taken as the monthly

mean temperature and its deviation from the average figure in the corresponding month of each of the previous fifty years is expressed in degrees Fahrenheit.

This monthly deviation of rainfall and temperature from the average is shown in graphic form for each year from 1913 to 1947 together with the number of cases of poliomyelitis in the City and County of Aberdeen notified in each month. (Figs. 3 - 11)

From this it will be seen that there is no constant relationship apparent between the occurrence of cases of poliomyelitis on the one hand, and temperature and rainfall readings on the other. Comparing the two epidemic years - 1916 and 1947 - in particular, it can be seen that, in the latter part of each year corresponding to the notifications of poliomyelitis, the rainfall was, except for two months, above the average, while in 1947 the rainfall was, in each month when cases occurred, below the average. Similarly, the monthly mean temperature in 1916 was, for the most part, below the average while in 1947 it was, during the epidemic months, entirely above the average.

If, therefore, meteorological conditions play any part in the productions of epidemics of poliomyelitis, it is not made evident by the temperature and rainfall figures for Aberdeenshire during the past 35 years.

Relationship between Incidence of Poliomyelitis and Temperature and Rainfall

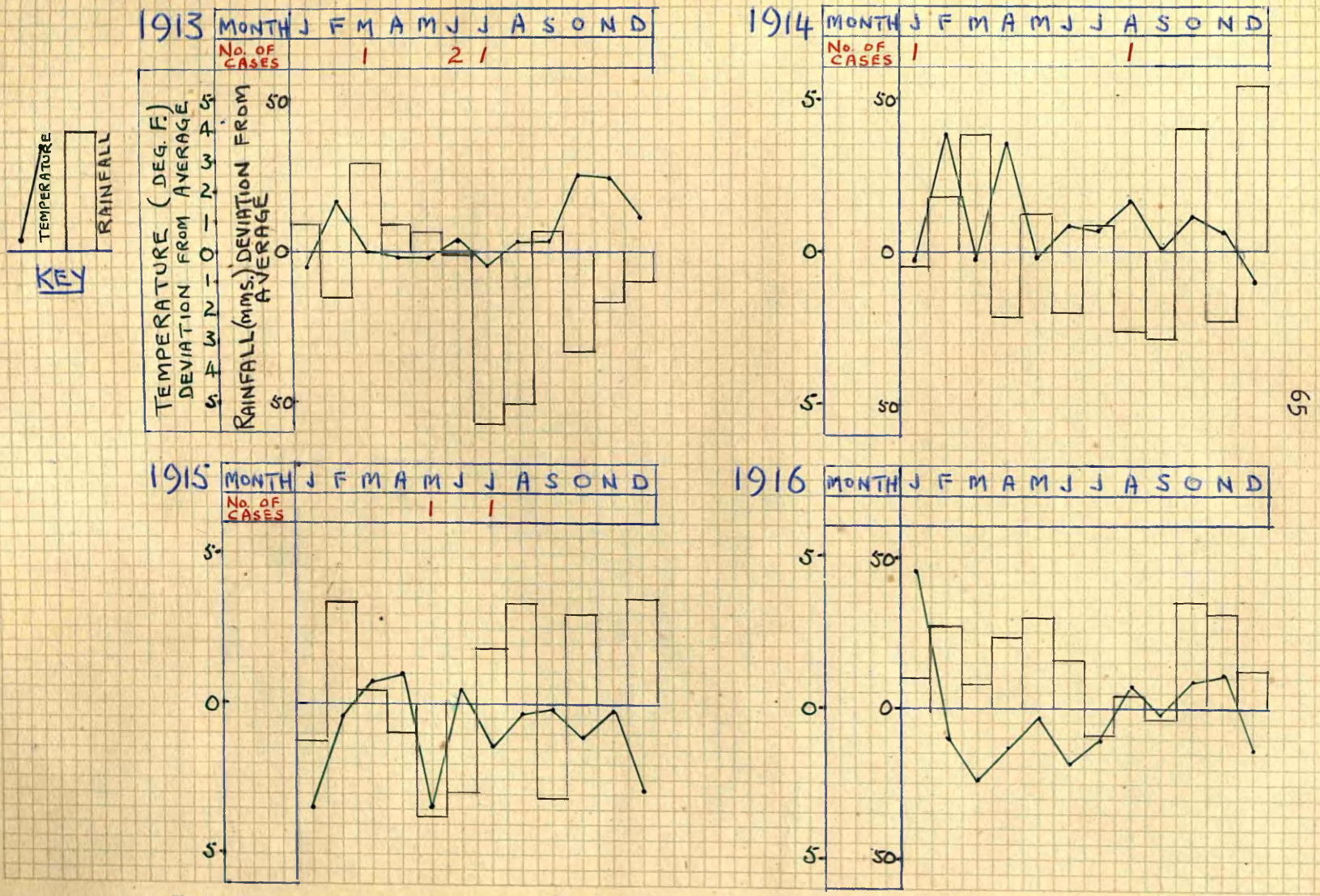


Fig. 3.

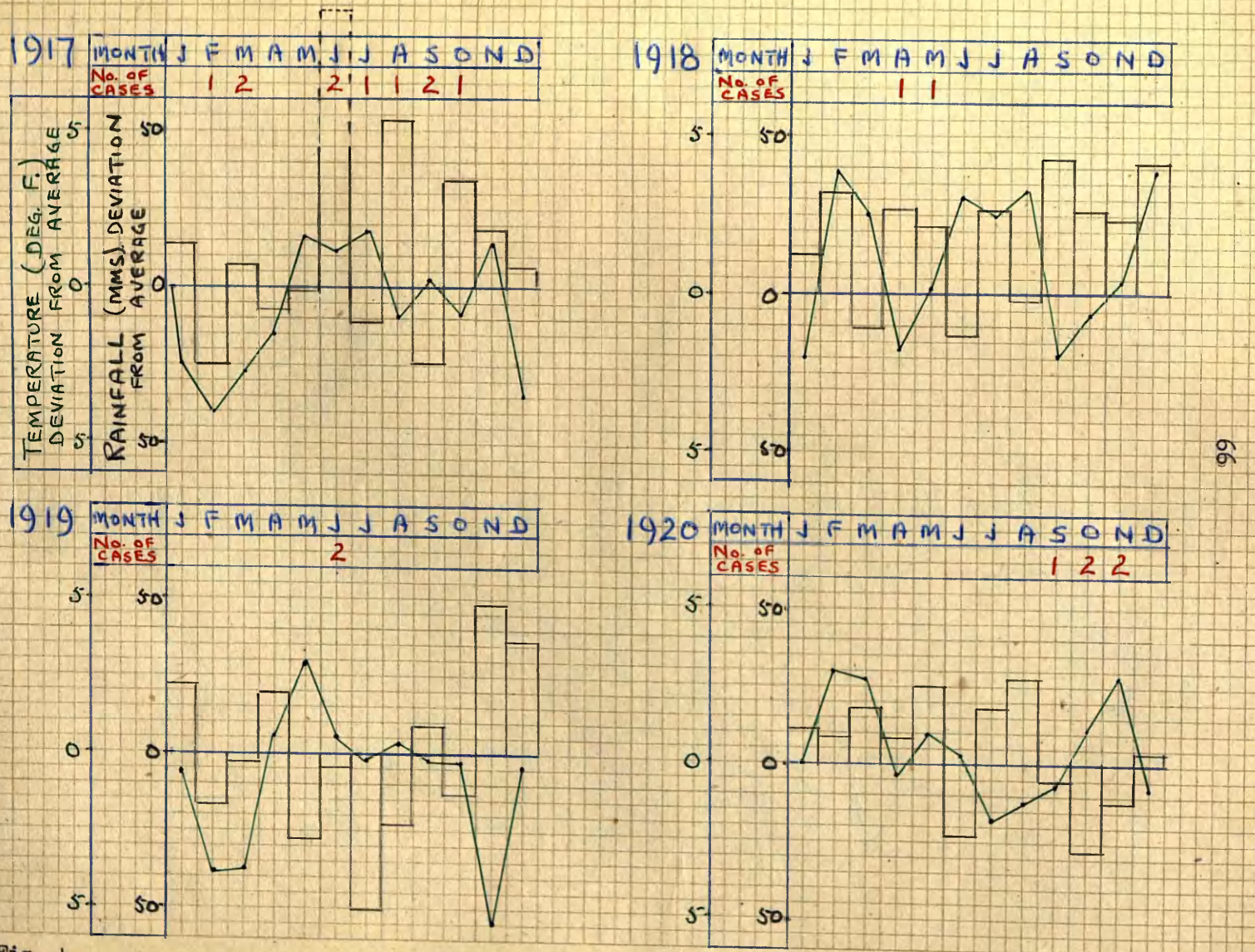

1917
MONTH J F M A M J J A S O N D
No. OF CASES 1 2 2 1 1 2 1
TEMPERATURE (DEG. F.) DEVIATION FROM AVERAGE
RAINFALL (MMS.) DEVIATION FROM AVERAGE
5
0
5
50
0
50
1918
MONTH J F M A M J J A S O N D
No. OF CASES 1 1
1919
MONTH J F M A M J J A S O N D
No. OF CASES 2
1920
MONTH J F M A M J J A S O N D
No. OF CASES 1 2 2

Fig. 4.

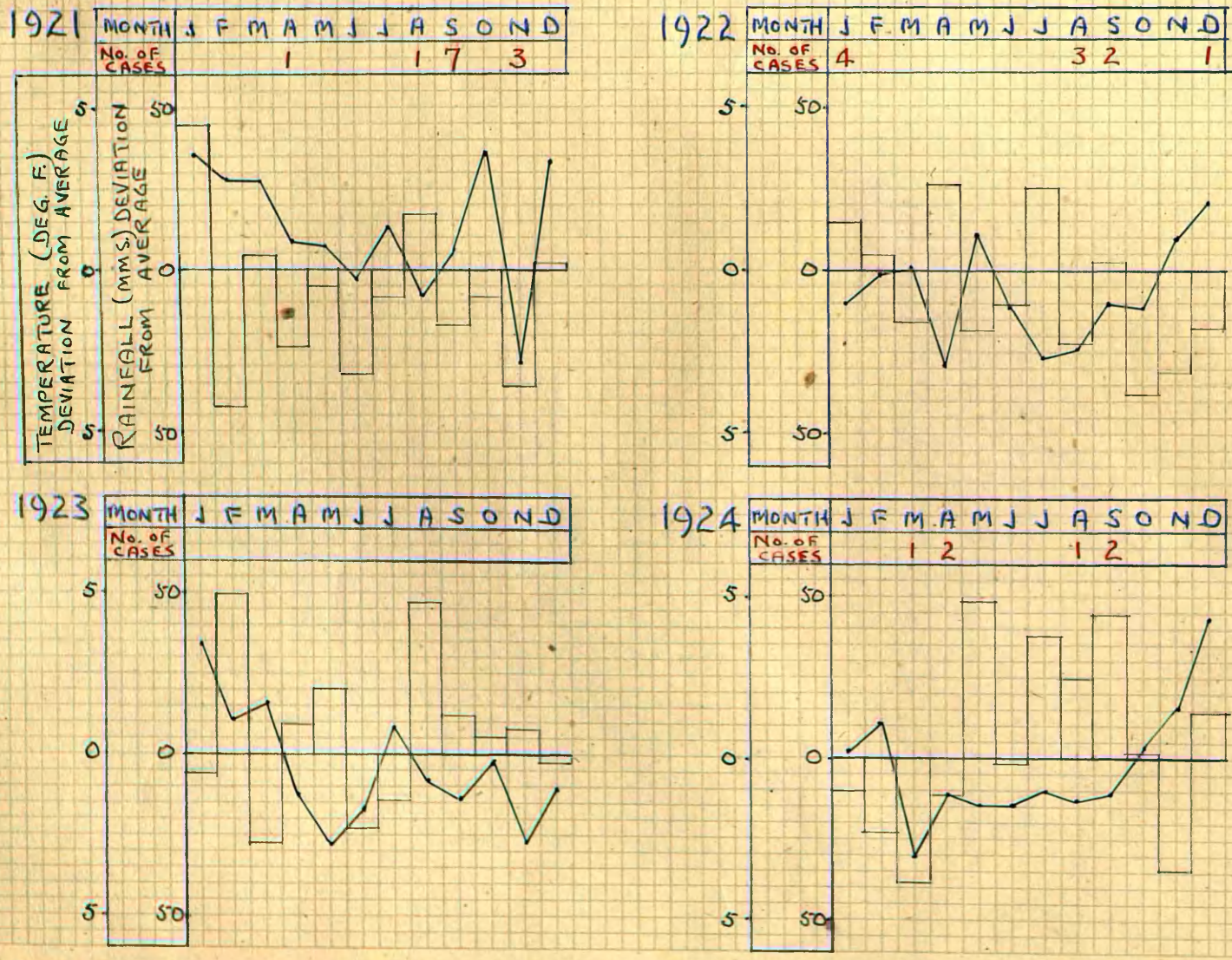

1921
MONTH J F M A M J J A S O N D
No. OF CASES 1 1 7 3
TEMPERATURE (DEG. F.) DEVIATION FROM AVERAGE
RAINFALL (MMS.) DEVIATION FROM AVERAGE
5 0 5
50 0 50
1922
MONTH J F M A M J J A S O N D
No. OF CASES 4 3 2 1
1923
MONTH J F M A M J J A S O N D
No. OF CASES
1924
MONTH J F M A M J J A S O N D
No. OF CASES 1 2 1 2

Fig. 5.

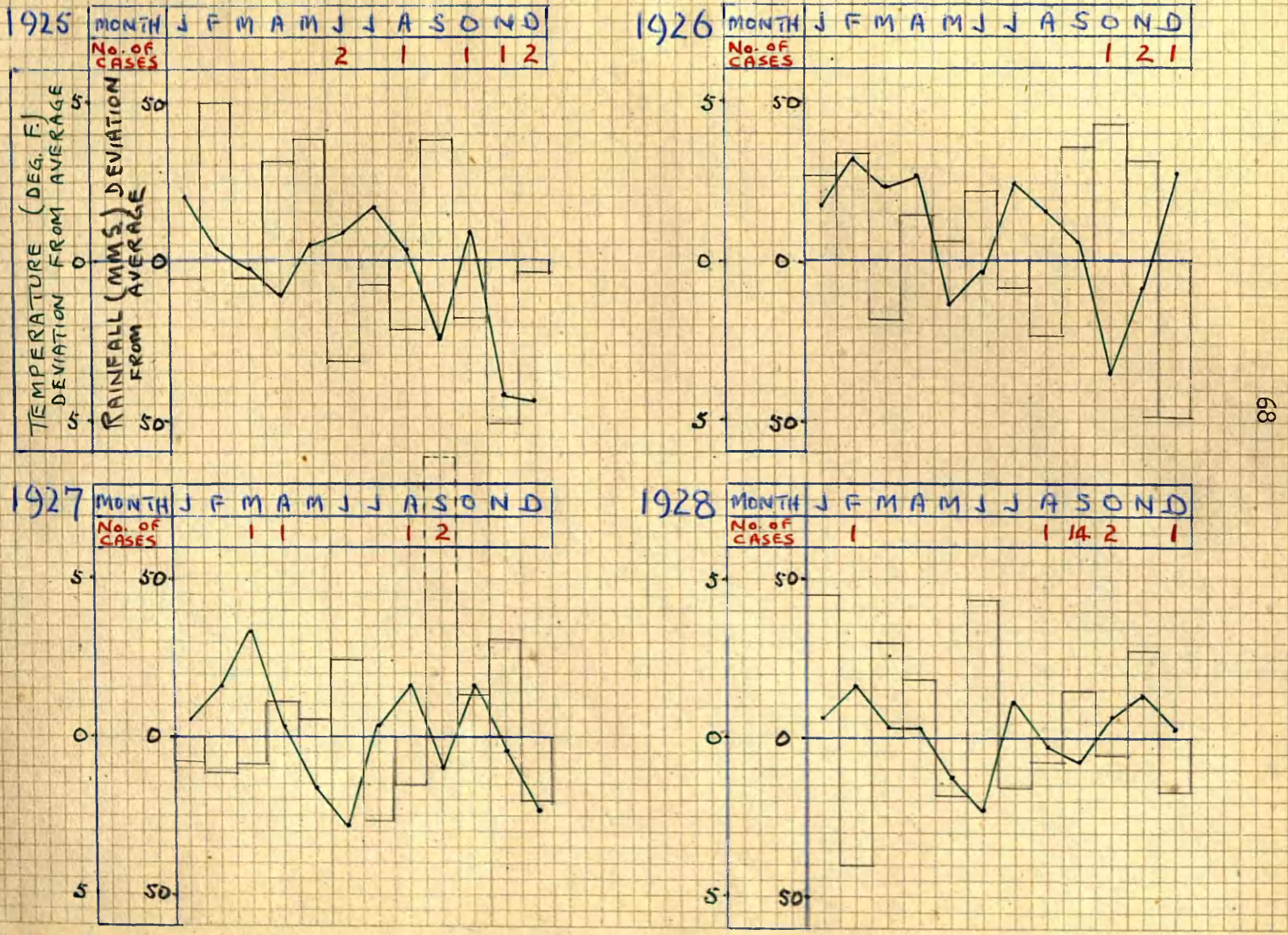

1925
MONTH J F M A M J J A S O N D
No. of CASES 2 1 1 1 2
TEMPERATURE (DEG. F.) DEVIATION FROM AVERAGE
RAINFALL (MMS.) DEVIATION FROM AVERAGE
5 0 5
50 0 50
1926
MONTH J F M A M J J A S O N D
No. of CASES 1 2 1
5 0 5
50 0 50
1927
MONTH J F M A M J J A S O N D
No. of CASES 1 1 1 2
5 0 5
50 0 50
1928
MONTH J F M A M J J A S O N D
No. of CASES 1 1 1 4 2 1
5 0 5
50 0 50

Fig. 6.

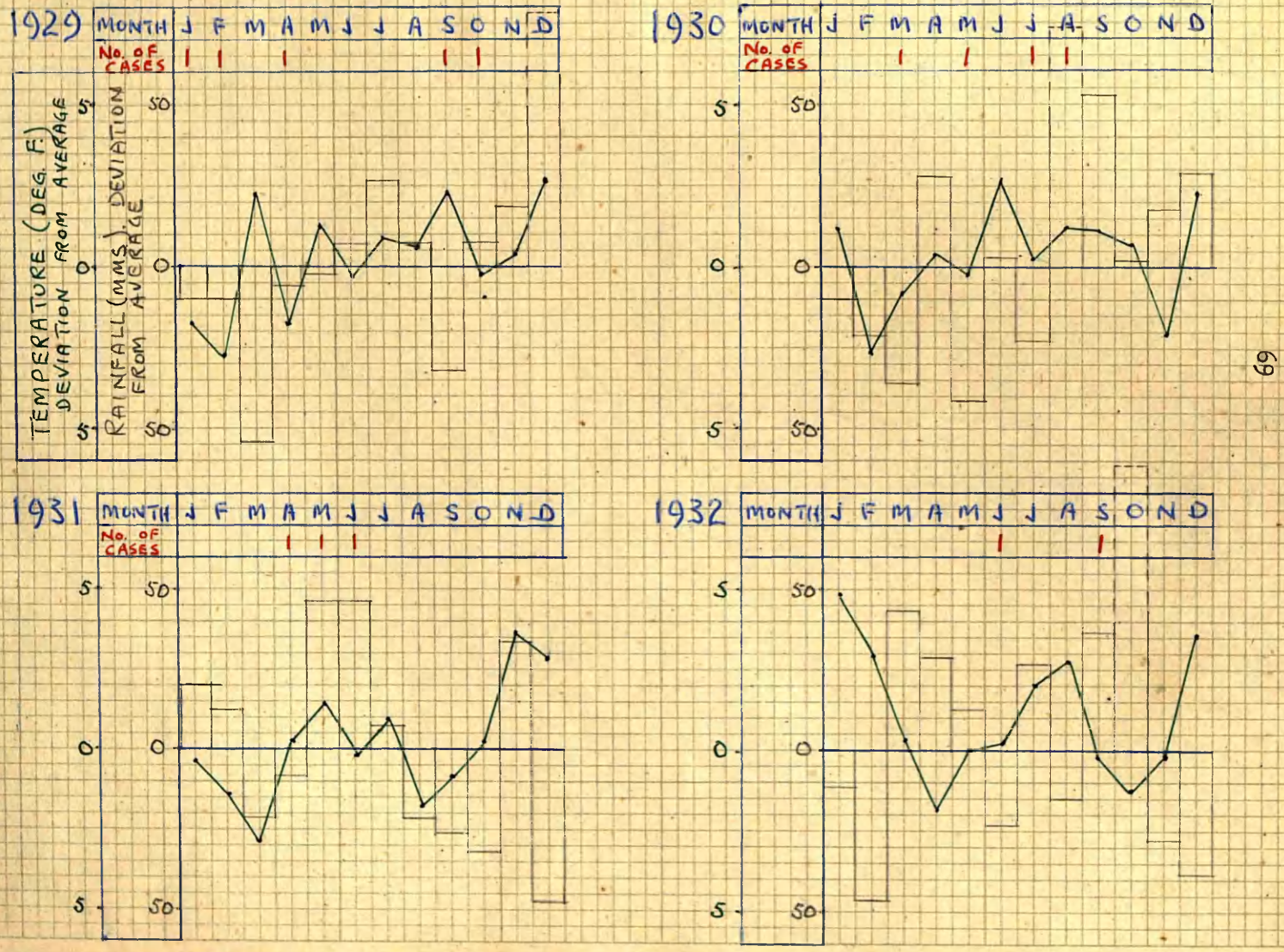

1929
MONTH J F M A M J J A S O N D
No. OF CASES 1 1 1 1 1
TEMPERATURE (DEG. F.) DEVIATION FROM AVERAGE
RAINFALL (MMS.) DEVIATION FROM AVERAGE
5 0 5
50 0 50
1930
MONTH J F M A M J J A S O N D
No. OF CASES 1 1 1 1
5 0 5
50 0 50
1931
MONTH J F M A M J J A S O N D
No. OF CASES 1 1 1
5 0 5
50 0 50
1932
MONTH J F M A M J J A S O N D
1 1
5 0 5
50 0 50

Fig. 7.

