

is thought to be possible, provided that complications do not arise during labour.

The selection of cases for trial and the management of labour are of first importance. A suggested routine is described.

The decision as to the management of future confinements may be based upon the experience gained from a trial labour.

REFERENCES

Bourne, A. (1934). *British Medical Journal*, 2, 963.
 Brown, R. C. (1935). *Ibid.*, 1, 1251.
 Cook, F. (1934). *Guy's Hosp. Gaz.*, 48, 46.
 Hunter, W. (1937a). *Newc. med. J.*, 17, 71.
 — (1937b). *Clin. J.*, 66, 469.
 Walker, A. (1936). *Proc. roy. Soc. Med.*, 29, 1477.

ACUTE ANTERIOR POLIOMYELITIS
 FOUR SIMULTANEOUS CASES IN A SCHOOL

BY

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The occurrence of four simultaneous cases of anterior poliomyelitis in a boys' boarding school has afforded an interesting opportunity for inquiry into the possible method of infection, and the evidence suggests a route which might be worthy of further experimental investigation.

History of the Outbreak

On July 15 four boys were admitted to the sanatorium from the same house for different groups of symptoms, which are given below in detail in each case history. No boy seemed really ill, and all were put to bed for observation. By the next morning it seemed evident that they bore a major resemblance to one another, each suggesting a more or less severe disturbance of the central nervous system in its earliest stage. A fifth case was admitted as suspicious, but it was soon seen that no physical signs were present. There was sufficient justification to call in expert advice, and the four boys were seen that afternoon by Sir Charles Wilson and Drs. Camps and Maclean, who confirmed a provisional diagnosis of acute anterior poliomyelitis. Lumbar puncture was done on three of the cases. On July 17 three of the boys showed definite signs of paralysis of more than one group of muscles; the fourth showed definite weakness in one muscle of the arm only, and continued alteration of the abdominal reflexes. The fifth was definitely settled as negative.

On July 22 Dr. Brinton saw the four cases after paralysis had set in, and assessed the amount of paralysis in each case. Further details of the cases are supplied in the table.

Shortly after the notification of these cases information was received that six others had occurred during the previous fortnight in villages about ten miles away, on the opposite side of Braintree. During the subsequent eight weeks cases were notified scattered throughout the district of Essex surrounding Braintree, which is the largest town in the neighbourhood. But there were many villages and small towns within the area which were without a case. The accompanying chart shows all cases on the days of their first development, and their distances from Braintree, where the largest number of cases eventually occurred.

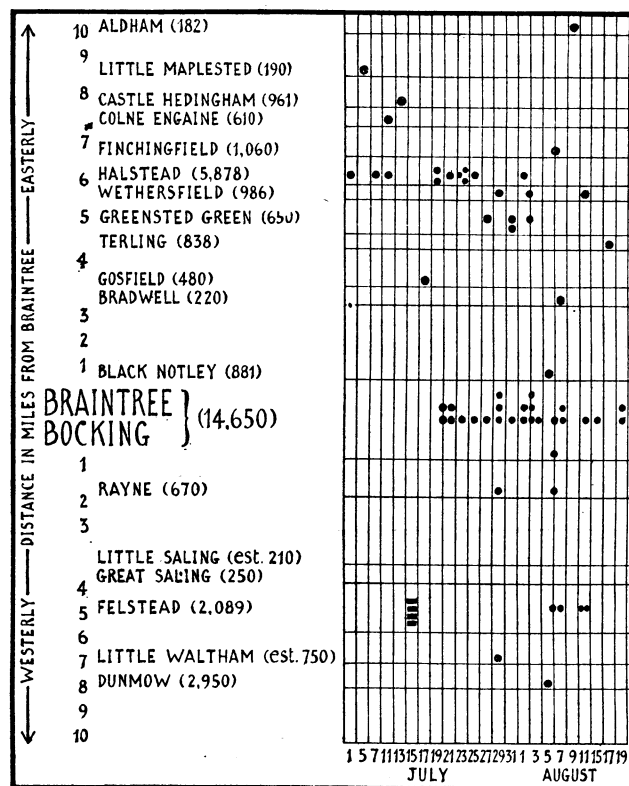


CHART I.—Date of onset of cases occurring in Braintree and the surrounding district. (The figures in parentheses refer to the population.)

Possible Sources of Infection to the Four Schoolboys

As the four cases in the school all occurred on the same day, and none are recognized as having occurred there before or afterwards, a close analysis was made of the daily habits and lives of these four boys in order to discover any factors common to all four which would point to the source of infection. It certainly appears unlikely that the presence of a carrier in the house was the explanation, as one would then expect a series of cases at intervals rather than such an isolated batch. But it is important to note that cases did appear later in the village, and that two of them occurred in the family of one of the maids employed by and living in the house. These cases were attributed to the maid having carried the infection home. As she was in the house for the rest of the term and no further cases occurred there, and as one attempt to infect a monkey from her proved negative, there is no actual evidence in our present state of knowledge that she was in fact a carrier. Analysis shows:

1. All the four cases occurred in the same house of fifty boys. This house has separate feeding arrangements and a separate domestic staff from the rest of the school.
2. Inside the house only two of the boys slept in the same dormitory; these two were quite friendly and sat near each other in the dining-room. The other two were in different dormitories, and each was unconnected with the others in any activity.
3. In the house-room all four were in different corners, and one of them was only present occasionally as a house prefect.
4. In the school all four were well scattered, being in different classes and other categories.