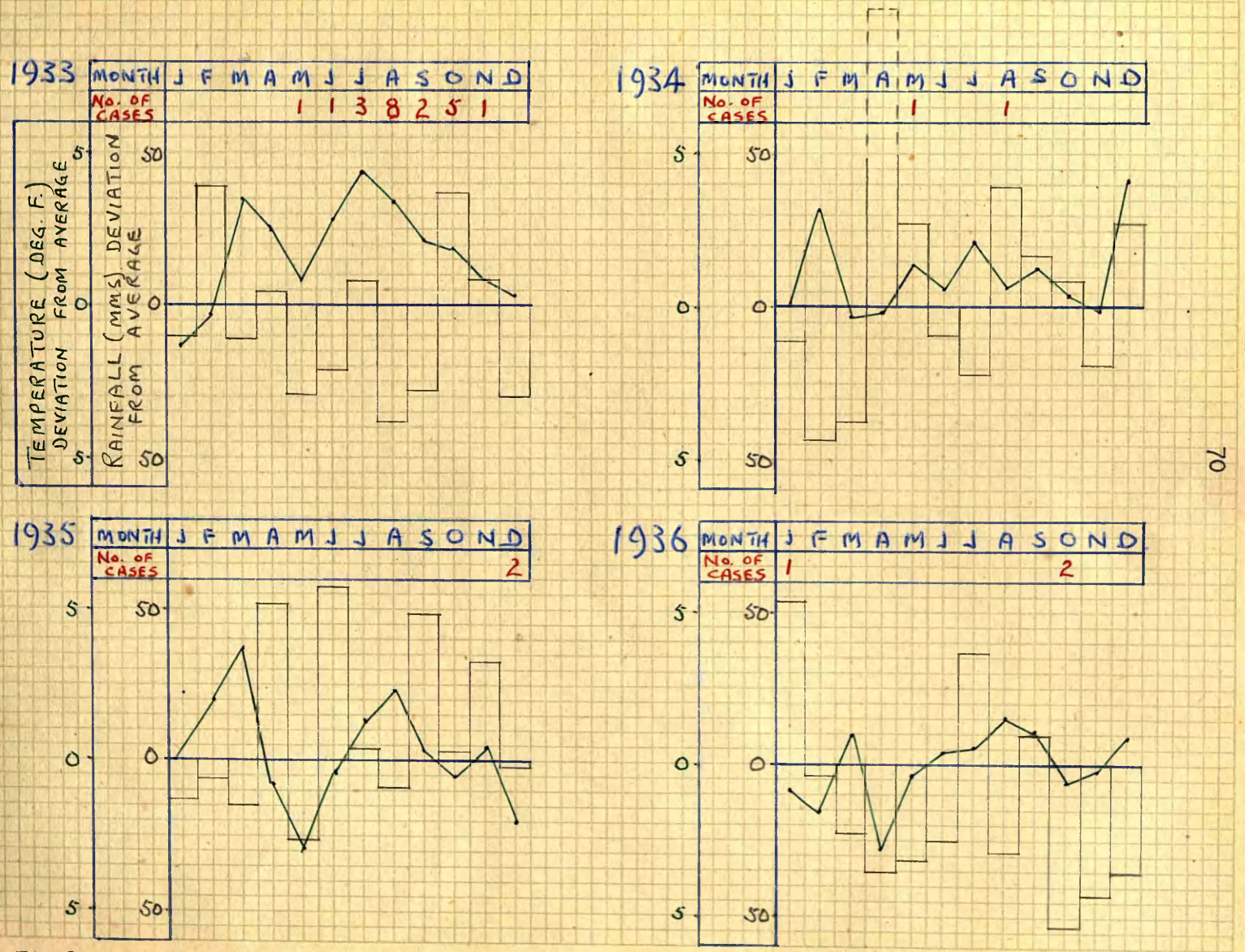

1933
MONTH J F M A M J J A S O N D
No. OF CASES 1 1 3 8 2 5 1
TEMPERATURE (DEG. F.) DEVIATION FROM AVERAGE
5 0 5
RAINFALL (MMS) DEVIATION FROM AVERAGE
50 0 50
1934
MONTH J F M A M J J A S O N D
No. OF CASES 1 1
5 0 5
50 0 50
1935
MONTH J F M A M J J A S O N D
No. OF CASES 2
5 0 5
50 0 50
1936
MONTH J F M A M J J A S O N D
No. OF CASES 1 2
5 0 5
50 0 50

Fig 8.

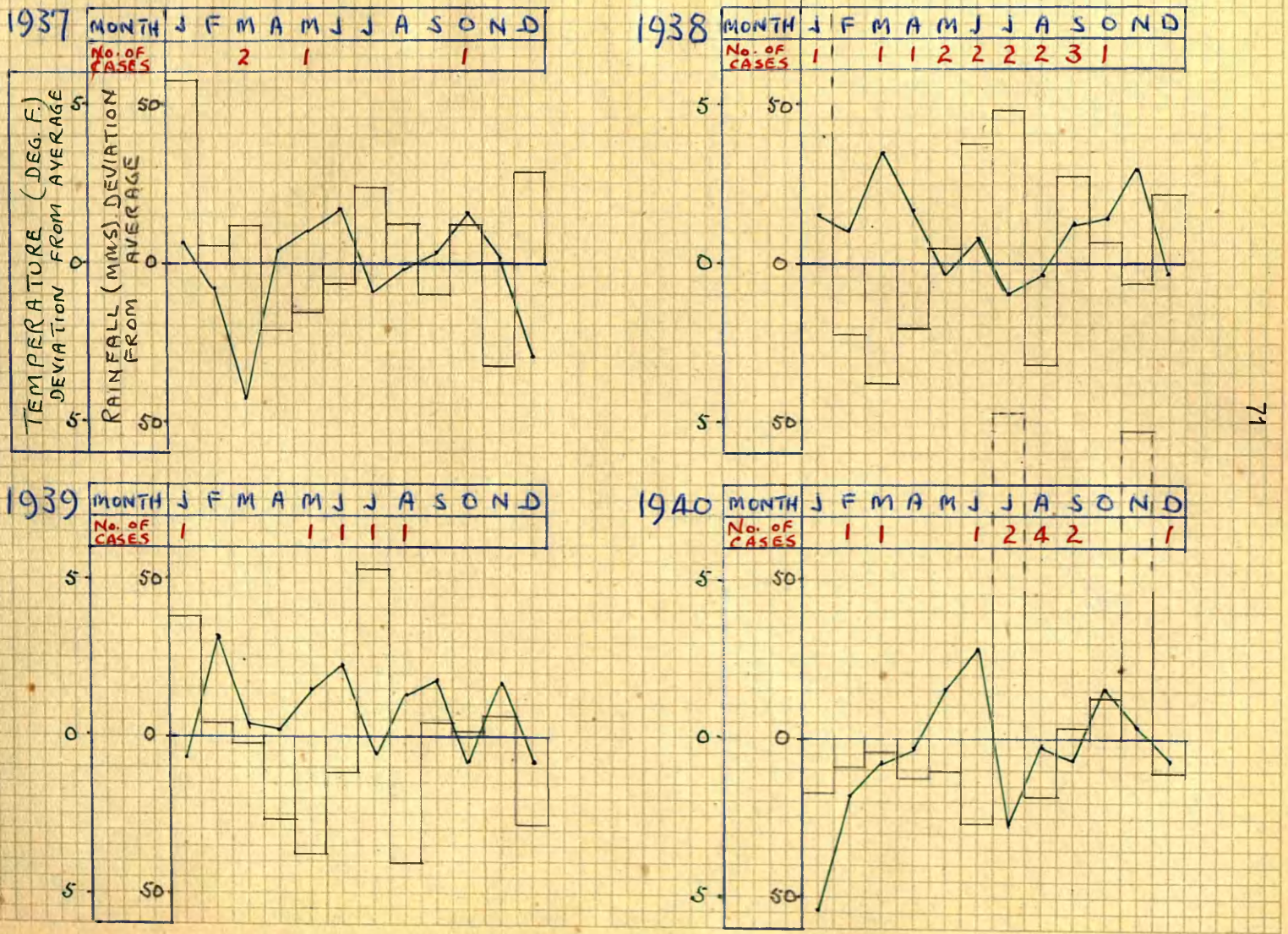

1937
MONTH J F M A M J J A S O N D
No. OF CASES 2 1 1
TEMPERATURE (DEG. F.) DEVIATION FROM AVERAGE
RAINFALL (MMS.) DEVIATION FROM AVERAGE
5 0 5
50 0 50
1938
MONTH J F M A M J J A S O N D
No. OF CASES 1 1 1 2 2 2 2 3 1
1939
MONTH J F M A M J J A S O N D
No. OF CASES 1 1 1 1 1
1940
MONTH J F M A M J J A S O N D
No. OF CASES 1 1 1 2 4 2 1

Fig. 9.

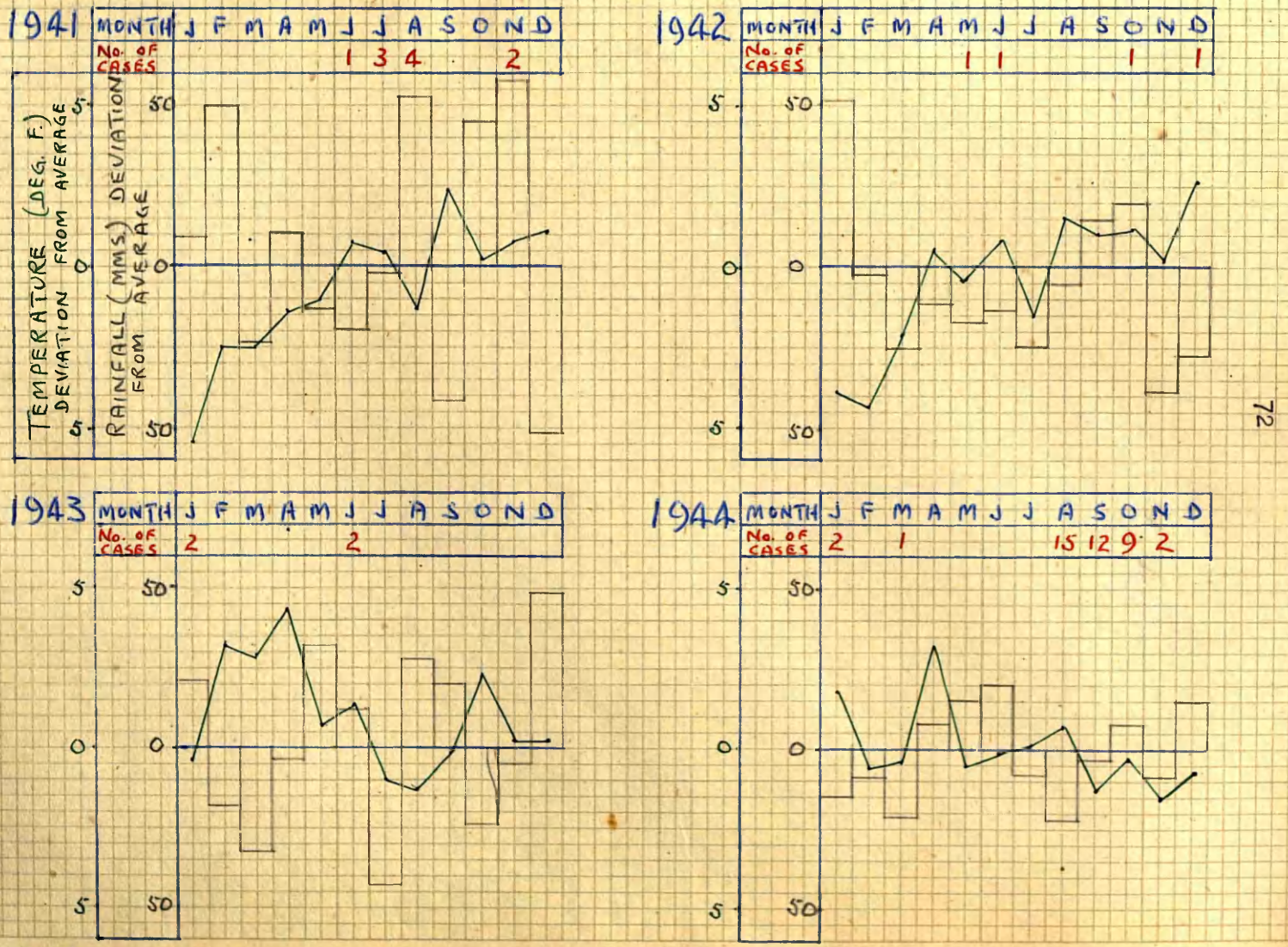
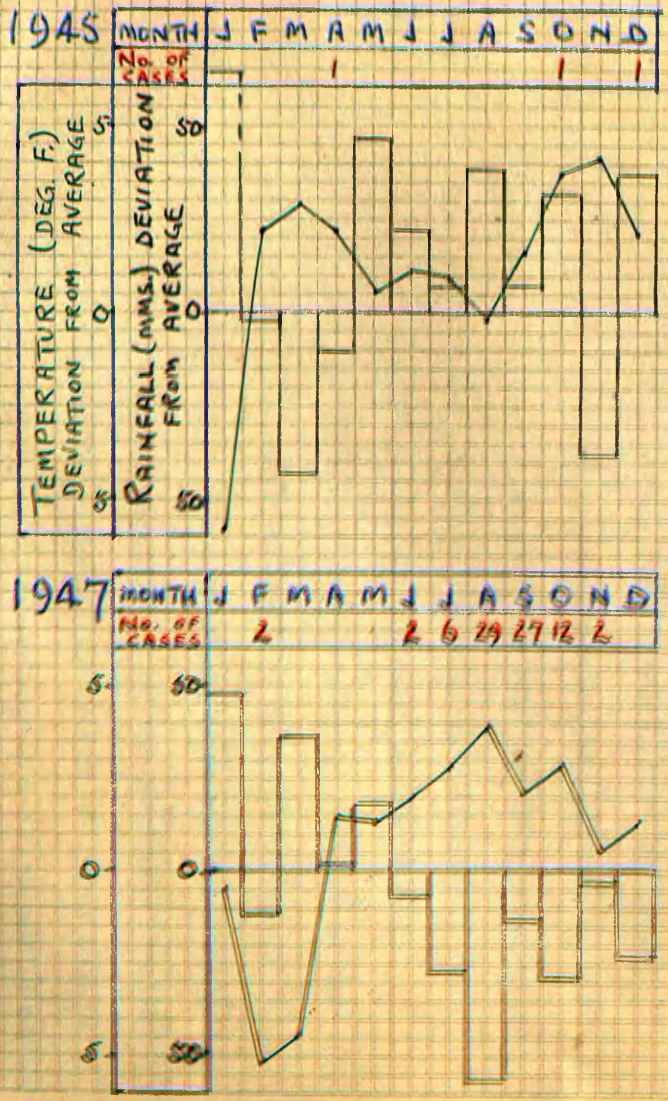

1941
MONTH J F M A M J J A S O N D
No. OF CASES 1 3 4 2
TEMPERATURE (DEG. F.) DEVIATION FROM AVERAGE
RAINFALL (MMS.) DEVIATION FROM AVERAGE
5 0 5
50 0 50
1942
MONTH J F M A M J J A S O N D
No. OF CASES 1 1 1 1
1943
MONTH J F M A M J J A S O N D
No. OF CASES 2 2
1944
MONTH J F M A M J J A S O N D
No. OF CASES 2 1 15 12 9 2

Fig 10.


1945
MONTH
J F M A M J J A S O N D
No. of CASES
1 1 1
TEMPERATURE (DEG. F.) DEVIATION FROM AVERAGE
RAINFALL (MMS.) DEVIATION FROM AVERAGE
5
0
5
50
0
50
1947
MONTH
J F M A M J J A S O N D
No. of CASES
2 2 6 29 27 12 2
5
50
0
0
5
50

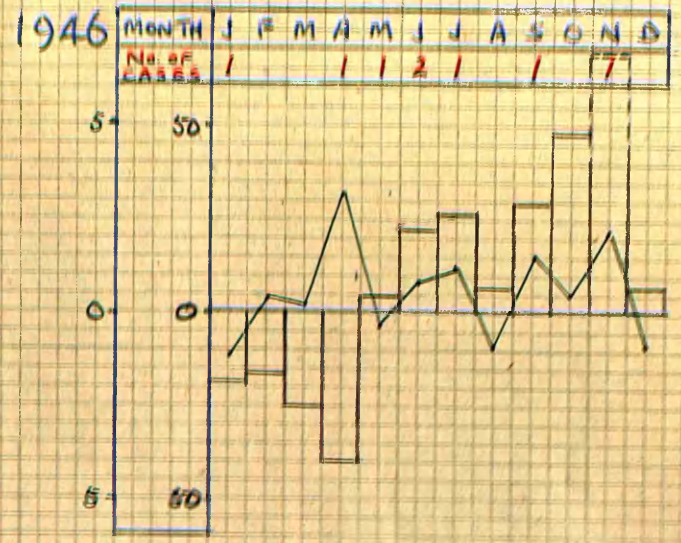
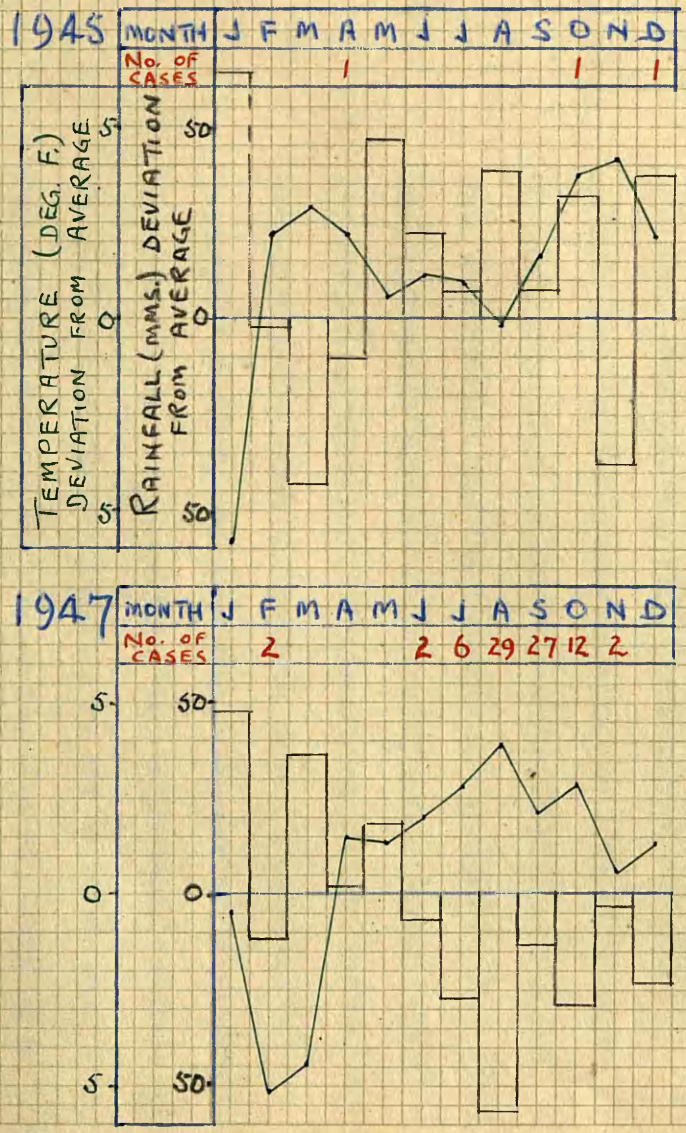
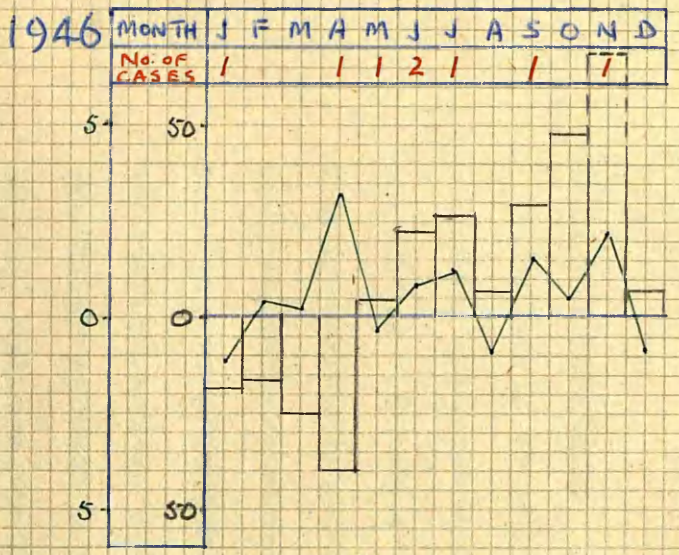

1946
MONTH
J F M A M J J A S O N D
No. of CASES
1 1 1 2 1 1 7
5
50
0
0
5
50

Fig. 11.


1945
MONTH J F M A M J J A S O N D
No. OF CASES 1 1 1
TEMPERATURE (DEG. F.) DEVIATION FROM AVERAGE
RAINFALL (MMS.) DEVIATION FROM AVERAGE
5 0 5
50 0 50


1946
MONTH J F M A M J J A S O N D
No. OF CASES 1 1 1 2 1 1 1
5 0 5
50 0 50

1947
MONTH J F M A M J J A S O N D
No. OF CASES 2 2 6 29 27 12 2
5 0 5
50 0 50

Fig. 11.

Contact between Cases.

The lack of obvious connection between cases is a striking and constant feature in the epidemiology of the disease. In only a small percentage of cases is it usually possible to obtain a history of exposure to a case, and while connected cases do occur, infection never seems to proceed regularly from cases to contacts in a well-defined series. Halliday (1929) reported that in the Glasgow epidemic in 1928, comprising 100 cases, in only two instances, or 2%, was it possible to trace a direct association among cases.

With regard to the 1947 Aberdeenshire outbreak, special care was taken in eliciting the history of each patient's illness to enquire for any possible association with other cases, and the findings are summarised for each individual case in the Appendix, under "Recent Activities and Contacts". These showed that it is the exception rather than the rule to obtain a clear cut history of contact, and also that, when one was obtained, there was often the possibility of the patients involved having a common and unidentified source of infection. For example, Cases 1 & 2, Cases 10, 12 & 13, Cases 25 & 31 and Cases 42 & 44 were very closely associated with each other, but there may well have been a common source

of infection for each group. Apart from these patients, there were 7 others in which histories of direct contact were obtained; in one of these (Case 7) the connection was very doubtful, and in another (Case 16) there was the possibility of a common source of infection with a patient who was treated in Edinburgh. In 12 of the patients there was a history of indirect contact with other proved cases - i.e. some close associate of the patient had been in contact with a proved case about the time that the latter's illness began.

The possibility of calculating the incubation period of the disease in those cases where a history of direct or indirect contact with a known case was elicited, was considered. The difficulties of this procedure are stressed by Aycock et al. (1929) who point out that in family outbreaks there is always the possibility of simultaneous infection; that milk-borne outbreaks, and those precipitated by tonsillectomy, may have quite separate incubation periods; and that the various clinical types of the disease probably have different ones again. However, they thought that the incubation period lay between 6 and 20 days. Wickman (1913)

on the other hand, thought that it was from 6 to 10 days. Moreover, estimations of the incubation period from histories of contact would appear to be of little value in the light of the work of Brown et al. (1945) who demonstrated the presence of the virus in the stool of a patient 19 days before the onset of paralysis.

Nevertheless, wherever reasonable deductions could be made on the grounds of direct or indirect contact, the incubation periods of the 1947 Aberdeenshire cases were calculated and are set down in the Appendix Summary and in tabular form in Tables 16 and 17. Histories of contact with a minor illness wich might have been "abortive" poliomyelitis, have been excluded from the tables. They show that out of 81 cases, in only 17 (21%) was it possible to calculate an incubation period from a history of contact with proved cases of the disease.

I feel that the evidence on which these figures are based is so circumstantial as to be of little value and my impression in this respect of the cases under review is that the disease was probably not often spread by contact between proved cases. In this connection it is of interest to note that Sweetman (1948)

Poliomyelitis in Aberdeenshire - 1947.

Table 16. Possible Incubation Periods of Poliomyelitis cases as calculated from a history of Direct Contact with a known case of the disease.

Case No.	Incubation Period.
1	10 days
12	2 or 6 days
13	4 days
31	4 days
44	3 days
51	4 - 5 days
60	22 days
67	9 days

Table 17. Possible Incubation Periods of poliomyelitis cases as calculated from a history of Indirect Contact with a known case of the disease.

Case No.	Incubation Period.
17	9 days
26	7 - 14 days
30	3 days
52	12 days
68	14 days
70	16 days
73	16 days
75	1 day
80	2 months +

and Lavinder et al. (1918) found contact histories in 20% and 30% of their cases respectively, while Lowe (1912) made this finding in only 3 of 62 cases (4.8%). All these investigators excluded abortive or non-paralytic cases from their series.

If, however, minor illnesse of contacts of patients are considered to be mild non-paralytic attacks of poliomyelitis, the association of cases becomes clearer. Histories of such minor illnesses were obtained in 14 of the 81 Aberdeen cases, and if these and all the patients with direct and indirect contacts are added, it is found that 36 (44.4%) were units of multiple groups of patients with the disease. That all the minor illnesses were in fact poliomyelitis is unlikely, but there is evidence that in some cases at least, this might be so. Casey et al. (1945) showed that minor illnesses are 4 - 6 times more common amongst contacts of known cases than amongst controls, while McAlpine et al. (1947) found histories of illness of contacts in 19 of 54 patients and of 8 of them admitted to hospital, 3 had pleocytosis of the cerebro-spinal fluid. Many of the patients admitted to hospital in Aberdeen in 1947 with non-paralytic poliomyelitis had very mild systemic

disturbances and for very short periods, and it is felt that there must have been many other such cases who were never admitted to hospital. Experimental evidence has shown that the virus may be present in contacts without even minor upset. Howe and Bodian (1947) and Gordon et al. (1947) have demonstrated its presence in the throats, and Brown et al. (1945) in the stools, of healthy contacts.

It is suggested then, on epidemiological and experimental evidence, that the disease is spread by contact, probably mostly from healthy carriers, or by those whose immunity permits of only minor or abortive attacks of the disease, but two further points may be illustrated from the 1947 outbreak in Aberdeen. In the first place, in two cases (Nos. 29 and 72), there was definite proof that three years previously each had been in close contact with a paralytic case of the disease and neither had had any symptoms at the time. Another patient (No. 18) had been a close contact of a case of "aseptic meningitis", which might well have been a case of non-paralytic poliomyelitis, eleven months previously, and yet did not develop poliomyelitis at that time. Secondly, the fact that some extremely close associates

do not develop the disease is illustrated by Case 54, who had an identical twin sister. The two were always together, even sleeping in the same bed, yet one contracted the disease and the other did not.

Institutional outbreaks of poliomyelitis are rare, and certainly there were no secondary cases amongst the patients or staff at the City Hospital, Aberdeen in 1947; while, of the patients admitted to the Hospital with the disease, 30 were attending 25 different schools or similar institutions at the time of onset of their symptoms. That institutional outbreaks of the disease can occur was proved by Aycock (1927) who instanced 34 cases in schools during the Broadstairs epidemic in 1926, and by the International Committee (1932) who quote 3 out of about 300 nurses in a hospital in Massachusetts contracting it in 1927. On the other hand, Batten (1916), in 30 years at Great Ormond Street, observed no secondary cases amongst patients or staff. It may be mentioned here, however, that two highly localised outbreaks of poliomyelitis have appeared in the North East of Scotland since notification became compulsory. The first of these was in 1928, when, out of a total of 17 cases notified from the County of Aberdeen, no less than 13 came from Fyvie, and are depicted on the County map.

Of the 13 cases, 3 were from one family and 2 from another, but no such close connection could be traced between the others. Six of the patients attended three Fyvie schools and, according to the Report of the Medical Officer of Health for that year, there had apparently been an "influenzal cold epidemic" amongst the school children about six weeks prior to the recognition of the first case of poliomyelitis. In the second localised outbreak, in 1941, out of 8 notifications from the County of Aberdeen, 4 were from the village of Collieston - 2 being brother and sister.

From these observations in relation to contacts of poliomyelitis, it appears to me that histories of direct among those admitted to hospital with the disease are rare enough to suggest that there must be some other reservoir of infection present in the community. It seems probable, from the evidence in the literature, that this reservoir consists of healthy carriers and people who have very mild attacks of the disease. The fact that institutional outbreaks and secondary cases amongst the very closest of contacts seldom occur, suggests that a widespread though somewhat irregular immunity to the disease is present amongst the population.

The question of Swimming Baths as a factor in the spread of the disease.

A good deal of interest was aroused in both the medical and the lay press during the 1947 epidemic of poliomyelitis as to whether the disease could be contracted in public swimming baths. It has been shown by Sabin (1947) that the virus is present in the stools and throats of apparently healthy people, as well as paralysed people, during epidemics, and there seems no reason whatever to doubt that it could gain access to the water. The question arises as to whether or not the virus can survive in the chlorinated water of the baths. In Aberdeen, at the Justice Mill Lane Baths and the Beach Baths - neither of which were closed to the public during the 1947 epidemic - the chlorine content of the water is not less than 0.2 parts per million, and not more than 0.5 parts per million (figures obtained from the City Engineer's Department). Levatiti et al. (1931) showed that 0.4 mgms. per litre (i.e. 0.4 parts per million) sterilised water which was heavily contaminated with the virus. Thus the chlorine-treated water in the Aberdeen Baths would probably kill the virus quite satisfactorily.

Of the 81 Aberdeenshire cases of poliomyelitis in 1947, five gave a history of having bathed in one of the two Aberdeen Swimming Baths and a further two of having attended other Swimming Pools - all within a period of 3 weeks before development of their first symptoms. The details are as follows:

Attended Justice Mills Lane Baths, Aberdeen.

Case 7 - on the day of onset of symptoms.
Case 48 - 13 days before first symptoms.
Case 80 - 3 weeks before first symptoms.

Attended Beach Baths, Aberdeen.

Case 20 - 3 weeks before first symptoms.
Case 54 - 2 weeks before first symptoms.

Attended Stonehaven Swimming Pool.

Case 51 - 2 weeks before first symptoms.

Attended Inverness Swimming Baths.

Case 67 - 13 days before first symptoms.

Of these seven cases only one (No. 7) developed paralysis. It seems most unlikely that the three who attended the Justice Mill Lane Baths , or the two who went to the Beach Baths, were in any way connected, in view of the time intervals and the poor chance of survival of the virus.

It also seems unlikely that Swimming Baths in general can be a potent source of infection, in view of the number of people who attend them and yet do not develop the disease. Between 1st July and 30th November, 1947, 87,285 people paid for admission to the Justice Mills Lane Baths, Aberdeen, and during the same period 40,406 attended the Aberdeen Beach Baths. Some of these people must have attended two or more times, but even so, it seems likely that more than 5 would have contracted poliomyelitis if the Baths had been a factor in the spread of the disease.

The question of Tonsillectomy as a factor in the spread of the disease.

Operations upon the nose and throat have been considered to have a causal relationship to the development of poliomyelitis, and the occurrence of the disease following tonsillectomy has been reported on several occasions. A striking example is given by Francis et al. (1942), who observed the disease in five children of one family 12 - 14 days after their tonsils had been removed. Three of the children died.

In Aberdeenshire, as elsewhere, during the 1947 outbreak, tonsillectomy operations were postponed and can therefore be excluded as an etiological factor in the disease. In point of fact, none of the 81 cases admitted to hospital gave histories of recent operations upon either nose or throat.

THE CEREBRO-SPINAL FLUID.

The 1947 Aberdeenshire cases of poliomyelitis have been classified into four types (page 22) in relation to the occurrence or otherwise of paralysis and to the severity of the paralysis when present. In this section I have analysed the findings in the cerebro-spinal fluid in relation to these four types of disease. This was done with a view to determining if possible whether any prognostications as to the outcome of the disease could be made from an examination of the fluid on the patient's admission to hospital.

The cerebro-spinal fluid was examined in all cases and as soon as possible after admission, and the findings for each type of the disease have been set down in Tables 18 - 21 (Case 37 has been omitted as the fluid obtained on admission was unsuitable for examination.) The constituents obviously vary during the course of the disease in any single patient, but the aim of the present investigation was to discover whether there was anything characteristic at first examination in relation to the four types of disease.

Analysis of the cerebro-spinal fluid

in type of disease 'M' (Meningeal reaction)

Table 18.

Case No.	Protein (mgms. per 100 c.c.)	Chlorides (mgms. per 100 c.c.)	Cells per cu.mm.	Per cent. Lymphs.	Per cent. Polys.
10	40	736	8	100	-
11	40		42	86	14
14	50	720	31	100	-
15	50	730	95	100	-
17	60		18	100	-
18	40	720	45	100	-
19	60	740	30	97	3
20	60	700	68	75	25
21	40	736	120	97	3
22	40		26	100	-
23	55	725	120	100	-
25	80	720	300	97	3
29	40		18	100	-
32	40		24	100	-
33	35	732	24	100	-
34	40	734	45	98	2
39	35	726	75	99	1
40	35		18	100	-
41	40		27	100	-
45	40	740	56	100	-
48	50		71	100	-
51	50		36	100	-
54	50	730	75	96	4
57	50		90	98	2
61	45		24	100	-
64	50		240	94	6
67	40		45	100	-
68	40	740	26	100	-
72	50		108	98	2
79	50	726	15	100	-
80	40		36	100	-

Analysis of the cerebro-spinal fluid

in type of disease 'R' (Paralysis which recovered)

Table 19.

Case No.	Protein (mgms. per 100 c.c.)	Chlorides (mgms. per 100 c.c.)	Cells per cu.mm.	Per cent. Lymphs.	Per cent. Polys.
6	60	700	104	96	4
9	60	740	16	100	-
26	40		36	100	-
27	60		225	98	2
31	60	729	221	97	3
43	40	715	150	100	-
52	40		39	100	-
53	60	728	360	98	2
58	50		90	96	4
62	55		72	98	2
65	45		105	96	4

Analysis of the cerebro-spinal fluid

in type of disease 'P' (Paralysis which did not recover)

Table 20.

Case No.	Protein (mgms. per 100 c.c.)	Chlorides (mgms. per 100 c.c.)	Cells per cu.mm.	Per cent. Lymphs.	Per cent. Polys.
1	50		42	100	-
2	40		18	98	2
3	50		3		
4	40		69	100	-
5	40		30	100	-
7	60		9	100	-
8	40		72	100	-
24	60	730	38	100	-
28	60		56	99	1
30	60		75	98	2
35	40		45	99	1
36	80	720	224	97	3
38	60	724	291	96	4
42	30	740	57	94	6
46	80	720	139	67	33
47	40	725	11	100	-
49	80	715	406	60	40
50	70		5		
55	40		27	100	-
59	45		15	100	-
60	80		300	97	3
63	40		30	100	-
66	50		80	98	2
69	45		56	98	2
70	80	726	30	100	-
71	55		9	100	-
73	50		42	100	-
74	60	728	720	98	2
76	40		24	100	-
77	50		48	96	4
78	50		36	100	-
81	20	759	2		

Analysis of the cerebro-spinal fluid in type of disease 'D' (Deaths)

Table 21.

Case No.	Protein (mgms. per 100 c.c.)	Chlorides (mgms. per 100 c.c.)	Cells per cu.mm.	Per cent. Lymphs.	Per cent. Polys.
12	50		129	93	7
13	40		53	97	3
16	60	720	44	98	2
44	20	725	15	100	-
56	50		108	96	4
75	70		108	96	4

The Tables show that there was no single factor in the cerebro-spinal fluid examination on which an estimate of the outcome of the disease could be made. Case 44, who died, showed a normal amount of protein and only 15 cells per cu.mm. whereas Case 64, who had no paralysis, had 60 mgms. of protein per 100 c.c. and 240 cells per cu.mm. The highest protein level in any group was 80 mgms. per 100 c.c. and was found in three of the paralytic cases, but the same amount was also found in one of the non-paralytics. Amongst those who died, the highest protein value was 70 mgms. per 100 c.c.

The number of cells varied within wide limits, and although the greatest number was seen in a paralytic patient (700 per cu.mm.), 300 per cu.mm. were found in one of those who did not develop paralysis.

The chloride content was estimated in some but not all cases, in view of its possible value in differentiating tuberculous meningitis, but was found to be of no significance whatever in relation to the different clinical types of poliomyelitis. It was always within the limits of normality.

Lotscher (1941) found that there was no parallelism in poliomyelitis between the cerebro-spinal fluid cell count on the one hand and the gravity of the disease on the other; he also attached no prognostic significance to any increase in the albumen content.

From the findings in the 1947 Aberdeenshire cases I have concluded that the results of cerebro-spinal fluid examination, made at the earliest opportunity, can not be used to measure the extent of the damage being caused to the central nervous system.

PHYSICAL ACTIVITY IN RELATION TO THE SEVERITY OF THE DISEASE.

Attention has been drawn by Russell (1947) to the physical activities of the patients in the prodromal stages of the disease in relation to its eventual severity; he states that the more violent these activities are, the more severe will be the paralysis. Russell made an analysis of 40 cases in this connection and came to the conclusion that "physical activity in the preparalytic stage of the disease gravely reduces the resistance of the spinal cord cells to the virus, and should therefore be avoided at all costs".

No similar analysis can be offered with regard to the 1947 Aberdeenshire cases because sufficient details of their physical activities prior to admission to hospital were not obtained, but it seems possible, in view of Russell's findings, that the earlier a patient with poliomyelitis is admitted to hospital, and thereby put at complete rest, the less likely he would be to develop paralysis. That is to say that one might expect the average time interval between the onset of the first symptom and the admission to hospital of non-paralytic

patients to be shorter than the time interval between the first symptom and the appearance of paralysis in paralytic patients. In order to test this assumption, these time intervals for the 1947 Aberdeenshire patients have been set out in Tables 22 - 25, for each of the four types of disease.

These show that the mean times between the onset of the first symptom and admission to hospital in the non-paralytics are rather less than the average duration of symptoms prior to onset of the paralysis in the paralytics, but the ranges of the times are not significantly different in any of the groups. If one might judge, therefore, "physical activity" in terms of the length of duration of symptoms before admission, it might be concluded that it does not have any bearing on whether a patient will have the paralytic or non-paralytic form of the disease.

That rest in bed appears to have little effect, or none at all, upon the progress of the paralysis once it has started, or once the preparalytic stage is over, may be seen from the number of patients whose paralysis appeared or became more extensive while they were in

hospital - Table 24. Of those who died, only one showed paralysis before admission; in four it was first observed on admission; and in one it did not appear until after admission. In eight of the patients in Group "P" the paralysis appeared after admission, and in another eight it progressed for varying lengths of time during their stay in hospital. But in Group "R", only one patient developed paralysis after admission to hospital.

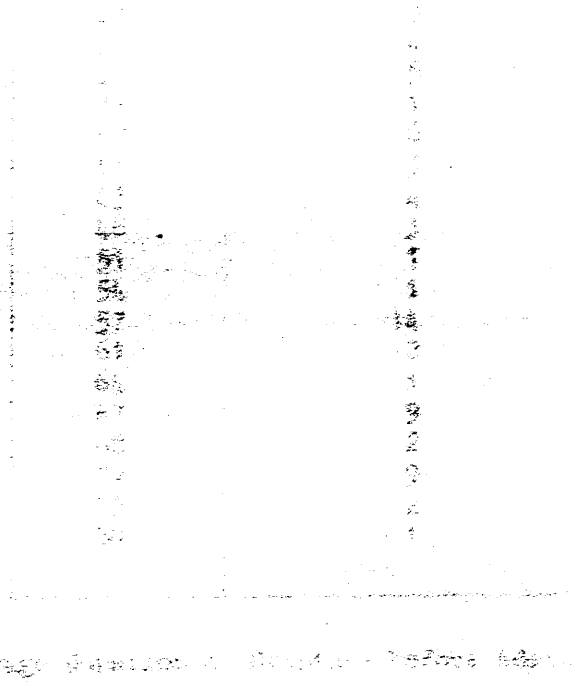


Table 22. **Duration of Symptoms prior to Admission in Type of Disease "M" (Non-paralytic cases).**

Case No.	Duration of Symptoms (Days)
10	6
11	3
14	3
15	2
17	5
18	5
19	2
20	1
21	5
22	9
23	2
25	7
29	3
32	3
33	4
34	5
39	1
40	6
41	3
45	4
48	3
51	1
54	1
57	14
61	2
64	1
67	9
68	2
72	9
79	2
80	1

Average duration of Symptoms before Admission = 3.9 days.

Duration of Symptoms prior to the onset of Paralysis in Type of Disease "R" (Paralysis which recovered).

Table 23.

Case No.	Duration of Symptoms (days)
6	7
9	4
26	4
27	6
31	0.5
37	6
43	0.5
52	15
53	2
58	2
62	5
65	10
12	

Average duration of Symptoms before admission = 5.1 days

Cases in which paralysis appeared before admission: = 2

" " " " " on " = 9

" " " " " after " = 1

12

Duration of Symptoms prior to the onset of Paralysis in Type of Disease "P" (Paralysis which did not recover).

Table 24.

Case No.	Duration of Symptoms (days)
1	1
2	6
3	?
4	2
5	2
7	1
8	4
24	3
28	?
30	4
35	3
36	16
38	4
42	3
46	7
47	7
49	5
50	10
55	4
59	1
60	6
63	5
66	4
69	2
70	4
71	4
73	7
74	15
76	2
77	1
78	7
81	10
32	

Average duration of Symptoms before onset of paralysis = 5.0days

(Cases 3 and 28 have been omitted because the time of onset of the first symptom was uncertain).

Table 24 (cont'd).

Type of Disease "P" (Paralysis which did not recover)

Cases in which paralysis appeared					before	admission:	= 19
"	"	"	"	"	on	"	= 5
"	"	"	"	"	after	"	= 8
							32

Cases in which paralysis was present before admission and in which it progressed after admission (Nos. 4,7,24,35,36,50,60,71) = 8

Duration of Symptoms prior to the onset of Paralysis

Table 25. in Type of Disease "D" (those who died)

Case No.	Duration of Symptoms (days)
12	1
13	3
16	18
44	2
56	1
75	3
6	

Average duration of Symptoms before onset of paralysis = 4.6 days

Cases in which paralysis appeared before admission: = 1

" " " " " on " = 4

" " " " " after " = 1

6

SOME PROBLEMS IN DIFFERENTIAL DIAGNOSIS.

The occurrence of the poliomyelitis epidemic in 1947 brought into prominence some of the difficulties which may be encountered in the diagnosis of the disease, especially in its non-paralytic forms. These difficulties arose at the City Hospital, Aberdeen, as elsewhere and in this section five diseases which may have similar clinical features to poliomyelitis, and which have been encountered in Aberdeen, are discussed. They are

1. Benign lymphocytic meningitis.
2. Glandular Fever.
3. Guillain-Barre disease.
4. "Landry's" Paralysis.
5. Tuberculous meningitis.

Benign Lymphocytic Meningitis - and similar syndromes.

Cases of meningitis in which the etiological agent was unrecognised have been known for many years and have usually been classed as "aseptic". Most frequently the tentative diagnosis of tuberculous meningitis was modified when no tubercle bacilli could be found in the cerebro-spinal fluid and the patient recovered. These features were

scarcely regarded as sufficiently specific to constitute a disease 'sui generis', but rather as due to a variety of unrecognised organisms or other factors. Wallgren (1925) was the first to present seriously the claims for such recognition. He used the term "acute aseptic meningitis" to designate a clinical syndrome in man that he thought was a nosologic entity. He described it as an acute febrile, non-fatal malady characterised by symptoms and signs of meningeal irritation and associated at times with infection of the upper respiratory tract. Now it is known that acute aseptic meningitis is not a nosologic entity, but represents a clinical syndrome which may be caused by more than one etiologic agent, one of which is the virus of lymphocytic choriomeningitis. This virus was accidently discovered by Armstrong and Lillie (1934) in a monkey being used for study of the virus of St. Louis encephalitis. The name lymphocytic choriomeningitis virus was given to the agent because of the marked reaction produced by it in the choroid plexus and meninges of monkeys. Rivers and Scott (1935) recovered the active agent from the cerebro-spinal fluid of human beings ill with what had been diagnosed as Wallgren's acute aseptic meningitis.

The difficulties of the differential diagnosis of this syndrome and non-paralytic poliomyelitis are evident, and until such time as a convenient method of isolating the virus is discovered, the latter disease must continue to be diagnosed on clinical and epidemiological grounds. The case notes of ten patients who were admitted to the City Hospital, Aberdeen during the past ten years, have been found, and they illustrate this point. They all show histories of the acute onset of a febrile illness, with more or less severe headache and vomiting. The patients were all living in Aberdeen or Aberdeenshire at the time of onset of the illness, and the following features were common to all the cases:

1. There was no apparent cause of the illness.
2. No treatment with sulphonamide was recorded.
3. Culture of the cerebro-spinal fluid was sterile.
4. No tubercle bacilli were found in the fluid.
5. None showed any evidence of paralysis.
6. All made uninterrupted recoveries.
7. All were diagnosed as lymphocytic meningitis.

The ages of the patients ranged from 5 to 26 years, 6 being males and 4 females. The findings in the

cerebro-spinal fluid in each case are set out in Table 26.

Each one of these cases might well have been diagnosed as poliomyelitis had they appeared in an epidemic period, and contrariwise, all the non-paralytic cases of poliomyelitis in the recent outbreak might have been diagnosed as lymphocytic meningitis had they occurred sporadically.

McAlpine et al. (1947), writing on this topic, state "there can be no doubt that in this country (Great Britain) a form of benign or aseptic meningitis exists, which is caused by one or more strains of virus described by Armstrong and Lillie (1934). Further, it must be realised that this form of virus meningitis may appear in epidemic form. Nevertheless we are not convinced that up to the present time this condition has been at all common in this country, although it should be added that many cases were seen in Service personnel in the Middle East during the late war. The symptomatology is so similar to that of poliomyelitis that no separate description is called for. Involvement of the central nervous system is rare in benign lymphocytic meningitis. The degree of pleocytosis in the cerebro-spinal fluid is as a general rule greater than in poliomyelitis,

Cerebro-spinal fluid findings in the ten patients referred to on page 103.

Table 26.

Case No.	Protein (mgms. per 100 c.c.)	Cells per cu.mm.	Per cent. Lymphs.	Per cent. Polys.
1	70	3		
2	70	120	90	10
3	80	12	100	-
4	70	133	82	18
5	50	445	73	27
6	60	36	100	-
7	65	108	52	48
8	40	26	100	-
9	80	360	91	9
10	65	480	87	13

counts of over 500 cells being not uncommon".

An epidemic of a similar type of meningitis occurred in the south east of France in the spring and autumn of 1945, and was described by Sohier and Gaubert (1946). Their observations were based upon ".... 1,000 known cases in certain Departments.....and several hundred others". They described the illness as an acute infectious condition of sudden onset, with headache and high fever. The cerebro-spinal fluid showed 10 to 200 lymphocytes per cu.mm. and 40 to 60 mgms. of protein per 100 c.c. The fever resolved and the patients, apart from a "fairly pronounced residual asthenia", felt well again in from 4 to 7 days. Sohier and Gaubert transmitted the disease to human volunteers by intravenous inoculation of cerebro-spinal fluid taken from patients in the fourth day of the illness, and the authors concluded that it was caused by a virus. The capricious distribution of the cases is stressed, and appears very similar to that pertaining in poliomyelitis. They dispose of the suggestion that the syndrome might have been lymphocytic meningitis on the grounds that they could find no record of this disease occurring in epidemic form.

There is thus evidence that sporadic and epidemic benign lymphocytic meningitis and another form of epidemic meningitis running a benign course - all indistinguishable from non-paralytic poliomyelitis - can occur, and there is therefore the possibility that the non-paralysed cases in a poliomyelitis outbreak might in fact be cases of benign lymphocytic meningitis of a concurrent epidemic.

Glandular Fever.

The fact that there may be involvement of the central nervous system in glandular fever has been referred to by several workers. Slade (1946) describes this occurrence and states that the signs and symptoms most frequently met with are, in order of their development, headache, nuchal rigidity, lethargy, muscle twitching, facial paralysis, nausea and photophobia. He also states that in these cases the cerebro-spinal fluid shows a lymphocytic pleocytosis with an increased amount of protein.

Landes et al. (1941) published the notes of a case of infectious mononucleosis with involvement of the central nervous system. The patient was a man aged 21 who was admitted to hospital on May 19, 1940. Ten days

previously he had developed pain in one calf, and this was followed the next day by headache and dizziness. His gait gradually became unsteady and he had frequent attacks of nausea and vomiting. On admission to hospital he was very irritable, becoming lethargic later; he complained of severe frontal headache and his temperature was 101°F. His speech was slurred and muscular co-ordination in arms and legs was poor. There was no paralysis. The cerebro-spinal fluid was examined on admission to hospital and on two further occasions at ten day intervals. No increase in cells was found at any time, but the amounts of protein at the three examinations were 156, 170 and 77 mgms. per 100 c.c. respectively. The blood on the sixth day after admission showed 14,000 white cells per cu.mm., 88% of them lymphocytes, while the heterophile agglutination test was positive in a dilution of 1 in 1024.

The writers stress the fact that lymphadenopathy did not appear until twelve and splenomegaly until fifteen days after admission, and they make a plea for carrying out the heterophile agglutination test in the presence of acute cerebral symptoms of unknown etiology.

Tidy (1945), writing on Glandular Fever, referred to the neurological manifestations of the disease. He stated that the brain, meninges, spinal cord and cranial and peripheral nerves might be involved, but that the commonest form was the meningeal. There was no constant order in which the typical blood changes, glandular enlargement and neurological symptoms developed, and their comparative severity varied.

In a further article Tidy (1946) discussed the differential diagnosis of benign lymphocytic meningitis and glandular fever. He was unable to find any case report in the literature in which benign lymphocytic meningitis had been diagnosed to the exclusion of glandular fever by haematological and serological tests. The impossibility of distinguishing non-paralytic poliomyelitis from benign lymphocytic meningitis has been pointed out in a previous paragraph, and now evidence has been set down to show that lymphocytic meningitis sometimes cannot be distinguished from glandular fever. Thus the question arises as to whether some of the patients diagnosed as having poliomyelitis in the 1947 outbreak did not in fact have glandular fever.

Thelander and Shaw (1941) observed, with regard to a series of cases of glandular fever which they studied, that the diagnosis was very strongly suggested in each case at the first examination. Moreover, during the previous two years they had studied numerous obscure illnesses in children haematologically and serologically without the presence of glandular fever being discovered. One might infer, therefore, that even when meningeal symptoms are caused by glandular fever, one could expect to find other more typical signs of the disease such as lymphadenopathy, splenomegaly or faucial membrane. In the same article these authors state that more often than not, cerebrospinal fluid examination in patients with glandular fever reveals normal findings, but they produced evidence that changes may occur. Details of the cerebro-spinal fluid findings in 13 cases showed that the number of cells varied from 6 to 630 per cu.mm. The symptoms of these patients were variable; some had sluggish speech and mental confusion; none had peripheral paralysis.

In contradistinction to these writers, Tidy (1945), quoted above, emphasised that the disease might present

with neurological signs and symptoms alone - the glandular phase having passed unnoticed or not yet having become evident. In conclusion he stated "the diagnosis of glandular fever is clearly liable to be overlooked in the presence of neurological complications. This is specially so in the severe form, in which glandular enlargement is usually late and slight, and the early blood count shows a moderate polymucleosis or normal result, and is not often repeated".

In November, 1946, a child aged 11 months developed a febrile illness followed by pain and limpness in the right arm. A week later he was sent to hospital as a case of poliomyelitis. There the paralysis was noted, but confirmatory cerebro-spinal fluid examination was not possible owing to the fluid being contaminated with blood. The following day he was transferred to the City Hospital, Aberdeen where examination revealed: an alert child; afebrile; no neck rigidity; several enlarged lymph nodes in both groins; spleen palpable one inch below the left costal margin; appreciable weakness of the right deltoid, triceps, biceps brachii and gluteal muscles. Cerebro-spinal fluid examination showed no

abnormality, but the blood picture showed 12,500 white cells per cu.mm., with 74% lymphocytes. The normal white blood cell count at the end of the first year of life is stated to 15,000 per cu.mm., with 60% to 65% lymphocytes (Sheldon, 1946), so that although the patient's count was for practical purposes within the limits of normality, the whole syndrome suggested that the child might be suffering from glandular fever, complicated possibly by poliomyelitis. But without means for identifying the etiological agent, the true diagnosis in this case, as in others of a like nature, remains uncertain.

Guillain-Barré Disease.

Guillain, Barré and Strohl first described this syndrome in 1916, and pointed out that the main differences from poliomyelitis were the absence of gross wasting and, characteristically, a marked cell-protein dissociation in the cerebro-spinal fluid - "hyperalbuminose du liquide céphalo-rachidien sans réaction cellulaire".

Holmes (1917) described twelve cases of a similar type, two of whom died. He called the syndrome "acute infective polyneuritis", and noted degenerative changes

in the sciatic nerves and no changes in the central nervous system, except for minimal chromatolysis in the anterior horn cells and in the Betz cells of the motor cortex. He stated, however, that examination of the cerebro-spinal fluid was made in only three of his cases, and that no abnormality was found in any of them.

Thirty-five cases were collected by Gilpin et al. (1936) at the Mayo Clinic between 1918 and 1935. They stated that there was usually a febrile onset followed, after a latent period varying from a few days to two months, by paraesthesia, pain in the limbs and progressive paralysis. There was no true atrophy of the affected muscles. The cerebro-spinal fluid contained from 100 to 800 mgms. of protein per 100 c.c. and from 1 to 80 cells per cu.mm. Only three cases had more than 24 cells per cu.mm.

As regards the pathological findings, they pointed out that the changes in the spinal nerve trunks were degenerative and not inflammatory, and also that "the ganglion cells of the anterior horns of the spinal cord were normal, and there was no degeneration in the nerve roots within the spinal canal".

These authors also stated that where recovery occurred in their patients, it was practically always complete; ..."We have seen some faulty regeneration or slight residual weakness, but never any gross defect".

In the records of the Aberdeen City Hospital, the Aberdeen Royal Infirmary and the Royal Aberdeen Hospital for Sick Children, case notes have been found relating to 12 patients, admitted to one or other of these institutions, in whom the diagnosis of Guillain-Barré disease might well be substantiated. Details of these patients have been set out in Table 27 along with the results of cerebro-spinal fluid examination noted in the case records.

These show, in the first place, that none of the cases were, apparently, associated with each other, as they came from widely separated parts and the dates of onset were quite distinct. Thus, if the condition is caused by an infectious agent - which is really unlikely in view of the pathological findings - then it is not highly infectious.

Secondly, with the exception of case 12, the protein content of the cerebro-spinal fluid differed

Table 27. Details of the twelve patients referred to on page 114

Case No.	Age (yrs)	Address	Admitted to hospital	Discharged from hospital	Report on examination of cerebro-spinal fluid
1	22	Hull	May, 1937	June, 1937	"Protein 250 mgms. per cent., no cells".
2	47	Aberdeen	Feb. 1938	Mar. 1938	"Protein 450 mgms. per cent., 35 cells per cu.mm."
3	14	Ellon, Aberdeenshire	Feb. 1941	Apr. 1941	"Protein 225 mgms. per cent, no increase in cells."
4	28	Aberdeen	May, 1942	May, 1942	"Protein 200 mgms. per cent., 10 cells per cu.mm."
5	16	Aberdeen	Nov. 1941	Mar. 1942	"Globulin increased ++, no increase in cells".
6	4	Aberdeen	Oct. 1941	Dec. 1941	"Protein 200 mgms. per cent., 22 cells per cu.mm."
7	4	Balmedie, Aberdeenshire	June, 1942	Aug. 1942	"Protein 300 mgms. per 100 c.c., no cells".
8	31	Aberdeen	June, 1942	June, 1942	"Protein 240 mgms. per cent., no increase in cells".
9	51	Fraserburgh, Aberdeenshire	Nov. 1944	Mar. 1945	"Globulin increased ++, no increase in cells".
10	53	Lerwick	Dec. 1944	Dec. 1944	"Protein 250 mgms. per 100 c.c., 20 cells per cu.mm."
11	46	Shetland	Feb. 1946	Apr. 1946	"Protein 240 mgms. per cent., no increase in cells".
12	58	Inverness	May, 1946	Died June, 1946	"Protein 45 mgms. per 100 c.c., no increase in cells".

widely from that found in poliomyelitis - the highest value found in the 1947 Aberdeenshire cases was 80 mgms. per 100 c.c. (Tables 18 - 21).

Case 12, who came to autopsy, was found to have acute changes in the ganglion cells of the spinal cord and medulla, and in the light of the findings of Gilpin et al. (1936) and Holmes (1917), it is doubtful if this was, in fact, a case of polyradiculoneuronitis. The view was expressed in the post-mortem report that there was insufficient perivascular lymphocytic infiltration to justify a diagnosis of poliomyelitis. The patient's history was of 12 weeks' progression of paralysis, and this too makes poliomyelitis a most unlikely diagnosis. But whatever the true diagnosis, I think it could not have been polyradiculoneuronitis.

"Landry's" Paralysis.

Landry, in 1859, described a case of acute ascending paralysis of flaccid type, involving the lower extremities, the trunk, the upper extremities and finally the vital centres. There were some objective sensory changes, and on post mortem examination

he found no abnormality in the central nervous system.

As a technical term, "Landry's paralysis" lacks precision, etiological or pathological, and its clinical worth is a little dubious. The name lingers on, but it is becoming increasingly clear that all patients with this type of paralysis have pathological changes to explain it.

Wickman (1913) recognised a form of poliomyelitis similar to Landry's paralysis and stated that the paralysis spread up, or more rarely down, the spinal cord, often involving the bulb. Cranial nerves were attacked, and as the respiratory centre became implicated, dyspnoea appeared, death occurring on the third or fourth day. If recovery took place, signs of a widespread spinal type of the disease, occasionally associated with cranial nerve paralysis, persisted.

In 1910 Pirie described ascending paralysis in a man of 41 years, and stated that there was histological evidence at post mortem that it was caused by poliomyelitis. Reference has already been made in the historical section to Monro's (1911) case of ascending paralysis.

In 1916 Harrington and Teacher described a case of

ascending paralysis in a woman of 48 years, and they made a diagnosis of "subacute anterior poliomyelitis".

Wilson (1940) states his opinion that the conception of a self contained Landry's paralysis has been reduced almost to vanishing point, and that the term "acute ascending paralysis" suffers alike from clinical ambiguity and pathological diversity. He points out that an ascending type of poliomyelitis is well recognised, but that there are several other causes of it.

From the records of the City Hospital, Aberdeen and the Royal Infirmary, Aberdeen, case notes have been found of six patients in whom the diagnosis of acute ascending myelitis or Landry's paralysis was made. With the exception of one patient who recovered completely, all died of respiratory failure, due apparently to bulbar involvement in four cases, and to intercostal and diaphragmatic paralysis in one case. Records of the result of cerebro-spinal fluid examination were made in only two of the cases. In one of these it showed normal findings and in the other showed 5 cells per cu.mm. and 250 mgms. of protein per 100 c.c. This latter case could therefore be classified as one showing the Guillain-Barre syndrome, already discussed.

In the 1947 Aberdeenshire outbreak of poliomyelitis, one patient (Case 75) died as a result of ascending paralysis, and the post mortem findings confirmed that the condition was due to poliomyelitis.

In five other patients, excluding those who died, there was evidence of spread of the lesion either up or down the spinal cord after the initial - and usually maximal - paralysis had set in (Cases 4, 7, 36, 60 and 71).

It may be concluded, therefore, that an acute ascending fatal type of poliomyelitis exists, but that other diseases may give a similar neurological picture; and also that the order of appearance of the paralysis in poliomyelitis, spread out over the space of some days, can suggest ascending or descending myelitis, or both.

Tuberculous Meningitis.

The advent of streptomycin has brought into prominence the importance of the early diagnosis of tuberculous meningitis, and there arises simultaneously the question of its differentiation from poliomyelitis.

Fourteen cases of tuberculous meningitis were admitted to the City Hospital, Aberdeen in 1947, and in seven of them the diagnosis was established by the

finding of tubercle bacilli in the cerebro-spinal fluid at first examination, while in the remainder it was clinched retrospectively as a result of guinea pig inoculation.

The onset of tuberculous meningitis is essentially insidious, while that of poliomyelitis is sudden and frequently followed by a period of wellbeing before meningeal symptoms appear.

Examination of the cerebro-spinal fluid, quite apart from the isolation of the tubercle bacillus from it, has, as a rule, been found to be of considerable assistance in the differentiation. The cerebro-spinal fluid findings in the fourteen cases of tuberculous meningitis mentione, are set out in Table 28. In comparison with those from the cases of poliomyelitis occurring in Aberdeen in 1947 (Tables 18-21), they show that, in the cases of tuberculous meningitis, the protein level was higher (average 174 mgms. per 100 c.c.) than in the cases of poliomyelitis (average 49 mgms. per 100 c.c.). The total and differential cell counts, on the other hand, are not markedly at variance in the two diseases.

The results of chloride estimations were not available routinely in all the poliomyelitis cases, but the lowest

Cerebro-spinal Fluid in Tuberculous Meningitis.

Table 28.

(at first examination)

Case No.	Age (yrs)	Protein (mgms. per 100 c.c.)	Chloride (mgms. per 100 c.c.)	Cells per cu.mm.	Per cent. lymphs.	Per cent. polys.	T.B.
1	38	250	610	84	89	11	-
2	8	230	695	240	98	2	-
3	7	130	680	68	98	2	-
4	21	120	620	56	98	2	+
5	20	100	670	126	98	2	+
6	3	110	685	60	98	2	-
7	21	270	600	300	-	-	+
8	20	120	610	130	-	-	+
9	18	280	-	132	-	-	+
10	32	240	640	-	98	2	-
11	6	150	670	270	98	2	-
12	20	170	665	340	47	53	-
13	40	140	630	360	98	2	+
14	15	120	680	350	99	1	+

figure recorded was 700 mgms. per 100 c.c. while the highest value in the cases of tuberculous meningitis was 695 mgms. per 100 c.c.

Recently, stress has been laid on the value of sugar estimations in the cerebro-spinal fluid in relation to the early diagnosis of tuberculous meningitis (Rubie and Mohun, 1949), but these estimations were not made routinely in the Aberdeen cases under review.

SUMMARY AND CONCLUSIONS.

Historical.

According to the available evidence, poliomyelitis has probably existed in Scotland since at least 1772. In the literature, no positive proof has been found that the disease was present in Aberdeenshire before the latter part of the nineteenth century.

Poliomyelitis first became notifiable in Aberdeen in 1913, and the greatest recorded epidemic there occurred in 1916.

Epidemiology.

The rural incidence of the disease in the North East of Scotland is slightly higher than the urban rate. It is suggested that this difference in incidence may become even less evident in view of the increasing facility of modern transport.

Only in the years 1916 and 1947 were there a sufficient number of cases in Aberdeenshire to warrant the title "epidemic" by the minimum American standards. Prior to 1947, the highest incidence of poliomyelitis

in England and Wales was 3.8 per 100,000; this figure has been surpassed on seven occasions in Aberdeenshire.

In the County of Aberdeen, cases of the disease have been widely scattered, during the years under review, but two highly localised outbreaks - in Fyvie and Collieston - have been mentioned. The case incidence in the City of Aberdeen bears no relationship to the population density.

The age incidence of poliomyelitis is higher in the County of Aberdeen than in the City.

Comparing the two epidemic years 1916 and 1947 in the City of Aberdeen, the age incidence was significantly higher in the latter year.

More males contract the disease than females.

The case fatality is highest in the years of low incidence. The overall death rate in the County of Aberdeen is not higher than that in Aberdeen City. In the 1947 epidemic, all those who died were City patients. Persons in the older age groups, although collectively more resistant to the disease, die more readily than those of any other age group, once they do contract it.

In seasonal incidence, the disease in Aberdeenshire is most prevalent between July and October. In the two years of greatest recorded incidence in Aberdeen - 1916 and 1947 - local meteorological conditions were markedly different.

Contacts between proved cases of the disease were the exception in the 1947 Aberdeenshire outbreak, but if minor illnesses of associates might be considered to be mild attacks of poliomyelitis, then nearly half the 1947 cases were units of small multiple outbreaks.

Swimming Baths did not appear to be sources of infection.

None of the 1947 Aberdeenshire cases had their illnesses precipitated by tonsillectomy.

Cerebro-spinal Fluid.

In none of the 1947 Aberdeenshire cases of poliomyelitis did examination of the cerebro-spinal fluid at the time of admission to hospital give any idea of what the subsequent course of the disease would be.

Physical Activity.

Rest in bed has no appreciable effect upon the progress of the paralysis in poliomyelitis once it has

started, or once the preparalytic stage is over.

Differential Diagnosis.

Five conditions which may have similar clinical features to poliomyelitis, and which have been met with in Aberdeen, have been discussed.

Without isolating the virus, it is impossible to distinguish between benign lymphocytic meningitis and non-paralytic poliomyelitis.

In this study I have tried to elucidate some of the problems that poliomyelitis presents. All that has in fact been accomplished has been to show that the same fundamental problems exist in Aberdeenshire as they do in other parts of the world - the appearance of epidemics, the changing age incidence, the high mortality in the lowincidence age groups, the apparent increase in the number of non-paralytic cases - and several more. I have tried to explain them in the light of published epidemiologic and experimental investigations, and

therein have accomplished nothing new.

There is, however, one impression that I have gained from compiling the Thesis, and it is that changes in the individuals or hosts are as important as changes in virus or environment in fostering the appearance of the disease. That the virus can change both in invasive power and immunologic type has been mentioned already, but I think that these factors, per se, are insufficient to account for anything like all the manifestations of the disease.

The virus to my mind must be very widespread in the community, and in order to explain isolated cases of the disease in both endemic and epidemic times - in every age group - attention should be directed not so much as to whether the virus is present in the alimentary tract, as to those circumstances which permit its entry from there to the central nervous system.

The high mortality amongst the higher age groups where the incidence is low might be explained on the rarity of a portal of entry appearing in the adult. Adults are presumably just as often exposed to the virus as children, and perhaps their defence arrangements are

such that the virus is seldom allowed to pass from the alimentary tract to the central nervous system, and then only because a portal of entry for some reason appears.

An explanation of why some individual who are known to have been in contact with the disease in the past, and yet did not contract it until some years later, might be made on the same theory.

That the very appearance of epidemics could be explained on these grounds also is, to my mind, not outwith the bounds of possibility. Increase in numbers and virulence, and changes in the immunologic type of the virus there may be, but I think that the reason for any one individual contracting the disease probably lies ultimately with him and not with the virus. The presence of healthy carriers has been proved, but it is possible that they might succumb to the disease if the virus were able to find a portal of entry to the central nervous system, instead of remaining in the pharynx or bowel, where it can do no harm.

The existence of this portal of entry is easier

to postulate than to locate or describe, and there is little new in the hypothesis itself. But from this study of the behaviour of the disease in Aberdeenshire, I have come to the conclusion that some such mechanism must be present to explain some of the problems that arise.

APPENDIX.

Summaries of all the 1947 cases in the City and County of Aberdeen are set out in the Appendix in chronological order of their admission to Hospital.

Explanatory notes of the various headings of the Summaries are set out below:

(1) Type of Disease - shown by a single letter at the top right hand corner of each summary.

'D' - patient died.

'P' - paralysis which did not recover completely.

'R' - paralysis which recovered completely.

'M' - meningeal reaction with no paralysis.

(2) Recent Activities and Contacts.

The activities of the patient within the three weeks prior to the onset of the disease were ascertained along with a list of child contacts during the same period, noting any minor illness amongst them. Special emphasis was laid upon visits to swimming baths and cinemas, but other possible sources of infection, e.g. public transport and shops, were not investigated. Past medical histories were seldom relevant and have therefore been excluded, but careful enquiry was made for family histories of poliomyelitis. None of the patients had recent histories of an operation upon the nose or throat.

(3) Incubation Period.

This has been indicated in possible cases when direct or indirect contact with proved cases was known.

(4) History of Onset.

This was obtained from the patient or relatives or both and is necessarily a mixture of symptoms and signs.

(5) Examination.

A brief summary of the salient clinical features is made in order to classify each case as to type of disease. It was difficult to assess paralysis in very young children especially when it was not extensive.

(6) Progress.

The patients were retained in the City Hospital for a minimum period of three weeks after admission and little has been said about their progress after discharge. All those with severe paralysis were transferred to Stracathro Hospital for orthopaedic treatment while the remainder were examined again at the orthopaedic department some four weeks after discharge from the City Hospital.

Case 1. P.

P.R., male, 32 years. Occupation - Minister of Religion.
Newtondee,
Bieldside, Admitted 16.2.47
Aberdeenshire. Discharged 4.3.47

Recent Activities and Contacts. Except for a visit to the cinema on 11.2.47, he had been at home. He was in contact at Newtondee Residential School with case 2, whose first symptom developed on 4.2.47.

Incubation period. If he was infected by Case 2, the incubation period must have been, at most, 10 days and that is if it is assumed that Case 2 was not infectious before his symptoms began.

History of Onset. On 14.2.47 he felt shivery and complained of headache. Next day he was unable to speak and had developed weakness of the right upper and lower extremities along with retention of urine.

Examination. Fevered; hiccough; delirium; nystagmus to the right; paresis of right lower face and right upper and lower extremities. All superficial and deep reflexes diminished on the right side, plantar responses equivocal.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 42 per cu.mm. - all lymphocytes.
Culture - sterile
Wassermann Reaction - negative.

Progress. Temperature normal on 17.2.47. His speech and mental state were restored by 25.2.47. Eleven months later the only disability that remained was slight weakness of the right hand.

Case 2. P.

J.L., male, 16 years. Mongol - at special school.
Newtondee,
Bieldside, Admitted 19.2.47.
Aberdeenshire. Discharged 4.3.47.

Recent Activities and Contacts. He had been under supervision at the institution and had not been away from it. In contact with all the other patients and with Case 1, whose first symptom appeared 10 days after the onset of Case 2's illness.

Incubation Period. Could not be calculated.

History of Onset. Became ill with headache and fever on 4.2.47, and pain and limpness in the right arm appeared on 10.2.47.

Examination. Mongoloid features; flaccid paralysis of right upper extremity.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 18 per cu.mm. - 98% lymphocytes
2% polymorphs.
Culture - sterile.

Progress. There was slight movement in the shoulder and fingers of the paralysed limb when he was discharged.

Case 3. P.

H.M., male, 7 years.
45 Burns Road,
Aberdeen.

Gordon's College School.
Admitted 7.7.47
Discharged 23.9.47.

Recent Activities and Contacts. Holidays from school began on 26.6.47 and he had not been out of Aberdeen. His brother aged 5 years remained well and no other pupil at Gordon's College is known to have contracted the disease.

Incubation Period. Could not be calculated.

History of Onset. On 4.7.47 he complained of pain in the left shoulder and inability to move the left arm.

Examination. Afebrile. Complete paralysis of the left upper extremity except for the adductors of the arm and some of the intrinsic muscles of the hand.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 3 per cu.mm.
Culture - sterile.

Progress. Power steadily returned to the arm and on discharge the deltoid was the only paralysed muscle in the limb.

Case 4. P.

W.C., male, 1¼ years.
15 Northfield Place,
Aberdeen.

At Home.
Admitted 11.7.47.
Discharged 8.10.47.

Recent Activities and Contacts. He had not been away from Aberdeen, but was at the beach on 4.7.47 and in a public park on 6.7.47. He had been inoculated against diphtheria on 26.6.47.

Incubation Period. Could not be calculated.

History of Onset. Frettiness and vomiting on 8.7.47. These symptoms continued and on 10.7.47 his right arm became limp.

Examination. Fever; photophobia; irritability; flaccid paralysis of the right arm, except for some movement in the hand.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 69 per cu.mm. - all lymphocytes.
Culture - sterile.

Progress. The temperature rose to 104.4°F. on 14.7.47 and on that day paralysis of the left upper and lower extremities and neck appeared, and at the same time the right hand became flail. Improvement thereafter was minimal, the muscles of the neck and the proximal groups of muscles in the affected limbs regaining none of their power.

Remarks. The case was unusual in that the original paralysis was followed 4 days later by further much more extensive paralysis.

Case 5. P.

V.K., female, 1½ years.
5 Kerloch Place,
Torry,
Aberdeen.

At Torry Day Nursery.

Admitted 11.7.47.
Discharged 26.7.47.

Recent Activities and Contacts. Her time was spent either at home or at the Day Nursery. Her two brothers aged 9 and 15 years, her sister aged 17 years, and the 10 other children in the same tenement had no symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. Sudden onset of left-sided convulsions 2 hours prior to admission. Cough, dyspnoea and shivering were also noted.

Examination. There was no fever, but the respiratory rate was 60 per minute and the pulse rate 160 per minute. Twitching of left face and arm; right pupil larger than left; abdominal reflexes absent; right plantar response extensor.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 30 per cu.mm. - all lymphocytes.
Culture - sterile.

Progress. Fever continued until 14.7.47. Paresis of the left upper extremity was first noted on 12.7.47, but on 22.7.47 there were no abnormal clinical findings. However, she was seen again on 29.9.47 when weakness and wasting were present and the child refused to lift herself with the arm.

Case 6. R.

J.G., male, 10 years.
30 Spital,
Aberdeen.

On holiday from a Glasgow School.
Admitted 13.7.47.
Discharged 26.7.47.

Recent Activities and Contacts. He was on holiday from Glasgow with his parents, and knew no one who had the disease or who later developed it.

Incubation Period. Could not be calculated.

History of Onset. Headache, nausea and fever on 6.7.47 followed by 6 days during which he felt off colour but not ill enough to go to bed. On 12.7.47 he went sunbathing and the following day developed severe headache and photophobia.

Examination. Fevered; photophobia; torticollis - with the head turned to the left; neck and back stiffness; Kernig's Sign present; twitching of the left shoulder and the right arm; retention of urine.

Cerebro-spinal Fluid. Protein - 60 mgms.%
Cells - 104 per cu.mm. - 96% lymphocytes
4% polymorphs.
Culture - sterile.
Chlorides- 700 mgms.%

Progress. Temperature was normal on 15.7.47 and urine was voided without resort to artificial means. All abnormal physical signs had disappeared by 19.7.47. Cerebro-spinal Fluid examination was repeated on this day and showed normal findings - 40 mgms. protein %, 3 cells per cu.mm. and culture sterile.

Case 7. P.

E.R., female, 12 years.
32 Belvidere Crescent,
Aberdeen.

On holiday from Aberdeen High School.
Admitted 21.7.47.
Discharged 26.9.47.

Recent Activities and Contacts. Two weeks before admission she had been on holiday in Argyllshire for a week, but had otherwise been at home. She attended the Aberdeen Town Swimming Baths on 19.7.47. There was no connection between this Case and Case 52 as, although they attended the same school, they were on holiday when this Case became ill and Case 52 was not admitted to Hospital until 8 weeks later. She had been in close contact with Case 36 in June at Girl Guide meetings but as Case 36 was not admitted to Hospital until 2.9.47, it seemed unlikely that their illnesses were in any way associated.

Incubation Period. Could not be calculated.

History of Onset. Headache, fever, dizziness, photophobia and anorexia all began on 19.7.47. Vomiting, stiffness in the neck, back and legs appeared the next day along with weakness of her legs.

Examination. Fevered; tenderness of neck muscles; weakness of the extensors of the right knee and foot.

Cerebro-spinal Fluid.
Protein - 60 mgms.%
Cells - 9 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. The paralysis progressed during the next 4 weeks and by 14.8.47 the whole of the right lower extremity had become flail and all the muscles in the left lower extremity were involved to a greater or lesser degree. On her discharge from Hospital there was some improvement on the left side but none in the right leg in which trophic vascular changes had appeared.

Remarks. The progression of the paralysis in this Case was an unusual feature.

Case 8. P.

H.R., female, 13 months. At home.
17 South Mile End Avenue, Admitted 24.7.47.
Aberdeen. Discharged 7.10.47.

Recent Activities and Contacts. She had been at home except for a visit to a Child Welfare Clinic in Aberdeen 2 weeks previously, for inoculation against diphtheria and whooping cough. Her closest contact was her sister aged 3 years who had no symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. Fevered and fretty on 17.7.47 and the following day. She then had no symptoms until 21.7.47 when her left arm was noticed to be limp.

Examination. Fevered; scarlatiniform rash on shoulders and chest; numerous small palpable lymph nodes on both sides of neck; photophobia; flaccid paralysis of left upper extremity, most marked in the proximal muscle groups, with tenderness in the affected muscles.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 72 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. Temperature normal on 25.7.47 with all round improvement of the paralysed muscles except for the deltoid, which showed no recovery. There was complete resolution of the enlarged cervical lymph nodes.

Remarks. An interesting feature of this case was the rash and lymphadenopathy. There appeared to be no focus of streptococcal infection.

Case 9. R.

R.McB., male, 20 years. Farmer.
Benview,
Inverurie, Admitted 31.7.47.
Aberdeenshire. Discharged 21.8.47.

Recent Activities and Contacts. Most of his time had been spent at Inverurie but he had visited Huntly, Aberdeen, Kintore, Insch, Fyvie, Old Meldrum, Moneymusk, Alford, Kemnay and Aboyne. He knew of no contact who had developed the disease and all his immediate associates remained symptom-free.

Incubation Period. Could not be calculated.

History of Onset. Headache, drowsiness and pain in the eyes began on 27.7.47. Later his back and neck became stiff and he vomited several times.

Examination. Fevered; photophobia; neck rigidity; retention of urine.

Cerebro-spinal Fluid. Protein - 60 mgms.%
Cells - 16 per cu.mm. - all lymphocytes
Culture - sterile
Chlorides- 740 mgms.%

Progress. Catheterization required on 1.8.47 but thereafter he rapidly recovered and there was no abnormal sign on 3.8.47

Case 10. M.

B.N., male, 3 years.
6 Tullos Place,
Torry,
Aberdeen.

On holiday from Torry Nursery School.

Admitted 6.8.47.
Discharged 5.9.47.

Recent Activities and Contacts. He had not been outside Torry. He had been a close contact of Cases 12 and 13 who lived next door, but he was the first of the three to become ill.

Incubation Period. Could not be calculated.

History of Onset. Illness began on 31.7.47 with frettiness and fever; then followed pain in the back and neck, vomiting, constipation, dysuria, eye rolling and generalized twitchings.

Examination. Twitching of right arm; eye rolling; absence of upper abdominal reflexes.

Cerebro-spinal Fluid.
Protein - 40 mgms.%
Cells - 8 per cu.mm. - all lymphocytes.
Culture - sterile
Chlorides- 736 mgms.%

Progress. The temperature rose to 100°F. on 7.8.47 but became normal next day. The signs of cerebral irritation gradually subsided and there were no abnormal clinical findings by 20.8.47.

Remarks. The severity of the prodromata and mild pleocytosis in the cerebro-spinal fluid were features of this case. In a follow-up examination on 15.10.47 no abnormality was discovered.

Case 11. M.

S.L., female, 14 years.
2 Ferrier Crescent,
Woodside,
Aberdeen.

On holiday from Powis Junior School.

Admitted 7.8.47.
Discharged 8.9.47.

Recent Activities and Contacts. She had not been out of Aberdeen but went to the cinema a great deal. There were 3 close contacts - a sister aged 12 years and two brothers aged 16 and 21 years respectively - none of whom had any symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. Vague abdominal discomfort on 4.8.47. Headache, pain on moving the eyes and sleeplessness occurred on 6.8.47.

Examination. Fevered; slight neck stiffness; diminution of left ankle jerk.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 42 per cu.mm. - 86% lymphocytes 14% polymorphs.
Culture - sterile.

Progress. There were no abnormal clinical signs on 11.8.47 except for the diminished left ankle jerk, and this became normal by 20.8.47.

Case 12. D.

C.G., female, 2 years. At home.
8 Tullos Place,
Torry, Admitted 7.8.47.
Aberdeen. Died 9.8.47.

Recent Activities and Contacts. She had been at home except for visits to the Beach Ballroom on 23.7.47 and the Beach Carnival, Aberdeen on 2.8.47. She had been in close contact with case 10 who lived next door and whose first symptom appeared on 31.7.47, and with her brother, case 13, who became ill with the disease on 4.8.47. There were 18 other children in the same tenement block, but they all remained symptom-free.

Incubation Period. If it is argued that this patient was infected by Case 10 then the incubation period would have been 6 days; but if she was infected by her brother - Case 13 - then it would have been 2 days. There remains the possibility, however, that all 3 cases had a common unidentified source of infection.

History of Onset. The child seemed perfectly normal on the morning of 6.8.47, but in the evening she became listless and fevered and her mother noticed twitching of the left eye and the left side of the mouth. She slept fairly well but next morning was still fevered and began vomiting.

Examination. A flushed listless child; temperature 101.6°F.; pulse rate 146 per minute; respirations 46 per minute; slight neck rigidity; Kernig's sign present; on crying, right sided facial paresis became apparent.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 129 per cu.mm. - 93% lymphocytes
7% polymorphs.
Culture - sterile.

Progress. The next morning - 8.8.47 - her temperature had fallen to 100°F. and there was no change in the extent of the paralysis, but in the evening she became more irritable and greatly resented interference. Mucus began to collect in her oro-pharynx. A mucus extractor was used from time to time and atropine was given subcutaneously, but in spite of this

she had very noisy respirations and by the morning of 9.8.47 she had in fact lost her cough reflex and swallowing power completely. Artificial respiration by mechanical respirator wasxattempted but the respiration rate gradually increased so that it became uncountable and she died at midnight, i.e. 2 days after her first symptom.

Remarks. The clinical involvement of the 7th, 9th and 10th cranial nerves, the cyanosis in the absence of diaphragmatic or intercostal paralysis, made it appear that death was due to involvement of the vital centres - probably mostly the respiratory centre. The rate, depth and rhythm of her respirations could not be accurately observed after she had been placed in the mechanical respirator.

Case 13. D.

R.G., male, 3 years. At home.
8 Tullos Place,
Torry, Admitted 7.8.47.
Aberdeen. Died 8.8.47.

Recent Activities and Contacts. He had been on holiday from Torry Nursery School for the previous 4 weeks, and had been continually in contact with Case 10, and his sister, Case 12, and with 18 other children in the same tenement all of whom remained symptom-free.

Incubation Period. If he was infected by Case 10 at the time when Case 10 developed his first symptom, then the incubation period was 4 days. But the possibility of the 3 cases - 10, 12 and 13 having had a common source of infection at some previous date is not outwith the bounds of possibility.

History of Onset. On 4.8.47 he was listless and disinclined for food. The next day he was flushed and complained of abdominal pain, but did not vomit and did not complain of headache. That night he was restless and there was twitching of the left side of his face. On the morning of 6.8.47 the twitching recurred and he rolled his eyes and complained bitterly of pain in his back.

Examination. Flushed and ill; face expressionless owing to bilateral facial paralysis. Temperature 99.6°F., pulse rate 90 per minute and respiration rate 32 per minute. His breathing was very noisy and there was frothing at the mouth. There was marked neck rigidity, the presence of Kernig's sign, lateral nystagmus and some weakness of the left arm.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 53 per cu.mm. - 97% lymphocytes
3% polymorphs.
Culture - sterile.

Progress. Next day - 8.8.47 - there was complete paralysis of the soft palate and fluids put into the mouth regurgitated through the nose. The weakness of the left arm was more marked but there was no evidence of paralysis of the diaphragm or of the intercostal muscles. His respiratory

rate and temperature varied little during the next 12 hours, but there was progressive tachycardia and he died at 2 p.m. on 8.8.47.

Remarks. Death was probably due to destruction of the vital centres in the bulb in this case. He was not cyanosed at any stage. There was evidence of involvement of the 7th, 9th and 10th cranial nerves, and the progressive rise in pulse rate might perhaps indicate that death was due to involvement of the cardiac centre in particular.

Case 14. M.

F.N., male, 25 years. Farm servant.
8 Duke Street,
Fetterangus, Admitted 9.8.47.
Aberdeenshire. Discharged 31.8.47.

Recent Activities and Contacts. Apart from visits to Strichen Gala a week previously, and Aikey Fair two weeks previously, he had been at home. None of his associates were ill at this time.

Incubation Period. Could not be calculated.

History of Onset. On 6.8.47 developed severe headache followed by fever, vomiting and pain in his back.

Examination. Fauces injected; slight neck rigidity; stiffness of back muscles; diminished right upper and lower abdominal reflexes.

Cerebro-spinal Fluid.
Protein - 50 mgms.%
Cells - 31 per cu.mm. - all lymphocytes
Culture - sterile
Chlorides- 720 mgms.%

Progress. There were no symptoms on 10.8.47 and no abnormal clinical sign on 12.8.47.

Case 15. M.

W.N., male, 9 years.
11 North Lane,
Fraserburgh,
Aberdeenshire.

On holiday from Central School.

Admitted 13.8.47.
Discharged 5.9.47.

Recent Activities and Contacts. Two weeks previously he had been in Buckie for one day, but apart from this he had been at home.

Incubation Period. Could not be calculated.

History of Onset. Acute onset on 11.8.47 with headache and fever; cough, epistaxis and vomiting developed later, and he had aching pain in his back and behing his knees.

Examination. Fevered; herpes of lips; a moderate degree of stiffness of the neck and back.

Cerebro-spinal Fluid.

Protein - 50 mgms.%
Cells - 95 per cu.mm. - all lymphocytes
Culture - sterile
Chlorides- 730 mgms.%

Progress. He felt well and was afebrile on the evening of 14.8.47 but neck stiffness remained until 20.8.47.

Case 16. D.

A.M., male, 12 years. Schoolboy.
99 Hammerfield Avenue, Admitted 13.8.47.
Aberdeen. Died 18.8.47.

Recent Activities and Contacts. He attended a Scout Camp in the South of Scotland from 26.7.47 until 9.8.47, and N.G., another boy of the same age, who was his close associate there, developed poliomyelitis on 31.7.47 and was admitted to the Edinburgh City Hospital where his right lower extremity became paralysed. There was no illness amongst the rest of the boys at the camp.

Incubation Period. His first symptom was on 27.7.47 and that of his friend N.G. on 31.7.47. No possible source of infection could be ascertained, so that the incubation period could not be calculated.

History of Onset. On 27.7.47 he felt unwell with abdominal pain and headache. This continued for a few days and he was seen by a doctor, but there was nothing in the examination to indicate the true nature of the illness. He was not confined to bed except for one day, when he had acute pain in his back. Then from 6.8.47 until 9.8.47 there was an asymptomatic period during which he felt perfectly fit. On the night of 9.8.47, while he was asleep, his breathing was noticed to be quick and shallow. On 10.8.47 fever developed but there were apparently no other abnormal clinical findings at that time. Next day fever was more marked and he complained of headache and pain in his left shoulder. He vomited several times. Stiffness and pain in his neck developed on 12.8.47 and his temperature was still elevated.

Examination. 13.8.47 - a bright alert child; speech thick. Temperature 100.2°F., pulse rate 112 per minute, respiration rate 22 per minute. There was marked rigidity of the neck and back and Kernig's sign was present. No paralysis was noted but his left upper and lower abdominal reflexes were absent.

Cerebro-spinal Fluid. Protein - 60 mgms.%
Cells - 44 per cu.mm. - 98% lymphocytes
2% polymorphs.
Culture - sterile
Chlorides- 720 mgms.%

Progress. On 14.8.47 his temperature fell to normal in the morning but rose again by one degree F. in the evening. His headache was not so severe but the signs of meningeal irritation were marked and there was definite difficulty in swallowing. Some weakness of flexion of the right knee was also observed. In view of the difficulty in swallowing and the possibility of his inhaling fluids taken by mouth, an intra-venous infusion of glucose-saline was given. His throat remained free of mucus but his voice became husky. The next day the paralysis had not progressed and his temperature remained normal. On 16.8.47 his respirations gradually rose to 60 per minute and he became cyanosed, although no weakness of the diaphragm or intercostal muscles or obstruction to his air passages could be demonstrated. The temperature rose again to 100°F. and he was placed in the mechanical respirator. Oxygen was administered. There appeared to be no increase in the paresis of his right leg and some movement of the soft palate was retained. On 17.8.47 his condition remained critical; he was unable to swallow, but, provided artificial respiration was continued, his colour remained good. On 18.8.47 - in the early morning - his pulse rate rose to 140 per minute. He suddenly became cyanosed, unconscious, and dies shortly thereafter.

Remarks. Respiratory and cardiac failure of central origin appeared to be the cause of death in this case.

Case 17. M.

A.G., female, 9 years.
Soft Hillock Cottage,
Inverurie,
Aberdeenshire.

On holiday from infant school.

Admitted 13.8.47.
Discharged 8.9.47.

Recent Activities and Contacts. Apart from a visit to the Circus in Aberdeen on 4.8.47 she had been at home. She was the niece of two Ward Maids who were in contact with Case 9 from the time he was admitted to Hospital on 31.7.47.

Incubation Period. If the patient was infected by one of her two aunts carrying the virus from Case 9, then the maximum possible incubation period was 9 days.

History of Onset. Fevered and fretty on 8.8.47 followed by stiffness of the back, epistaxis, headache and vomiting.

Examination. Afebrile; slight stiffness of the neck and back; fauces injected.

Cerebro-spinal Fluid.
Protein - 60 mgms.%
Cells - 18 per cu.mm. - all lymphocytes
Culture - sterile

Progress. Uneventful recovery, although the stiffness of her back remained until 4.9.47.

Case 18. M.

R.D., male, 16 years.
Easter Tillathrowie,
Gartly,
Aberdeenshire.

Farm Worker.

Admitted 14.8.47.
Discharged 5.9.47.

Recent Activities and Contacts. He had been in Dundee for a week, three weeks previously, but apart from this he had been working at Clatt - 10 miles from Gartly. His brother N.D. was a patient in the City Hospital, Aberdeen in September 1946 with an illness that the patient described as "meningitis". From the case notes it was found that N.D. had had an illness of acute onset, with fever and signs of meningeal irritation. His cerebro-spinal fluid showed the following: Protein - 70 mgms.%
Cells - 91 per cu.mm. - 89% lymphocytes
11% polymorphs.
Culture - sterile.

He had been diagnosed as a case of "lymphocytic meningitis" and made a straightforward recovery, but the whole picture, in retrospect suggests that it might well have been non-paralytic poliomyelitis.

Incubation Period. Even if it is assumed that his brother was a carrier of the virus, it is impossible to calculate the incubation period.

History of Onset. Gradual onset of frontal headache on 9.8.47 followed by pain on moving his eyes. Then followed sleeplessness and vomiting and the headache became extremely severe.

Examination. Fevered; fauces injected; Kernig's sign present on the right side only. Abdominal reflexes absent.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 45 per cu.mm. - 95% lymphocytes
5% polymorphs.
Culture - sterile.

Progress. His symptoms had disappeared and the abdominal reflexes had returned by 20.8.47.

Case 19. M.

K.McF., male, 10 years.
103 Park Street,
Aberdeen.

On holiday from King Street School.
Admitted 14.8.47.
Discharged 5.9.47.

Recent Activities and Contacts. Four days before he became ill he had returned from a week's holiday in Arbroath with his grandmother, five uncles, one aunt and two sisters aged 7 and 13 years respectively, none of whom were ill.

Incubation Period. Could not be calculated.

History of Onset. Sudden oset of sore throat and frontal headache on 12.8.47. Other symptoms were photophobia and vomiting.

Examination. Fever and slight neck rigidity.

Cerebro-spinal Fluid.
Protein - 60 mgms.%
Cells - 30 per cu.mm. - 97% lymphocytes
3% polymorphs.
Culture - sterile
Chlorides- 740 mgms.%

Progress. Temperature was normal on 15.8.47 but neck stiffness lasted until 23.8.47. There was no evidence of paralysis at any time.

Case 20. M.

P.H., female, 9 years.
20 Morven Place,
Torry,
Aberdeen.

On holiday from the Convent of the Sacred Heart.
Admitted 17.8.47.
Discharged 17.9.47.

Recent Activities and Contacts: She had not been out of Aberdeen but had visited the Carnival and the swimming baths at the beach three weeks previously. Her closest contact had been her brother aged 8 years and he was not ill at that time.

Incubation Period. Could not be calculated.

History of Onset. She awoke on 16.8.47 with a frontal headache and later in the day she felt nauseated and cold and shivery. Slight upper abdominal pain developed on the morning of the day she was admitted.

Examination. Fevered; neck and spinal rigidity; Kernig's sign present; all abdominal reflexes absent.

Cerebro-spinal Fluid. Protein - 60 mgms.%
Cells - 68 per cu.mm. - 75% lymphocytes 25% polymorphs.
Culture - sterile.

Progress. Temperature was normal on 20.8.47 and she was entirely symptom-free on 22.8.47, when the abdominal reflexes had also returned.

Case 21. M.

Mrs. M.C., 31 years. Housewife.
20 Gordon Street,
Huntly, Admitted 20.8.47.
Aberdeenshire. Discharged 10.9.47.

Recent Activities and Contacts. She had not been away from Huntly. Her son, aged 7 years, had a headache on the day that she became ill, but her other close contacts - a daughter of 4 years and two nephews of 7 and 9 years - remained symptom-free.

Incubation Period. Could not be calculated.

History of Onset. Headache, fever and sleeplessness began on 15.8.47. Then followed vomiting and diarrhoea; neck and back pain and stiffness; marked photophobia and dimness of vision.

Cerebro-spinal Fluid.
Protein - 40 mgms.%
Cells - 120 per cu.mm. - 97% lymphocytes
3% polymorphs.
Culture - sterile
Chlorides- 736 mgms.%

Progress. Pain on flexing her head remained until 8.9.47 but she had no symptoms thereafter.

Case 22. M.

J.B., male, 31 years. Electrician.
Culdain,
Gartly, Admitted 21.8.47.
Aberdeenshire. Discharged 10.9.47.

Recent Activities and Contacts. Had been to the Circus in Aberdeen on 7.8.47. He had travelled widely in Aberdeenshire during the previous three weeks. He had a son aged 6 years and a daughter 3 years, and the former was ill for two days with a headache on 1 and 2.9.47.

Incubation Period. Could not be calculated.

History of Onset. Backache and frontal headache began on 12.8.47 followed by two symptomless days. Backache again occurred on 16.8.47 but he did not stop work until 19.8.47 when fever, nausea and headache finally overcame him.

Examination. There were no abnormal clinical findings except minimal neck stiffness.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 26 per cu.mm. - all lymphocytes
Culture - sterile

Progress. He made an uneventful recovery and was symptom-free by 25.8.47.

Case 23. M.

H.D., male, 13 years. At School.
Denhill,
Auchmaliddy,
Maud, Admitted 22.8.47.
Aberdeenshire. Discharged 12.9.47.

Recent Activities and Contacts. He had been in Dufftown from 9.8.47 until 19.8.47 and was in contact there with a small baby and a boy of 13 years who apparently was also ill with headache and vomiting on 22.8.47. His cousin C.F. had poliomyelitis with paralysis in 1933.

Incubation Period. Could not be calculated even assuming that his cousin wasa carrier of the virus.

History of Onset. Transient abdominal pain on 20.8.47 followed by headache, sleeplessness and neck stiffness.

Examination. Fevered; marked neck rigidity and the presence of Kernig's sign; both upper and lower left abdominal reflexes absent.

Cerebro-spinal Fluid. Protein - 55 mgms.%
Cells - 120 per cu.mm. - all lymphocytes.
Culture - sterile
Chlorides- 725 mgms.%

Progress. The reflexes had returned and there was no clinical abnormality by 2.9.47.

Case 24. P.

D.R., male, $2\frac{3}{4}$ years.
26 Jute Street,
Aberdeen.

At home.
Admitted 22.8.47.
Discharged 23.9.47.

Recent Activities and Contacts. He had returned from a holiday in Monymusk on 2.8.47 and was in Banchory on 17.8.47. He had otherwise been at home and was in close contact with a child of 5 years who had no similar symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. On 19.8.47 he was fretty and sleepy. Next day he rolled his eyes and complained that his nose was sore. Facial paralysis appeared on the morning of the day he was admitted.

Examination. Fevered; neck rigidity and Kernig's sign; pharynx congested; lateral nystagmus; twitching of right arm; complete right facial paralysis; right hemi-paresis of the tongue.

Cerebro-spinal Fluid.
Protein - 60 mgms.%
Cells - 38 per cu.mm. - all lymphocytes.
Culture - sterile
Chlorides- 730 mgms.%

Progress. Temperature became normal on 23.8.47 but the next day he appeared moribund with gross cardiac irregularity, nystagmus and eye rolling. Next day, however, his heart was regular, they eye rolling had stopped and there was no nystagmus. The neck rigidity persisted until 7.9.47 and although the hypoglossal paresis recovered, the right face remained completely paralysed.

Case 25. M.

A.G., male, 35 years. Farmer.
Crichie House,
Stuartfield, Admitted 23.8.47.
Aberdeenshire. Discharged 11.9.47.

Recent Activities and Contacts. He had been to Aikey Fair a month previous to admission but had otherwise been at home. He was a close contact of a daughter aged 9 years who developed the disease on 27.8.47 (Case 31) and pf another daughter aged 6 years who remained symptom-free.

Incubation Period. Could not be calculated.

History of Onset. Fevered on 16.8.47 and remained in bed for 3 days. On 20.8.47 developed occipital headache, pain behind both eyes and stiffness of the back of his neck. Lumbar puncture performed at his home relieved the symptoms.

Examination. Abdominal and cremasteric reflexes on the right side were absent.

Cerebro-spinal Fluid. Protein - 80 mgms.%
Cells - 300 per cu.mm. - 97% lymphocytes
3% polymorphs.
Culture - sterile
Chlorides- 720 mgms.%

Progress. The abdominal and cremasteric reflexes on the right side were still absent on his discharge from hospital.

Case 26. R.

G.W., male, 11 years.
Upperton,
Millbrex,
Fyvie,
Aberdeenshire.

On holiday from Millbrex School.

Admitted 23.8.47.
Discharged 12.9.47.

Recent Activities and Contacts. He had not been out of Fyvie except between 5.8.47 and 12.8.47 when he had been living with his aunt in Aberdeen; she was in domestic service with Case 16's household. (Case 16 was admitted on 13.8.47.) He had no relations amongst the people who contracted poliomyelitis in the 1928 Fyvie outbreak.

Incubation Period. If his aunt carried the virus from Case 16 the incubation period must have been at least 7 days, and not more than 14 days.

History of Onset. Intermittent frontal headache began on 19.8.47 followed by sore throat, vomiting and fever.

Examination. Fevered; slight weakness of left face; neck rigidity; Kernig's sign; tendon reflexes in right arm less than those in left.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 36 per cu.mm. - all lymphocytes.
Culture - sterile

Progress. Temperature was normal on 24.8.47 and all the signs and symptoms had disappeared by the following day.

Case 28. P.

J.W., male, $2\frac{3}{4}$ years.
164 Crown Street,
Aberdeen.

At home.
Admitted 23.8.47.
Discharged 18.9.47.

Recent Activities and Contacts. Apart from visits to Culter, Aberdeenshire every Sunday, he had not been out of Aberdeen.

Incubation Period. Could not be calculated.

History of Onset. Vomiting, restlessness, fever and photophobia began on 22.8.47.

Examination. Fever; neck rigidity; left pupil larger than the right.

Cerebro-spinal Fluid. Protein - 60 mgms.%
Cells - 56 per cu.mm. - 99% lymphocytes
1% polymorphs.
Culture - sterile.

Progress. No paresis was demonstrated while he was in hospital and there were no abnormal clinical findings by 8.9.47, but at follow-up examination on 15.10.47 there was a slight degree of foot drop on the left side and his mother stated that he tended to trail his left foot when he was tired.

Remarks. This was a case in which the paresis did not become evident until normal use of the limb was resumed.

Case 29. M.

A.F., male, 16 years. Farm Servant.
Auchnieve Croft,
Old Meldrum Admitted 25.8.47.
Aberdeenshire. Discharged 18.9.47.

Recent Activities and Contacts. He went to the cinema regularly three times a week.. He had a brother of 18 years, a sister of 14 years and a cousin of 3 years in close contact with him, but none became ill. He was in the same class at school in 1944 with A.D. - a patient who developed poliomyelitis with paralysis in 1944. He had no symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. Frontal headache on 22.8.47 was followed by photophobia, vomiting and sleeplessness.

Examination. Afebrile; cervical and axillary lymphadenitis; both knee jerks diminished.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 18 per cu.mm. - all lymphocytes.
Culture - sterile.

Progress. Uneventful recovery.

Remarks. An interesting feature of this case was that he was in contact with a proved case of the disease in 1944, but did not develop it himself until 1947.

Case 30. P.

M.D., male, 4 years. At home.
136 Walker Road,
Torry, Admitted 26.8.47.
Aberdeen. Discharged 8.10.47.

Recent Activities and Contacts. He had returned from a month's holiday in Inverurue on 19.8.47 where he had been in contact with Case 17's uncle. Other close contacts were a sister of 7 years and two brothers aged 6 and 9 years, the former of whom was ill with headache and vomiting on 6.8.47.

Incubation Period. If Case 17's uncle was a carrier of the virus and infected this case, the incubation period must have been at least 3 days.

History of Onset. Frontal headache began on 22.8.47 and was followed by stiffness and pain in his back, neck and right leg, vomiting and sore throat.

Examination. Fevered; neck rigidity; complete paralysis of the right psoas and tibialis anterior; paresis of all the other muscles in the right lower extremity; weakness of the abductors of the left thigh and the right external oblique muscle of the abdomen.

Cerebro-spinal Fluid. Protein - 60 mgms.%
Cells - 75 per cu.mm. - 98% lymphocytes
2% polymorphs.
Culture - sterile.

Progress. Improvement was observed in all the affected muscles except the right psoas and the right quadriceps.

Case 31. R.

P.G., female, 9 years.
Crichie House,
Stuartfield,
Aberdeenshire.

Stuartfield School.

Admitted 27.8.47.
Discharged 18.9.47.

Recent Activities and Contacts. She was the daughter of Case 25, and was admitted 4 days after her father. She had not been out of Stuartfield, but was at school from 16.8.47 until 23.8.47. Sisters of 5 and 11 years and a brother of 3 years were not ill at this time, nor was there any known illness amongst the rest of the children at school.

Incubation Period. She and Case 25 may have had a common source of infection - but on the other hand she may have been infected by Case 25, in which event the incubation period would have been at least 4 days.

History of Onset. Occipital headache and dizziness began on the day of admission to hospital.

Examination. Fevered; slight weakness of left triceps; lower left abdominal reflex absent.

Cerebro-spinal Fluid.
Protein - 60 mgms.%
Cells - 221 per cu.mm. - 97% lymphocytes 3% polymorphs.
Culture - sterile.
Chlorides- 729 mgms.%

Progress. There were no clinical abnormalities on 28.8.47.

Remarks. The contact history was marked in this case.

Case 32. M.

G.D., female, 7 years.
Torry Road,
Huntly.

Gordon's School, Huntly.
Admitted 28.8.47.
Discharged 18.9.47.

Recent Activities and Contacts. She had been in Aberdeen on 5.8.47 but had otherwise been at home and at school. In contact with two brothers aged 11 and 12 years and with two sisters aged 9 and 5 years, none of whom had any symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. Headache and vomiting began on 25.8.47 and persisted until admission to hospital.

Examination. There was a maculo-papular eruption on the trunk but no other abnormal physical signs; her Doctor had noted some neck stiffness and had performed a lumbar puncture.

Cerebro-spinal Fluid.
Protein - 40 mgms.%
Cells - 24 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. The rash had entirely disappeared by 30.8.47 and there were no further symptoms.

Case 33. M.

A.M., female, 14 years.
Willow Cottage,
Memsie,
Aberdeenshire.

At Fraserburgh Academy.

Admitted 29.8.47.
Discharged 20.9.47.

Recent Activities and Contacts. She had stayed at Marno Farm (six miles from Memsie) for a week three weeks previously, but had otherwise been at home and at school. Her sister aged 9 years remained well and there was no known illness amongst the other children at school.

Incubation Period. Could not be calculated.

History of Onset. Frontal headache and anorexia on 25.8.47. The following day she complained of sleeplessness, pain in the back, neck and shoulders, and pain on moving the eyes.

Examination. Fevered; conjunctivae congested; some neck rigidity; tenderness in both loins; absent abdominal reflexes.

Cerebro-spinal Fluid.
Protein - 35 mgms.%
Cells - 24 per cu.mm. - all lymphocytes
Culture - sterile
Chlorides- 732 mgms.%

Progress. She had no complaints and the abdominal reflexes had returned by 1.9.47.

Case 34. M.

V.D., male, 22 years.
Upper Balfour,
Alford,
Aberdeenshire.

Farmer.

Admitted 30.8.47.
Discharged 19.9.47.

Recent Activities and Contacts. Apart from a visit to Glasgow two weeks previously he had been at home. His brother aged 12 years had a headache on 1.9.47, but his other contacts - a sister of 9 years and a son of 18 months - remained well.

Incubation Period. Could not be calculated.

History of Onset. Headache began on 25.8.47, together with general malaise. Later he began to vomit and developed sleeplessness, pain behind the eyes, photophobia and shivering.

Examination. Fevered; left abdominal reflex and both knee jerks absent; some tenderness in the left loin.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 45 per cu.mm. - 98% lymphocytes 2% polymorphs.
Culture - sterile

Progress. He had no complaints on 1.9.47 and the reflexes had returned to normal by 8.9.47.

Case 35. P.

M.C., female, 2¾ years.
4 Froghall Avenue,
Aberdeen.

At home.
Admitted 1.9.47.
Discharged 19.11.47.

Recent Activities and Contacts. Apart from shopping outings with her mother she had not been away from home. Her five sisters were her closest contacts - their ages ranging from 1 to 16 years - and none had symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. Cough began on 29.8.47. Limpness of left arm noticed on 1.9.47.

Examination. Fevered; neck rigidity; paralysis of left deltoid, biceps and triceps, and paresis of all other muscles of left upper extremity; absent abdominal reflexes.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 45 per cu.mm. - 99% lymphocytes
1% polymorphs.
Culture - sterile

Progress. Slight weakness of the right glutei became evident on 2.9.47. Fever continued until 3.9.47. When she was allowed out of bed on 25.9.47 walking was found to be impossible. Recovery had taken place, however by the time she was discharged from hospital when she was walking normally and the left upper extremity had recovered almost completely.

Case 36. P.

S.M., female, 16 years.
212 Rosemount Place,
Aberdeen.

Aberdeen Central School.
Admitted 2.9.47.
Discharged 5.10.47.

Recent Activities and Contacts. She had spent one day at Newburgh, Aberdeenshire two weeks previously, but had otherwise been at home, visiting the cinema several times and the circus once. She attended school at the commencement of the term on 1.9.47. She had been in close contact with Case 7 in June 1947 and with Case 60 who went to the same school but who did not become ill until 23.9.47.

Incubation Period. Could not be calculated.

History of Onset. Headache on 19.8.47 followed by vomiting the next day. Thereafter she had no symptoms until 31.8.47 when there was transient neck stiffness. On 1.9.47 she went to school for an hour but had to go home owing to headache and vomiting.

Examination. Fever and neck rigidity only.

Cerebro-spinal Fluid.

Protein - 80 mgms.%
Cells - 224 per cu.mm. - 97% lymphocytes
3% polymorphs.
Culture - sterile
Chlorides- 720 mgms.%

Progress. During the next few days paralysis of the proximal parts of the upper extremities, the intercostals and the diaphragm gradually appeared and on 6.9.47 treatment by the Both respirator was required. The respiratory musculature gradually improved and artificial respiration was no longer necessary by 20.9.47. On discharge from hospital the only muscles that had not shown any signs of recovery were both deltoids and the right pectorals.

Remarks. This was the only patient of the series whose life was saved by treatment in the mechanical respirator.

Case 37. R.

B.B., female, 1¾ years.
16 Urquhart Road,
Aberdeen.

At home.
Admitted 2.9.47.
Discharged 29.9.47.

Recent Activities and Contacts. She had not been away from home. A distant relative was reported to have had poliomyelitis 28 years ago, but this could not be confirmed.

Incubation Period. Could not be calculated.

History of Onset. She complained of abdominal pain on 26.8.47. The next day there was rhinorrhoea, fever and frettiness. On 1.9.47 she developed weakness of the left arm.

Examination. Neck rigidity; stiffness of the back; slight weakness of the left face and of the left upper extremity.

Cerebro-spinal Fluid. Lumbar puncture was performed with difficulty and the fluid obtained was blood-stained and unsuitable for analysis.

Progress. There was no evidence of paresis by 5.9.47, but signs of meningeal irritation persisted until 15.9.47. Uncontaminated cerebro-spinal fluid was not obtained until 15.9.47, when it revealed no abnormality.

Remarks. The diagnosis in this case was not confirmed by cerebro-spinal fluid examination.

Case 38. P.

T.C., female, 13 years.
14a Roslin Street,
Aberdeen.

St. Peter's School, Aberdeen.
Admitted 3.9.47.
Discharged 22.10.47.

Recent Activities and Contacts. She had returned from a two week's caravan holiday at Collieston, Aberdeenshire on 27.8.47. Her closest contact was a child of 2½ years who was not ill at this time.

Incubation Period. Could not be calculated.

History of Onset. She awoke on the morning of 29.8.47 with dizziness and frontal headache and felt shivery in the evening. Then followed 3 asymptomatic days during which she attended school. On 2.9.47, however, she had a headache and felt her neck stiff. She was unable to lift her left arm. She felt nauseated on the day of admission, but did not vomit.

Examination. Slight neck rigidity; abdominal reflexes absent; flaccid paralysis of the left upper extremity except for some movement in the wrist and hand.

Cerebro-spinal Fluid.

Protein	-	60 mgms.%
Cells	-	291 per cu.mm. - 96% lymphocytes, 4% polymorphs.
Culture	-	sterile
Chlorides	-	724 mgms%

Progress. The temperature rose to 100°F. on 4.9.47 but there was no increase in the extent of the paralysis and on discharge from hospital the left biceps and deltoid were the flail muscles in the limb. The abdominal reflexes had reappeared by 9.9.47.

Case 39. M.

D.T., male, 2 years.
30 Erskine Street,
Aberdeen.

At home.
Admitted 3.9.47.
Discharged 26.9.47.

Recent Activities and Contacts. Apart from a visit to New Machar, Aberdeenshire on 24.8.47 and to Stonehaven on 25.8.47, he had been at home. There was a remote relation living in the same house who had poliomyelitis with paralysis of one leg 30 years previously.

Incubation Period. Could not be calculated.

History of Onset. Pain in the back of the neck on 2.9.47 together with anorexia and fever. Very restless and fretty the next day.

Examination. Fevered; neck rigidity and Kernig's sign; absence of right ankle jerk.

Cerebro-spinal Fluid.

Protein - 35 mgms.%
Cells - 75 per cu.mm. - 99% lymphocytes
1% polymorphs.
Culture - sterile
Chlorides- 726 mgms.%

Progress. Neck stiffness remained until 12.9.47 and the right ankle jerk was still absent on discharge from hospital.

Case 40. M.

C.T., male, 5 years.
Loanhead Cottage,
Corse of Monelli,
Aberdeenshire.

Corse School.

Admitted 3.9.47.
Discharged 4.10.47.

Recent Activities and Contacts. Apart from going to school during the two weeks prior to admission, he had been at home. He was in close contact with a sister of 4 years and a brother of 1½ years. His father's brother had poliomyelitis at the age of 2, i.e. in 1909.

Incubation Period. Could not be calculated.

History of Onset. He was off school on 28.8.47 with severe occipital headache, but returned next day, saying that he felt quite well. That night he vomited and sat up in bed while he was asleep, gripping his hands and moaning. He then seemed normal until 1.9.47 when headache and vomiting recurred.

Examination. Afebrile; marked neck and back rigidity.

Cerebro-spinal Fluid. Protein - 35 mgms.%
Cells - 18 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. There were no abnormal clinical signs by 9.9.47, by he had an attack of gastritis with fever on 14..9.47 from which he rapidly recovered.

Case 41. M.

H.McL., male, 25 years. Unemployed.
4 Gairn Road, Admitted 4.9.47.
Aberdeen. Discharged 24.9.47.

Recent Activities and Contacts. Hev had not been out of Aberdeen but went to the cinema almost every day. On 21.8.47 he was admitted to the Aberdeen Royal Infirmary for treatment of inclusion dermoids of both great toes. While there he developed sciatic pain. Lumbar puncture was performed and examination of the cerebro-spinal fluid showed normal findings.

Incubation Period. Could not be calculated.

History of Onset. He was discharged from the Aberdeen Royal Infirmary on 26.8.47 but on 1.9.47, while at the cinema, he developed severe frontal headache and was nauseated. Backache and neck stiffness developed the following day.

Examination. Fevered; Tenderness of the muscles of the back and of the left thigh. There was possibly some weakness of the right upper and left lower extremities, but the patient was so unco-operative that this was discounted. Neck rigidity and Kernig's sign were present.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 27 per cu.mm. - all lymphocytes.
Culture - sterile.

Progress. The temperature was normal on 5.9.47 and by 8.9.47 there were no objective signs of the disease although he had a multitude of transitory symptoms until his discharge from hospital.

Case 42. P.

A.M., male, 3 years.
22 East North Street,
Aberdeen.

At home.
Admitted 4.9.47.
Discharged 4.11.47.

Recent Activities and Contacts. He had not been away from home and his two closest contacts - a sister of 5 years and a brother of 16 years - were not ill at this time. Case 44 lived in the same house, but became ill 3 days after Case 42.

Incubation Period. This case and case 44 may have had some common source of infection but what this could have been was not ascertainable.

History of Onset. He appeared perfectly well when he went to bed on the evening of 3.9.47 but groaned during the night and slept on until 10 a.m. the following day. He was listless and fevered and would not take food and complained that the right side of his neck was painful.

Examination. Fevered; irritable; follicular exudate on tonsils; some resistance to flexion of neck.

Cerebro-spinal Fluid.
Protein - 30 mgms.%
Cells - 57 per cu.mm. - 94% lymphocytes
6% polymorphs.
Culture - sterile
Chlorides- 740 mgms.%

Progress. A profuse growth of haemolytic streptococci was obtained from the throat and sulphonamide was prescribed. The temperature became normal on 6.9.47. On 5.9.47 there was marked neck rigidity and Kernig's sign was present; there was difficulty in micturition. Neck rigidity persisted until 16.9.47 but from 9.9.47 onwards there was gradual improvement in the paresis so that there was minimal disability on discharge from hospital. The child was very ataxiv when he was allowed up on 26.9.47 and it was not until the beginning of the next month that his normal gait wasx restored.

Remarks. The association of poliomyelitis with a throat infection and the ataxia were interesting features of this case.

Case 43. R.

K.I., male, 12 years.
3 Kerloch Place,
Torry,
Aberdeen.

Walker Road School.

Admitted 6.9.47.
Discharged 27.9.47.

Recent Activities and Contacts. He had not been out of Torry and had been atv school since 1.9.47. None of his five brothers and sisters - ages 5 to 13 years - were ill at this time but apparently five children were sent home from his school with vomiting on the day before his first symptom. Case 5, admitted on 11.7.47, came from a tenement in the same street.

Incubation Period. Could not be calculated.

History of Onset. Became ill on the day of admission with sore throat, occipital headache, fever vomiting and stiffness of the neck, shoulders and back.

Examination. Fevered; neck and back rigidity; Kernig's sign; some weakness of upper and lower right face.

Cerebro-spinal Fluid.
Protein - 40 mgms.%
Cells - 150 per cu..mm.. - all lymphocytes
Culture - sterile
Chlorides- 715 mgms.%

Progress. There were no clinical abnormalities on 7.9.47 except the facial paresis which persisted until 13.9.47.

Case 44. D.

A.B., male, 1½ years
22 East North Street,
Aberdeen.

At home.
Admitted 8.9.47.
Died 11.9.47.

Recent Activities and Contacts. He had not been out of Aberdeen and had rarely been away from East North Street. Case 42 who became ill with poliomyelitis on 3.9.47 lived in the same house. Living in the same house were 8 other children whose ages ranged from 3 to 11 years; none of them had any symptoms at this time.

Incubation Period. If it is presumed that this case was infected by Case 42 then the incubation period must have been at least 2 days, but it may well be that both cases were infected independently at some previous unknown date.

History of Onset. On 6.9.47 he developed a cold with discharging eyes and nose, cough and fever. The next day he would not take his food and was very fretty. His mother thought he had a headache. On the morning of 8.9.47 he had a cyanotic attack and was very restless and irritable.

Examination. The child was obviously very ill; temperature 102°F.; pulse rate 160 per minute; respirations 60 per minute. There was anterior and posterior nasal discharge and a loose cough with much sputum which he appeared unable either to swallow or expectorate. Moist sounds were heard throughout the chest. Cervical, axillary and groin lymph nodes were palpable. There was considerable rigidity of the back, neck retraction and the presence of Kernig's sign; apart from the swallowing difficulty there was no paralysis.

Cerebro-spinal Fluid.
Protein - 20 mgms.%
Cells - 15 per cu.mm. - all lymphocytes
Culture - sterile
Chlorides- 725 mgms.%

Progress. In view of the evidence of pneumonia, sulphonamide and penicillin were prescribed and he was fed through a gastric tube. On 9.9.47 his condition became much worse. The temperature was normal, but the respirations became faster and sh shallower, reaching 70 per minute. There was now evidence of consolidation in the left lung. Respiratory paralysis was evident and he was therefore placed in the mechanical

respirator. There was now nasal regurgitation of fluids. On 10.9.47 he was able to swallow a little but in spite of continued artificial respiration and the administration of oxygen he became cyanosed and died the following day.

Post Mortem Examination.

Brain and Meninges. Cerebral oedema was present with flattening of the convolutions. The brain weighed 1300 grammes - equal to that of a normal adult. The cut surface showed intense congestion and there were small haemorrhages in the brain stem.

Spinal Cord. This was under pressure and bulged through an incision made in the meninges. The cut surface showed congestion and small haemorrhages in the parenchyma.

Lungs. There was a left basal pneumonia.

Spleen. The spleen was moderately enlarged.

Histological Examination.

Cerebral Cortex. The meninges showed infiltration with lymphocytes and polymorph cells. The brain substance was congested.

Basal Nuclei. These showed congestion of the vessels.

Pons. This showed congestion and neuronophagia.

Medulla. There was neuronophagia and scanty infiltration of the Virchow-Robin spaces by lymphocytes and polymorphs.

Spinal Cord. Here there was intense congestion with perivascular "cuffing" by inflammatory cells and marked neuronophagia.

Remarks. Death was due to lobar pneumonia and acute anterior poliomyelitis, but while clinically death appeared due to destruction of the vital centres in the brain stem, post mortem appearances indicated that the more serious damage to the central nervous system fell upon the spinal cord.

Case 45. M.

A.S., male, 3 years.
448 George Street,
Aberdeen.

At Home.
Admitted 8.9.47.
Discharged 29.9.47.

Recent Activities and Contacts. He had not been out of the George Street region of the town. His mother was a friend of the mothers of Cases 3 and 44 but was unable to say when she saw either of them last. Case 3 became ill on 4.7.47 and developed paralysis and Case 44 had his first symptom on 6.9.47 and died of the disease. There were 7 other families in the same building as Case 45 but none of their members became ill at this time.

Incubation Period. Could not be calculated.

History of Onset. Symptoms began on 4.9.47 with a sore throat. The next day he developed headache, pain in the back of the neck and fever and the day after that he had delirium and eye rolling at night.

Examination. Fever; tenderness in the lumbar region and stiffness of the neck and back.

Cerebro-spinal Fluid.
Protein - 40 mgms.%
Cells - 56 per cu.mm. - all lymphocytes
Culture - sterile
Chlorides- 740 mgms.%

Progress. He was completely symptom-free on 12.9.47.

Case 46. P.

I.Y., female, 5 years.
64 Back Hilton Road,
Aberdeen.

Kittybrewster School, Aberdeen.
Admitted 9.9.47.
Discharged 4.10.47.

Recent Activities and Contacts. She returned from two weeks' holiday in Paisley on 1.9.47 and since then had been at home and at school. Her sister of 9 years remained well and there was no known illness amongst any of the other children at school at this time.

Incubation Period. Could not be calculated.

History of Onset. On 3.9.47 she was tired and feverish and delirious at night. Then there were no further symptoms until 7.9.47 when she complained of headache and pain in her back. On 8.9.47 she was fevered, her legs, shoulders and hips were painful, her back was stiff and again she was delirious at night.

Examination. Fevered; neck and back rigidity; Kernig's sign; slight tenderness in the lumbar region.

Cerebro-spinal Fluid.

Protein	- 80 mgms.%	
Cells	- 139 per cu.mm.	- 67% lymphocytes 33% polymorphs.
Culture	- sterile	
Chlorides	- 720 mgms.%	

Progress. Temperature became normal on 12.9.47 but paresis of all muscle groups in the right lower extremity appeared on 10.9.47 together with abolition of all the abdominal reflexes. On discharge from hospital, very slight weakness of the adductors, hamstrings and quadriceps was the only remaining disability.

Remarks. The high percentage of polymorphonuclear cells in the cerebro-spinal fluid was a feature in this case.

Case 47. P.

M.C., female, 5 years.
12 Dill Place,
Aberdeen.

Hayton School, Aberdeen.
Admitted 12.9.47.
Discharged 23.10.47.

Recent Activities and Contacts. Apart from going to school - the term started on 1.10.47 - she had not been away from home. On the day after her illness began her sister, aged 3 years, had an unexplained fever lasting three days. None of the school contacts was known to have any symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. The illness began with vomiting on 5.9.47 and during the first 2 days she was listless and fevered and complained that her back and limbs were stiff. On 9.9.47 her temperature was normal but she said that the right side of her face was painful and this symptom lasted until admission. She vomited on the morning of 12.9.47.

Examination. Neck rigidity; stiffness of the back; Kernig's sign; complete right sided facial paralysis.

Cerebro-spinal Fluid.
Protein - 40 mgms.%
Cells - 11 per cu.mm. - all lymphocytes
Culture - sterile
Chlorides- 725 mgms.%

Progress. Pain in the face had disappeared by 13.9.47 but the signs of meningeal irritation persisted until 19.9.47. There was no improvement in the facial paralysis.

Case 48. M.

G.C., female, 8 years.
43 Queen's Road,
Aberdeen.

St. Margaret's School, Aberdeen.
Admitted 13.9.47.
Discharged 3.10.47.

Recent Activities and Contacts. She came home from holiday at Grantown-on-Spey on 25.8.47 and since then had been to the Aberdeen town swimming baths on 31.8.47 and once to the cinema. A domestic servant in the household had been ill with fever and headache between 20.8.47 and 27.8.47. She was at school for 2 days only.

Incubation Period. Could not be calculated.

History of Onset. Lassitude and fever on 10.9.47; headache the following day and some stiffness of the neck.

Examination. Slight neck stiffness; equivocal left plantar response.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 71 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. There was no abnormality on 14.9.47.

Case 49. P.

Mrs. J.R., 33 years. Housewife.
102 Hammerfield Avenue, Admitted 13.9.47.
Aberdeen. Discharged 31.10.47.

Recent Activities and Contacts. She had recently been on holiday and had visited Dunoon, Stirling, Edinburgh and several other smaller places. Her closest contact was her husband and he had apparently felt unwell and had had a rigor on 11.9.47. He was an Ear, Nose and Throat surgeon.

Incubation Period. Could not be calculated.

History of Onset. The illness began on 11.9.47 with lassitude, dizziness and fever and these symptoms continued for 3 days. On the day of admission her back and legs began to ache. She complained rather bitterly of headache and neck stiffness and stumbled when she tried to walk.

Examination. Fevered; well marked neck and back rigidity; Kernig's sign; diminution of power of all the muscle groups of the left lower extremity; absent abdominal reflexes.

Cerebro-spinal Fluid.
Protein - 80 mgms.%
Cells - 406 per cu.mm. - 60% lymphocytes 40% polymorphs.
Culture - sterile
Chlorides- 715 mgms.%

Progress. There was no fever on 13.9.47, the abdominal reflexes had returned by 20.10.47 and on discharge from hospital power had returned to the affected limb except for the glutei, the hip flexors and the tibialis anterior.

Remarks. The high percentage of polymorphonuclear cells in the cerebro-spinal fluid was a feature in this case.

Case 50. P.

W.R., male, 13 years. At school.
Kirkhill Cottage,
Dyce, Admitted 14.9.47.
Aberdeenshire. Discharged 6.10.47.

Recent Activities and Contacts. He went to school when the term commenced on 24.8.47 and had not been away from Dyce. None of the other school children were known to be ill at this time.

Incubation Period. Could not be calculated.

History of Onset. He remained from school on 4.9.47 on account of a headache but had no symptoms the next day. On 6.9.47 the headache returned and was followed during the succeeding days by fever, cough, delirium at night, vomiting, stiff legs and spasmodic cramp-like pain in them.

Examination. Neck rigidity; stiffness of the back; Kernig's sign; paralysis of the right half of the soft palate; absent left cremasteric reflex.

Cerebro-spinal Fluid. Protein - 70 mgms.%
Cells - 5 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. Weakness of dorsiflexion of the left foot appeared on 15.9.47 together with minimal weakness of the muscles of the right side of the anterior abdominal wall. On discharge from hospital the hemiparesis of the soft palate and the paresis of the anterior tibial group of muscles persisted.

Remarks. There was clinical evidence of very widespread involvement of the central nervouse system in this case.

Case 51. M.

A.McB., female, 9 years.
48 Carlton Place,
Aberdeen.

St. Margaret's School, Aberdeen.
Admitted 15.9.47.
Discharged 4.10.47.

Recent Activities and Contacts. She had attended the Stonehaven swimming baths two weeks previously and had been to the Braemar Gathering on 5.9.47. Her closest contacts were her two brothers of 10 and 12 years and the former had been in bed for one day with vomiting on 11.9.47. She went to the same school as Case 48.

Incubation Period. If it is assumed that she was infected at school by Case 48, then the incubation period must have been 4 or 5 days, because Case 48 was at school on 9.9.47 and 10.9.47 only.

History of Onset. Illness began on 14.9.47 with headache, followed by anorexia and a suggestion of weakness in the right arm.

Examination. No paresis, but diminution of all the tendon reflexes in the right upper limb. No other abnormal clinical finding.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 36 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. On 16.9.47 the reflexes in the right upper limb were normal and remained so thereafter. Her headache disappeared on the same day.

Case 52. R.

S.C., female, 16 years.
57 Anderson Drive,
Aberdeen.

Aberdeen High School.
Admitted 16.9.47.
Discharged 6.10.47.

Recent Activities and Contacts. She had not been out of Aberdeen. She was in close contact with three children aged 1, 4 and 6 years all of whom remained well. She was also in contact until 20.8.47 with her sister who was a nurse at Gray's Hospital, Elgin. Case 7 attended the same school as herself but was admitted on 21.7.47 during the holidays. The school term started on 1.9.47.

Incubation Period. It seems feasible that her sister might have carried the virus from Elgin where cases of poliomyelitis were being nursed. If this was so, then the minimum incubation period was 12 days.

History of Onset. Headache, fever and abdominal pain on 1.9.47 followed by an asymptomatic interlude until 13.9.47 when the same symptoms returned together with generalised pains and stiffness of her back.

Examination. Fevered; neck rigidity; tenderness in the right iliac fossa; retention of urine; diminution of the right biceps jerk; absent abdominal reflexes.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 39 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. The temperature, which was 105°F. on admission was normal on 19.9.47; on the same day the reflexes were normal and the bladder paralysis had disappeared.

Remarks. The initial fever was exceptionally high.

Case 53. R.

A.C., male, 4 years.
3 Gordonmills Place,
Aberdeen.

At home.
Admitted 18.9.47.
Discharged 8.10.47.

Recent Activities and Contacts. He had not been away from home except on shopping outings with his mother. He had a brother of 9 months who had no symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. He awakened on the night of 17.9.47 with headache, vomiting and fever.

Examination. Fevered; slight upper abdominal tenderness; moderate neck rigidity; diminution of right knee jerk.

Cerebro-spinal Fluid.
Protein - 60 mgms.%
Cells - 360 per cu.mm. - 98% lymphocytes
2% polymorphs.
Culture - sterile
Chlorides- 728 mgms.%

Progress. Weakness of the right face became apparent on 19.9.47 but disappeared the following day. The signs of meningeal irritation and the fever did not resolve until 26.9.47.

Case 54. M.

M.McL., female, 7 years.
345 Holburn Street,
Aberdeen.

Broomhill School, Aberdeen.
Admitted 19.9.47.
Discharged 9.10.47.

Recent Activities and Contacts. She had been at school and at home apart from one visit to the Aberdeen town baths two weeks previously. Her closest contact was an identical twin sister who slept with her but remained symptompfree.

Incubation Period. Could not be calculated.

History of Onset. Developed a very severe headache on 18.9.47, vomited and had marked photophobia. The same evening she complained of pain in the back of her neck and in her legs.

Examination. Fevered; neck rigidity; diminution of right ankle jerk.

Cerebro-spinal Fluid. Protein - 50. mgms.%
Cells - 75 per cu.mm. - 96% lymphocytes
4% polymorphs.
Culture - sterile
Chlorides- 730 mgms.%

Progress. There was no abnormality by 22.9.47.

Remarks. It is of interest that her twin remained symptom-free.

Case 55. P.

D.R., male, 11 months. At home.
330 Anderson Drive North, Admitted 22.9.47.
Aberdeen. Discharged 23.10.47.

Recent Activities and Contacts. He had not been away from home. His two brothers of 2 and 7 years and his sister of 4 years had no symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. On 17.9.47 he was fevered, fretty and refused his feeds. Thereafter, according to his mother, he appeared quite normal except for being rather listless at times until, on 21.9.47, his face was noticed to be twisted.

Examination. Fevered; neck rigidity; complete left facial paralysis.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 27 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. Temperature was normal on 23.9.47. There was no improvement in the facial paralysis on his discharge from hospital.

Case 56. D.

J.McM., male, 10 years. Schoolboy.
9 Beechgrove Gardens, Admitted 23.9.47.
Aberdeen. Died 25.9.47.

Recent Activities and Contacts. He had not been out of Aberdeen during the three weeks prior to admission. He had been to the cinema two weeks previously and to a football match ten days previously. His cousin, with whom he was in close contact, had a 3 day illness starting on 9.9.47 during which there was fever, headache and eye rolling. Case 56 had a brother of 13 years and a sister of 5 years who both remained symptom free.

Incubation Period. If his cousin's illness quoted above was in fact non-paralytic poliomyelitis and he infected Case 56 on the day his symptoms began, then the incubation period was 13 days.

History of Onset. He went to bed on 22.9.47 complaining of nausea. He got up in the evening but complained that everything was "jumping up and down". He vomited at mid-night, thereafter slept soundly, but refused to take his breakfast the next morning. The same day he complained of a sore throat and of pain in his left eye and face. Later he developed frontal headache and stiffness in the neck and back.

Examination. Flushed but mentally alert; temperature 101°F.; pulse rate 120 per minute; respirations 24 per minute; there was slight neck and back rigidity; both iliac fossae tender on palpation; left upper abdominal reflex absent; paresis of the left side of the face and paralysis of the left side of the soft palate.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 108 per cu.mm. - 96 % lymphocytes
4% polymorphs.
Culture - sterile.

Progress. On 25.9.47 he was still fevered but there was no increase in the extent of the paralysis and he was swallowing satisfactorily. On 26.9.47 fluids began to regurgitate down the nose. At mid-day his respirations rose to 58 per minute

and he became cyanosed. There was marked weakness of the diaphragm and intercostal muscles. He was placed in the mechanical respirator but his condition deteriorated rapidly and he died one hour thereafter.

Remarks. There was clinical evidence of both bulbar and spinal implication in this case.

Case 57. **M.**

S.F., male, 4 years.
448 Great Northern Road,
Aberdeen.

At home.
Admitted 23.9.47.
Discharged 14.10.47.

Recent Activities and Contacts. He had not been far away from his home and from a garage in Great Northern Road where his fathervworked. His brother aged 1 year had no symptoms at this time. There was a very indirect contact between this case and Case 7 in that the husband of the domestic servant who was employed in Case 57's home, worked with Case 7's father.

Incubation Period. Even if the infection was carried in the indirect method outlined above, it is impossible to estimate the incubation period.

History of Onset. Developed a sore throat on 9.9.47 which lasted for 3 days. On 22.9.47 he became fevered and complained of pain in his neck, back and legs.

Examination. Fevered; twitching of both sides of face, especially the right; neck rigidity and Kernig's sign; pain and stiffness in the neck and back; absence of the abdominal reflexes on the right side.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 90 per cu.mm. - 98% lymphocytes
2% polymorphs.
Culture - sterile.

Progress. The fever and signs of meningeal irritation had disappearedby 27.9.47 but the abdominal reflexes had not returned until 6.10.47.

Case 58. R.

J.R., male, 10 months. At home.
46 West Road,
Fraserburgh, Admitted 23.9.47.
Aberdeenshire. Discharged 22.10.47.

Recent Activities and Contacts. Apart from being taken out for a walk on 16.9.47 he had not been out of his home. His father worked next door to Case 15's home (Case 15 was admitted on 13.8.47.).

Incubation Period. The very limited activities of this patient suggest that infection was introduced into his home. But even if it is assumed that his father carried the virus from Case 15 - and in view of the time interval, this seems unlikely - the incubation period could not be calculated.

History of Onset. He vomited on 20.9.47 and then had no symptoms for 2 days. Then he began to be fretty and restless and cried when handled. Later gurgling in his throat began and food taken into his mouth regurgitated through his nose.

Examination. Fevered; neck stiffness; twitching of right shoulder; paralysis of the right face and the right side of the soft palate; inability to swallow; diminished tendon reflexes in the right lower extremity and absent right lower abdominal reflex.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 90 per cu.mm. - 96% lymphocytes 4% polymorphs.
Culture - sterile

Progress. He was fed by gastric tube until 27.9.47 and by 15.10.47 all the paralysis had disappeared.

Remarks. It is remarkable that the extensive bulbar paralysis in this case recovered so completely.

Case 59. P.

A.C., female, 14 months. At home.
Buchanhaven,
Peterhead, Admitted 23.9.47.
Aberdeenshire. Discharged 11.11.47.

Recent Activities and Contacts. Apart from being out with her mother while shopping, she had been at home. Her sister of 3 years and her brother of 7 years had no symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. The only abnormality noticed by her parents was frettiness on 16.9.47. The following day her left leg was limp and apparently became more so during the next few days.

Examination. Absent abdominal reflexes; weakness of dorsiflexors of left foot.

Cerebro-spinal Fluid. Protein - 45 mgms.%
Cells - 15 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. There was fever still present on 25.9.47 but no increase in the paralysis. Paresis of the affected muscles was still demonstrable on her discharge from hospital.

Case 60. P.

J.B., female, 15 years.
15 Anderson Avenue,
Aberdeen.

Aberdeen Central School.
Admitted 27.9.47.
Discharged 7.11.47.

Recent Activities and Contacts. The school term started on 1.9.47 and Case 36, who was admitted to hospital on 2.9.47, was at school then for one hour. Her activities included visits to the cinema and church. Her brother of 2 years and sister of 18 years had no symptoms at this time.

Incubation Period. If she was infected by Case 36, the incubation period must have been 22 days.

History of Onset. Slight headache and pain in the shoulders began on 23.9.47. On 26.9.47 the headache was much more severe and she had stiffness of the back, fever and nausea.

Examination. Fevered; slight neck rigidity; absent abdominal reflexes.

Cerebro-spinal Fluid. Protein - 80 mgms.%
Cells - 300 per cu.mm. - 97% lymphocytes
3% polymorphs.
Culture - sterile.

Progress. There was fever until 30.9.47 and with it were marked signs of meningeal irritation. Her back and abdominal muscles, bladder, lower five intercostals and proximal parts of both lower extremities became paralysed during the three days after admission. Recovery was very slow and incomplete. Bladder function returned on 6.10.47, chest expansion appeared full on discharge from hospital, but the abdominal, back and thigh muscles had improved only slightly.

Case 61. M.

A.B., male, 14 years. Farm Worker.
Milton of Fochal,
St. Catherine's, Admitted 29.9.47.
Aberdeenshire. Discharged 20.10.47.

Recent Activities and Contacts. Apart from a visit to the cinema on 25.9.47, he had been at home. He was a close contact of two brothers aged 2 and 8 years and of two sisters aged 5 and 13 years, all of whom remained free from symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. He was listless and tired on 27.9.47 but felt well next morning. on the evening of that day he developed severe frontal headache and pain in the back of his neck.

Examination. Neck rigidity and Kernig's sign were present; no other clinical abnormality.

Cerebro-spinal Fluid. Protein - 45 mgms.%
Cells - 24 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. Uninterrupted recovery.

Case 62. R.

H.W., female, 13 years. At school.
Fordmouth,
Auchnagatt, Admitted 1.10.47.
Aberdeenshire. Discharged 23.10.47.

Recent Activities and Contacts. Apart from going to school, she had not been away from home. It appeared that the school teacher had been absent for a week from 26.9.47 with "suspected poliomyelitis" and an unspecified number of the pupils were ill about this time with "sickness". Her six brothers and sisters - ages ranging from 1 to 11 years - remained well.

Incubation Period. Could not be calculated.

History of Onset. Headache began on 26.9.47; the next day she developed neck stiffness, epistaxis and there was delirium at night; pain in both lower limbs occurred on 29.9.47.

Examination. Fevered; neck rigidity and Kernig's sign; tenderness in the right iliac fossa; left facial weakness.

Cerebro-spinal Fluid. Protein- 55 mgms.%
Cells - 72 per cu.mm. - 98% lymphocytes
2% polymorphs.
Culture- sterile.

Progress. The facial paresis was no longer evident on 2.10.47 but retention of urine developed on that day and bladder function was not restored until 4.10.47.

Case 63. P.

E.M., female, 1½ years. At home.
12 St. Peter's Street,
Peterhead, Admitted 1.10.47.
Aberdeenshire. Discharged 14.11.47.

Recent Activities and Contacts. Apart from short walks in the surrounding gardens, she had not been out of the house. Her sister aged 7 years and her brother who was born on 29.9.47 had no abnormal symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. She vomited and was fevered on 27.9.47; next day she developed generalised tenderness and stiffness of her neck.

Examination. Fevered; slight neck rigidity; tenderness in the upper abdomen; abdominal reflexes and left ankle jerk absent.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 30 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. The temperature was normal on 3.10.47, but on the same day paralysis of the upper part of the left arm appeared. Recovery was complete except for slight weakness of the left deltoid.

Remarks. This case was, during the prodromata, in close contact with a newly born baby who, on enquiry one month later, had shown no symptoms of illness from birth.

Case 64. M.

J.B., male, 1½ years. At home.
70 Wales Street, Admitted 2.10.47.
Aberdeen. Discharged 22.10.47.

Recent Activities and Contacts. He had been taken to the cinema two weeks previously. He had not been away from Aberdeen. His only close contacts were his parents and neither had any symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. He had had frequent stools for the previous two weeks. On 1.10.47 he was off his food and unable to sit up and movement of his legs appeared to cause pain.

Examination. Fevered; neck and spinal rigidity.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 240 per cu.mm. - 94% lymphocytes
6% polymorphs.
Culture - sterile.

Progress. Temperature became normal on the 4th day after admission but neck stiffness remained until 10.10.47.

Case 65. R.

B.P., male, 3 years.
120 Stockethill South,
Aberdeen.

At home.
Admitted 2.10.47.
Discharged 27.10.47.

Recent Activities and Contacts. His time was mainly spent in palying with 9 or 10 other children who lived in close proximity to his own home. None of these children nor his brother of 1 year were known to be ill at this time.

Incubation Period. Could not be calculated.

History of Onset. Abdominal pain began on 22.9.47 and continued until admission to hospital. Other symptoms were nausea, fever, pain in the back and neck and stiffness of the legs.

Examination. Fevered; right facial weakness; Kernig's sign; absence of both ankle jerks; weakness of left sterno-mastoid.

Cerebro-spinal Fluid. Protein - 45 mgms.%
Cells - 105 per cu.mm. - 94% lymphocytes 6% polymorphs.
Culture - sterile.

Progress. Fever settled on 3.10.47 but retention of urine occurred on that day. Thereafter he steadily improved and the facial and sterno-mastoid paresis finally disappeared on 11.10.47.

Case 66. P.

S.G., female, 14 years. Baker's assistant.
Mill of Dyce Cottage,
Dyce, Admitted 2.10.47.
Aberdeenshire. Discharged 7.11.47.

Recent Activities and Contacts. She worked in a baker's shop in Aberdeen and travelled from Dyce every day. She was an adopted child and her closest contact was a girl of 23 years who had no symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. She was listless, fevered and tired on 28.9.47 and later developed headache, vomiting and pain in the back of her neck. She could not sleep on the night of 1.10.47 owing to pain in her legs and stiffness in her legs.

Examination. Fevered; neck and back rigidity; paresis of all muscles of the right lower extremity except the abductors of the thigh; paresis of the adductors and quadriceps of the left thigh; marked tenderness over both sciatic nerves and manoevres to stretch the nerves caused severe pain; the muscles of the anterior abdominal wall seemed lax, but their power could not be satisfactorily tested owing to the rigidity of the back.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 80 per cu.mm. - 98% lymphocytes
2% polymorphs.
Culture - sterile.

Progress. Fever persisted until 7.10.47 and signs of meningeal irritation until 16.10.47. Complete paralysis of the anterior abdominal muscles and minimal paresis of the back extensors became evident on 3.10.47. Paresis of the flexors of the left thigh appeared on 4.10.47. Recovery was slight, the back muscles being the only ones in which it was complete.

Remarks. Bilateral sciatic pain and tenderness were striking features in this case.

Case 67. M.

A.S., male, 7 years.
413 Holburn Street,
Aberdeen.

Broomhill School, Aberdeen.
Admitted 6.10.47.
Discharged 26.10.47.

Recent Activities and Contacts. Apart from going to school and to the cinema on one occasion a week previously, he had not been away from home. His father stated that two of his relatives developed "withered legs" - a sister in 1914 and a cousin in 1905 - both after acute illnesses. The patient had six close contacts, their ages ranging from 1 to 13 years, and all remained symptom free. Case 54 attended the same school.

Incubation Period. If the patient was infected by Case 54, then the incubation period must have been at leats 9 days.

History of Onset. Headache on the evening of 27.9.47 followed by an asymptomatic interlude until 4.10.47 when the headache returned and he complained of pain when his neck was moved. Vomiting occurred and sleeplessness developed on 5.10.47.

Examination. Fevered; slight neck rigidity; absent upper abdominal reflexes.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 45 per cu.mm. - all lymphocytes.
Culture - sterile.

Progress. There was no neck stiffness of fever on 6.10.47 but the abdominal reflexes did not return until 14.10.47.

Remarks. The family history in this case is interesting.

Case 68. M.

J.D. male, 13 years.
66 Queen's Road,
Aberdeen.

Gordonstown School.
Admitted 7.10.47.
Discharged 25.10.47.

Recent Activities and Contacts. He had been on holiday at Ballater but was at home for ten days before returning to school on 26.10.47. He was in contact with Case 51's brother 14 days before the onset of his own illness. His mother's brother developed a transient paralysis of both legs after an illness 45 years ago.

Incubation Period. It is possible that the virus was conveyed to the patient by Case 51's brother,, in which case the incubation period was 14 days.

History of Onset. Frontal headache began on 5.10.47 and he later developed fever and vomiting.

Examination. No abnormal clinical findings.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 26 per cu.mm. - all lymphocytes
Culture - sterile
Chlorides- 740 mgms.%

Progress. Uninterrupted recovery.

Case 69. P.

C.M., male, 5 years.
184 Bankhead Road,
Bucksburn,
Aberdeenshire.

Stoneywood School, Bucksburn.

Admitted 12.10.47.
Discharged 1.11.47.

Recent Activities and Contacts. Apart from going to school he had not been away from home. His mother stated that several other children at school were "suspect cases" of poliomyelitis, but the grounds for these suspicions could not be elucidated. His brother of $2\frac{1}{2}$ years had no symptoms at this time. There was a remote connection between this case and Case 2 in that a deaf and dumb boy, who lived in the adjoining house in Bankhead Road, had been a daily contact with Case 2 until the latter's admission to hospital on 19.2.47.

Incubation Period. Even if it is assumed that his neighbour referred to above was the carrier of the virus to this patient, the incubation period could not be calculated.

History of Onset. The illness began with headache and vomiting on 9.10.47. Thirst and restlessness at night followed, and on 11.10.47 his mouth twitched and he was unable to sit up in bed.

Examination. Fevered; almost complete paralysis of the right side of the face; absent abdominal reflexes.

Cerebro-spinal Fluid. Protein - 45 mgms.%
Cells - 56 per cu.mm. - 98% lymphocytes
2% polymorphs.
Culture - sterile.

Progress. There was no fever on 13.10.47. On discharge from hospital, weakness of the upper part of the right face remained.

Case 70. P.

R.McI., male, 11 weeks. At home.
13 St. John's Road,
Bucksburn, Admitted 13.10.47.
Aberdeenshire. Discharged 7.11.47.

Recent Activities and Contacts. Apart from a visit to the Child Welfare Clinic at St. John's Road for weighing on 5.10.47 he had not been away from home. Three weeks previously - 22.9.47 - his mother had been in contact with Case 73, who was admitted on 15.10.47.

Incubation Period. If the virus was carried from Case 73, the maximum incubation period possible was 16 days.

History of Onset. The illness began on 8.10.47 with rapid breathing and irritability when handled. Then followed fever, lassitude and complete loss of voice. His legs became limp and he refused his feeds.

Examination. Neck rigidity; twitching of right face; very feeble cry; very limited movement of intercostal muscles; diminished power in left deltoid, biceps and triceps; complete paralysis of left lower extremity.

Cerebro-spinal Fluid. Protein - 80 mgms.%
Cells - 30 per cu.mm. - all lymphocytes
Culture - sterile
Chlorides- 726 mgms.%

Progress. The left arm recovered completely and the intercostals partially, but in the left lower limb, only the quadriceps, psoas, glutei and hamstrings showed improvement, and that was minimal. The voice recovered completely.

Remarks. The was the youngest patient of the series.

Case 71. P.

A.S., male, 1½ years.
9 School Terrace,
Aberdeen.

At home.
Admitted 13.10.47.
Discharged 23.11.47.

Recent Activities and Contacts. Apart from visits to his grandmother in Aberdeen, he had not been away from home. Two brothers of 10 and 15 years remained symptom-free at this time. His father had poliomyelitis in 1905 and still shows evidence of some wasting of the left leg.

Incubation Period. Could not be calculated.

History of Onset. On 8.9.47 it was thought that the child was cutting a tooth as he was fretty and slightly fevered and off his food. The next day there was still anorexia and he refused to walk.

Examination. The only abnormality detected clinically was some weakness of dorsiflexion of the left foot.

Cerebro-spinal Fluid. Protein - 55 mgms.%
Cells - 9 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. Although he was apyrexial throughout, the paralysis progressed for two weeks, by which time the left quadriceps, calf muscles, anterior tibials and intrinsic muscles of the foot were completely flaccid and the left hamstrings were very weak. On discharge from hospital the hamstrings and quadriceps had recovered, but the muscles in the distal part of the limb showed no improvement.

Remarks. Interesting features in this case were the family history and the progression of the paralysis.

Case 72. M.

I.S., male, 7 years.
10 Kirk Place,
Cults,
Aberdeenshire.

Cults School.

Admitted 15.10.47.
Discharged 4.11.47.

Recent Activities and Contacts. Apart from going to school, he had not been away from home. His cousin, G.S., had poliomyelitis with paralysis 3 years previously (notified 12.8.44). One other boy at the same school had been ill with a headache a week before Case 72 took ill.

Incubation Period. Could not be calculated.

History of Onset. Headache began on 6.10.47 and lasted two days. He had no symptoms from then until 13.10.47 when the headache returned. Other symptoms which occurred next day were neck and back stiffness, anorexia and some pain in the arms and legs.

Examination. Fevered; neck rigidity; Kernig's sign.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 108 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. Temperature was normal on 16.10.47 and the next day there was no evidence of meningeal irritation and he had no complaints.

Case 73. P.

G.M., male, 9 months. At home.
213 Stoneywood Road,
Bucksburn, Admitted 15.10.47.
Aberdeenshire Discharged 7.11.47.

Recent Activities and Contacts. He had been to Cruden Bay in Aberdeenshire for a day three weeks previously and to Aberdeen for a day two weeks previously but had otherwise been at home. His contact with case 70's mother has been mentioned already in connection with that case. The first symptom of both these patients appeared on 8.10.47 and it seemed feasible that the virus, coming from some common source, invaded Case 73 directly and was carried to Case 70 by his mother.

Incubation Period. If the theory of infection outlined above was true, then the incubation period must have been at most 16 days.

History of Onset. On 8.10.47 he was very listless and vomited several times. Two asymptomatic days followed thereafter but on 11.10.47 frettiness and fever occurred and continued until the day of admission when his right arm was noted to be limp.

Examination. Flaccid paralysis of the right upper extremity except for some power retained in the flexors and extensors of the fingers.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 42 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. There was considerable improvement in all the affected muscles with the exception of the deltoid, which showProgress. There was considerable improvement in all the affected muscles with the exception of the deltoid, which showed no improvement.

Case 74. P.

C.W., male, 4 years.
2 Viewfield Gardens,
Aberdeen.

Miss Oliver's School, Aberdeen.
Admitted 18.10.47.
Discharged 14.11.47.

Recent Activities and Contacts. He had been at school and at home only. His brother of 1 year ahd no symptoms at this time but his mother had an unexplained attack of vomiting on 8.10.47. Case 75's son attended the same school but neither he nor any of the other pupils were known to have had any symptoms at this time.

Incubation Period. It seems feasible that Case 75's son and this patient were both exposed to the virus at school and that while Case 74 developed the disease, Case 75's son carried it to his mother and remained symptom-free himself.

History of Onset. Fever, frettiness, anorexia and abdominal pain developed on 14.10.47 and were followed by three asymptomatic days. On the day of admission headache occurred and fever returned and he complained of pain in the back.

Examination. Fevered; neck rigidity; left supinator jerk greater than right.

Cerebro-spinal Fluid.
Protein - 70 mgms.%
Cells - 720 per cu.mm. - 98% lymphocytes 2% polymorphs.
Culture - sterile
Chlorides- 728 mgms.%

Progress. Weakness of the dorsiflexors of the right foot and adductors and flexors of the right knee first became apparent on 29.10.47, but, with the exception of the tibialis anterior, all recovered completely.

Remarks. The delayed appearance of the paralysis was a feature of this case.

Case 75. D.

Mrs. H.E., 40 years. Housewife.
9 Forest Road, Admitted 18.10.47.
Aberdeen. Died 22.10.47.

Recent Activities and Contacts. Once each week for the previous three weeks she had been to Braemar for one day. She went to a dancing class once each week and to the cinema one week prior tomadmission. Apart from these outings, her activities had been those of a housewife. She had a daughter of 8 years and a son of 4 years who remained symptom-free. Her son attended the same school as Case 74, whose first symptom developed on 14.10.47.

Incubation Period. It seemed unlikely that this patient's son carried the virus to his mother from Case 74 the day the latter's first symptom started as this would mean an incubation period of only one day for Case 75.

History of Onset. On 15.10.47 she noticed pain in her back. On 16.10.47 the pain grew worse and in the evening became "almost unbearable". She slept very little that night and the following one. On 18.10.47 she complained of very severe headache and vomited several times. She noticed that she had lost a good deal of power in her legs.

Examination. A very alert and co-operative patient; she understood the nature of her illness and her thoughts were mainly concerned with the type of wheel chair she would get if her legs did not recover; temperature 100°F.; pulse rate 86 per minute; respirations 22 per minute; both lower limbs completely flaccid and powerless except for the left quadriceps, in which there was still some movement; abdominal reflexes absent; retention of urine; well marked neck rigidity; stiffness of the back, making it difficult to estimate the tone of the abdominal muscles.

Cerebro-spinal Fluid. Protein - 70 mgms.%
Cells - 108 per cu.mm. - 96% lymphocytes
4% polymorphs.
Culture - sterile.

Progress. On 19.10.47 the abdominal and back muscles were paralysed and later on that day there was weakness of

both upper arms, the intercostals and the diaphragm. Her temperature, pulse rate and respiratory rate all rose and she had difficulty in coughing up mucus from her throat. She was much concerned about her difficulty in breathing and was placed in the mechanical reppirator. On 20.10.47 when she was temporarily removed from the respirator for nursing purposes she became cyanosed within 10 seconds and was given oxygen under pressure. In the afternoon of 21.10.47 she became unable to swallow. She had a complete flaccid quadriplegia and paralysis of all the muscles of the neck and trunk and died the following day.

Post Mortem Examination. The Brain and Spinal Cord were oedematous and very congested and the Meninges were likewise affected.

Histological Examination. There was marked congestion and massive infiltration with polymorphonuclear and small round cells in the spinal cord. There was also well marked peri-vascular "cuffing" and no healthy neurones were seen in the anterior horns of either side. The medulla showed congestion and peri-vascular "cuffing" but to a lesser degree than the spinal cord. Neuronophagia was seen. Similar appearances were seen in the mid-brain, but the motor cortex showed congestion only.

Remarks. This was a case of acute ascending polio-encephalo-myelitis similar to that described by Landry and discussed on page

Case 76. P.

D.M., male, 3 years.
22 St. Andrew's Street,
Aberdeen.

At home.
Admitted 19.10.47.
Discharged 24.11.47.

Recent Activities and Contacts. Two weeks previously he had been to the cinema but had otherwise been at home and playing about the streets. His sister aged 7 and 18 other children living in the same building were not known to have any symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. On 16.10.47 he was fretty, fevered and complained of a sore neck and a sore throat. Later he ground his teeth and vomited several times. His symptoms had largely disappeared on 18.10.47 but it was noticed then that his right arm was limp.

Examination. Fevered; flaccid paralysis of the whole of the right upper extremity.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 24 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. There was slight return of power in all the muscles distal to the right elbow but none in those of the upper arm.

Case 77. P.

S.C., female, 1½ years. At home.
139 Victoria Road,
Torry, Admitted 21.10.47.
Aberdeen. Discharged 19.11.47.

Recent Activities and Contacts. She had not been out of Aberdeen but she and her parents had moved their home from Broomhill Road to Victoria Road on 15.10.47.

Incubation Period. Could not be calculated.

History of Onset. Fever and anorexia appeared on 17.10.47 and next day she was noticed to drag her right leg when she attempted to walk.

Examination. Fauces injected; lower abdominal reflexes absent; doubtful weakness of right lower extremity; inability to sit up in bed.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 48 per cu.mm. - 96% lymphocytes
4% polymorphs.
Culture - sterile.

Progress. Paresis of all the muscle groups in the right lower extremity except the plantar flexors and the intrinsic muscles of the foot became apparent by 26.10.47, but on discharge from hospital the only paresis apparent was in the dorsiflexors of the foot.

Case 78. P.

J.H., male, 1½ years. At home.
North Coullie,
Udny, Admitted 5.11.47.
Aberdeenshire. Discharged 28.11.47.

Recent Activities and Contacts. He had been to Newburgh, Aberdeenshire on 9.10.47 but had otherwise been at home except for short walks locally. His three brothers aged 4, 9 and 12 years had no symptoms at this time, but there had apparently illness with headache and vomiting amongst several of the pupils at Udny Green School - the school that his two elder brothers attended.

Incubation Period. Could not be calculated.

History of Onset. On 29.10.47 he was fevered and listless and was thought to be teething. Then he had no further symptoms until 4.11.47 when his neck was noted to be stiff and he appeared to have abdominal pain.

Examination. Neck rigidity and Kernig's sign; weakness of left face; diminished tone in right lower extremity with absent ankle jerk.

Cerebro-spinal Fluid. Protein - 50 mgms.%
Cells - 36 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. The facial paresis had disappeared by 11.11.47 but it became clear that the loss of tone in the right lower limb was associated with paresis of the dorsiflexors of the foot. This muscle group had not completely recovered on his discharge from hospital.

Case 79. M.

I.K., female, 3 years.
9 Seton Drive,
Aberdeen.

At home.
Admitted 8.11.47.
Discharged 28.11.47.

Recent Activities and Contacts. She had not been away from home except to Sunday School. She was the youngest of a family of twelve - their ages ranging from 2 to 23 years. None of these others had any symptoms at this time.

Incubation Period. Could not be calculated.

History of Onset. On 6.11.47 she had transitory pain in her right ear. Then she had no further symptoms until the day of admission. At mid-day she asked for a "piece" and appeared to be perfectly normal but a few minutes later she suddenly became cyanosed and frothed at the mouth. She cried to her mother that she could neither see nor hear and in a few more minutes became unconscious and remained so for half an hour.

Examination. Fully conscious; right pupil larger than the left; rigidity of the neck and back; Kernig's sign.

Cerebro-spinal Fluid.
Protein - 50 mgms.%
Cells - 45 per cu.mm. - all lymphocytes
Culture - sterile

Progress. The evidence of meningeal irritation was no longer present by 15.10.47 and her progress otherwise was uneventful.

Remarks. The very acute onset of the disease in this case was a striking feature.

Case 80. M.

H.M., male, 9 years.
7 Sandilands Drive,
Aberdeen.

Kittybrewster School, Aberdeen.
Admitted 15.11.47.
Discharged 4.12.47.

Recent Activities and Contacts. He had not been out of Aberdeen but he had been to the cinema a great deal and three weeks previously he had attended the Aberdeen town swimming baths. Casev 11 lived in the same road, but had the acute episode of the disease 3 months previously and there had been no known contact between the two. There was no illness at the time amongst his close contacts - a sister of 2 years and two brothers of 15 and 17 years, the former of whom slept in the same bed as the patient. Case 46 attended the same school, but her first symptom appeared on 3.9.47.

Incubation Period. If he was infected at school by Case 46, the incubation period must have been more than two months.

History of Onset. Frontal headache and vomiting appeared on 14.11.47. He was very restless at night and complained of abdominal pain and photophobia.

Examination. Minimal neck rigidity only.

Cerebro-spinal Fluid. Protein - 40 mgms.%
Cells - 36 per cu.mm. - all lymphocytes
Culture - sterile.

Progress. Uneventful recovery.

Case 81. P.

B.E., male, 12 weeks.
120 Ugie Park,
Peterhead,
Aberdeenshire.

At home and at Peterhead Day Nursery.

Admitted 22.11.47.
Died 15.12.47.

Recent Activities and Contacts. He attended the Day Nursery for several hours each day but had otherwise been at home. There was no illness amongst any of the other babies in the nursery at this time.

Incubation Period. Could not be calculated.

History of Onset. On 10.11.47 he became very fretty and had a discharging nose. The next day he refused most of his feeds and on 12.11.47 his mother thought that he had lost some of the power in his left leg.

Examination. Eager and alert; paresis of all the muscle groups in the left lower extremity.

Cerebro-spinal Fluid.
Protein - 20 mgms.%
Cells - 2 per cu.mm.
Culture - sterile
Chlorides- 759 mgms.%

Progress. The condition of the left leg remained unchanged but the child developed acute gastro-enteritis on 12.12.47 and died on 15.12.47.

Remarks. There is no doubt that this child died from acute gastro-enteritis and not primarily from poliomyelitis. The case has therefore not been classified as a poliomyelitis death in this series.

BIBLIOGRAPHY.

ADAMS, F. - The genuine works of Hippocrates, Translated from the Greek by Francis Adams. Vol. 1. The Epidemics Book, p. 362. 1849.

ARMSTRONG, C. and LILLIE, R.D. - Pub. Hlth. Rep. Wash. 49: 1019, 1934.

AYCOCK, W.L. - Am. J. Hyg. 7: 791, 1927.

AYCOCK, W.L. - J. Prev. Med. 3: 245, 1929a.

AYCOCK, W.L. - ibid. 3: 252, 1929b.

AYCOCK, W.L. and EATON, P. - Am. J. Hyg. 4: 356, 1924a.

AYCOCK, W.L. and EATON, P. - ibid. 4: 681, 1924b.

AYCOCK, W.L., and LUTHER, E.H. - J. Prev. Med. 3: 103, 1929.

BARLOWE, W.H. - Liverp. and Manch. M. and S. Rep. 6: 1, 1878.

BATTEN, F.E. - "Acute Poliomyelitis, Its Nature and Treatment". Reprinted from "Brain" Vol. 39, 1916. John Bale, Sons and Danielson, Ltd., London, 1916.

BODIAN, D. -. J.A.M.A. 134: 1148, 1947.

BOWERMAN, W.G. - Arch. paediat. 62: 57, 1945.

BRADLEY, W.H. and GALE, A.H. - Monthly Bull. Min. Hlth. P.H.L.S. 7: 56, 1948.

BRAMWELL, B. - Edin. M.J. 26: 745, 1881.

BRAMWELL, B. - Clin. Stud. 3: 189, 1905.

BRAMWELL, B. - ibid. 6: 346, 1908a.

BRAMWELL, B. - ibid. 6: 371, 1908b.

BRAMWELL, E. - Edin. M.J. 22: 345, 1919.

BROWN, G.C., FRANCIS, T. Junr. and PEARSON, H.E. - J.A.M.A. 129: 121, 1945.

BURNET, F.M. - "Virus as Organism". Harvard Univ. Press, 1945.

BUZZARD, T. - "Clinical Lectures on Diseases of the Nervous System". J.&A. Churchill, London, 1882.

CASEY, A.E., FISHBEIN, W.I. and BUNDESEN, H.N. - J.A.M.A. 129: 1141, 1945.

CAMPBELL, B.P. - Edin. M.J. 10: 501, 1913.

CAPPELL, D.F. - Tr. Med. Chir. Soc. Glas. 25: 78, 1930.

CENSUS of SCOTLAND, 1931 - H.M. Stationary Office, 1931.

CREIGHTON, C. - "A History of Epidemics in Britain" Cambridge Univ. Press, Vol. 1 - 1891. Vol. 2 - 1894.

CROCKATT, W. - Edin. M.J. 13: 434, 1868.

CRUICKSHANK, J.N. - Glas. M.J. 120: 44, 1933

DALE, J. - M.J. Australia, 152: 258, 1928.

DOULL, J.A., FERREIRA, J. and PARREIRAS M. - J. Prev. Med. 1: 503, 1927.

EPIDEMIC of Poliomyelitis in New York City in 1916. Published under the direction of the Department of Health of New York City, 1917.

FLEMING, G.B. - Glas. M.J. 115: 105, 1931.

FISHBEIN, M. - "A Bibliography of Infantile Paralysis 1789 - 1944". J.B. Lippincott Co., 1946.

FINLAYSON, - Glas. M.J. 16: 454, 1881.

FORSBECK, F.C. and LUTHER, E.H. - New Eng. J. Med. 203: 1115, 1930.

FOX, M.J. - J.A.M.A. 110: 1268, 1938.

FRANCIS, T. Junr., KRILL, C.E., TOOMEY, J.A. and MACK, W.N. - J.A.M.A. 119: 1392, 1942.

FROST, W.H. - Hyg. Lab. Bull. No. 90, 1913. Washington Govt. Printing Office, 1913.

FULTON, J.F. - "Physiology of the Nervous System" Oxford Med. Publications, 1943, 2nd ed.

GILL, C.A. - "The Genesis of Epidemics" Balliere, Tindall and Cox, London, 1928.

GILPIN, S.F., MOERSCH, F.P. and KERNCHAN, J.W. - Arch. Neurol. and Psychiat. 35: 937, 1936.

GORDON, F.B., SCHABEL, F.M. Junr., CASEY, A.E. and FISCHBEIN, W.L. - J.A.M.A. 135: 884, 1947.

HALLIDAY, J.L. - Med. Officer 41: 155, 1929.

HALLIDAY, J.L. - Tr. Med. Chir. Soc. Glas. 25: 56, 1930.

HAMMOND, - Diseases of the Nervous System, 1876, quoted by Stephen, 1886.

HARRINGTON, A.W. and TEACHER, J.H. - Glas. M.J. 85: 331, 1916.

HEALTH BULLETIN - 6: 4, 1948.

HIRSCH, A. - "Handbook of Geographical and Historical Pathology". Translated from the second German edition by Charles Creighton, London, 1883.

HOLMES, G. - B.M.J. 2: 37, 1917.

HORSTMAN, D.M. - Lancet 1: 273, 1948.

HOWE, H.A. and BODIAN, D. - J. Exp. Med. 81: 247, 1945.

INTERNATIONAL COMMITTEE for the Study of Infantile Paralysis. The Williams and Wilkins Co., Baltimore, 1932.

LANCET - 1: 541, 1948.

LANDES, R., REICH, J.P. and PERLOW, S. - J.A.M.A. 116: 2482, 1941.

LAVINDER, C.H., FREEMAN, A.W. and FROST, W.H. - U.S. Pub. Hlth. Bull. No. 91, 1918.

LEVADITI, C., KLING, C. and LEPINE, P. - Bull. Acad. de Med. Paris, 105: 190, 1931

LOCKHARD, J.G. - "The Life of Sir Walter Scott, Bart." Richard Clay and Sons Ltd., London. 1906.

LOTSCHER, P. - Helvet. med. Acta. 8: 578, 1941. (quoted by Fischbein, 1946)

LOWE, H.B. - Proc. Roy. Soc. Med. 5: 76, 1912.

McALPINE, D., BUXTON, P.H., KREMNER, M. and COWAN, D.J. - B.M.J. 2: 1019, 1947.

MELNICK, J.L. and HORSTMANN, D.M. - J. Exp. Med. 85: 287, 1947.

MONRO, T.K. - Tr. Med. Chir. Soc. Glas. 10: 147, 1911.

NESS, B.R. - Tr. Med. Chir. Soc. Glas. 10: 129, 1911.

PIRIE, J.H.H. - Rev. Neurol. Psychiat. 8: 70, 1910.

REPORTS of the MEDICAL OFFICER of HEALTH for the CITY and COUNTY of ABERDEEN, 1913 - 1946.

RIVERS, T.M. and SCOTT, T.F.M. - Science 81: 439, 1935.

ROGERS, - "Fevers in the Tropics". Henry Frowde, London, 1919.

RUBIE, J. and MOHUN, A.F. - B.M.J. 1: 338, 1949.

RUSSELL, W.R. - B.M.J. 2: 1023, 1947.

SABIN, A.B. - J.A.M.A. 134: 749, 1947.

SHELDON, W. - "Diseases of Infancy and Childhood". J. & A. Churchill, London, 1946.

SINKLER, W. - Am. J. Med. Sc. 69: 348, 1875.

SOHIER, R. et GAUBERT, Y. - La Presse Medicale Travaux Originaux. No. 30: p. 433, 1946.

SLADE, J. de R. - New Eng. J. Med. 234: 753, 1946.

STEPHEN, J.L. - Glas. M.J. 26: 275, 1886.

SWEETMAN, W.P. - Pub. Hlth. 61: 55, 1948.

THELANDER, H.E. and SHAW, E.B. - Am. J. Dis. Child. 61: 1131, 1941.

TIDY, H. - Practitioner, 155: 361, 1945.

TIDY, H. - Lancet, 2: 819, 1946.

TOOMEY, J.A. - Am. J. Dis. Child. 45: 1211, 1933.

TOOMEY, J.A. and AUGUST, M.H. - J. Paediat. 1: 601, 1932.

UNDERWOOD, M. - A treatise on Diseases of Children, Vol. 2. "Debility of the Lower Extremities" p. 53 Printed for J. Matthews, London, 1789. 2nd ed. (quoted by Fischbein, 1946)

van ROOYEN, C.E. - "Virus Diseases of Man" Oxford University Press, 1940.

WALLGREN, A. - Act. Paediat. 4: 158, 1925.

WEISENBERG, T.H. - J. Nerv. & Ment. Dis. 46: 443, 1917.

WICKMAN, I. - Nervous and Mental Diseases. Monograph Series No. 16, 1913, Authorised English translation by Dr. W.J.M.A. Maloney.

WRIGHT, B.W. - J. Urol. 36: 618, 1936.

MAP OF THE CITY OF ABERDEEN