Table showing Particulars of Cases

	School Details	Previous Symptoms	Symptoms on Admission	Physical Signs on 16/7/38	Later Physical Signs	Lumbar Puncture	Muscles	
							Paralysed	Weak
Case 1	Aged 17. Form VI. Dormitory 3	Two weeks before dull diffuse headache. One week before felt run down, due to pressure of exams.	Headache. Anorexia. Dizziness. Constriction around chest. Hazy vision	Temp. 103° F. Tongue furred. Nasopharynx injected. Optic fundus congested. Stiff neck; weakness in trunk muscles and dorsiflexors of feet. Reflexes: Pupils react R.>L. Absent abdominals. K.J. very diminished. Plantar flexor weak	Diminished reflexes of upper limbs. Temperature fell to 98.4° in six days; remained normal	Specimen grossly contaminated with blood		All upper limb muscles including pectorals. Trunk muscles; spinal; recti abdom. R>L. Dorsiflexors of glutei and ankles
Case 2	Aged 16 Form IVb. Dormitory 1	Epistaxis. Vomited one night before	Headache. Anorexia. Dizziness. Stiffness and feeling of weakness in lower limbs and back. Epistaxis	Temperature 101° F. Tongue very furred. Throat dry. Optic fundus congested. Stiff neck, rigid, weakness in sitting up; pain in lumbar muscles; pain on leg movement Reflexes: Pupils react equal. Abdominals: R. lower absent. K.J.: R. only reinforced; L. weak. Plantar flexor weak	Abdominal reflexes disappeared. K.J. absent Temperature rose to 99° at night for three weeks	Clear. No raised pressure. Sugar nil. Lymphocytes increased. Some blood cells	Extensors of knees. Dorsiflexors of ankles	Adduction of shoulders? Diaphragmatic muscles. Other muscles of lower limbs
Case 3	Aged 15. Form IVc. Dormitory 2. In dining-room sits next Case 4	One week before fell on head; headache for three days. Three days before sleepless, restless, and shivering	Headache. Anorexia and nausea. Dizziness. Stiffness of neck and feeling of extreme weakness and lassitude	Temperature 102° F. Tongue furred. Stiff neck; pain in back on movement; weakness in sitting up; cramps in legs. Reflexes: Pupils react equal. Abdominals absent. K.J.: R. only when reinforced; L. present. Plantar flexor weak. Kernig's, mild plus	Weakness in arms. Flaccid paralysis of legs. Temperature normal after eight days but rose to 99° F. each night for three weeks	Clear. Pressure plus. Sugar normal. Cells 10 per c.mm. Chlorides 750 mg. per 100 c.cm. Protein 20 mg. per 100 c.cm.	Dorsiflexors of L. wrist. Lower limbs complete (18/7/38)	All upper limb muscles, especially adduction of shoulders and extension of elbows. Diaphragm. Erector spinae. Glutei
Case 4	Aged 15. Form IVa. Dormitory 2. In dining-room sits next Case 3	Two days before headache after being hit on the head with a cricket ball	Headache. Nausea. Dizziness. Pain in chest on coughing	Temperature 102° F. Tongue furred. Abdominal reflexes absent, except weak lower R.	Absence of deep reflexes of left arms. Absent right biceps jerk (left-handed boy). Left grip and extensors weaker than right. Abdominals back to normal. Temperature normal after five days, but rose intermittently for a fortnight	Lumbar puncture not done		Left dorsiflexors of wrist and left extensors of elbow
Suspect	Aged 15. Form Lower VIa. Dormitory 6	Headache three days before with lassitude. Five days before, stiff in arms and legs, after lying on damp grass	No headache or any other symptoms. Pain in chest	Temperature 99° F. Tongue furred. Central nervous system all normal	None. Temperature normal after six days			

Investigation into the Food Supply

The only common factors appeared, therefore, to be as stated above in paragraph 1—namely, the same feeding arrangements and contact with the same domestic staff. So far as can be ascertained there was no occasion when any outside person was in the house in a temporary capacity, save for one "old boy" visitor; and contact with the domestic staff did not vary significantly from day to day so as to cause infection to all four boys on one day only. Speech Day had been held on July 1, when there was a large influx of parents and relations; but here again there was no occasion when this one house only was in contact with one particular outsider or group. The tendency on that day is for the whole school to mix more thoroughly than usual.

The supply of food was investigated as a matter of routine only, as the present ideas of the transmission of the virus point to a nasal route through the cribriform plate and the olfactory bulbs. There are, however, eminent upholders of the gastro-intestinal route (Toomey, 1934; Kling, 1937). And Walshe (1927), in his description of the clinical course of the disease, stresses the fact that in the pre-paralytic stage there are signs of a general

infection with changes in the gastro-intestinal tract and its associated mesenteric glands before the central nervous system shows signs of being affected.

The following are details of the food supply: Milk is supplied to the whole school by the same dairy. Water is from a well in the house; this has been in continuous use for many years. Analysis on August 2, 1938, for what it is worth, showed satisfactory chemical and bacterial purity. Bread is supplied from the same source as the rest of the school. Butter comes from a large store in a neighbouring unaffected town. Fresh fruit and vegetables are obtained from a fruiterer in Braintree who supplies this house only. I happened to remember that the house has an annual treat of strawberries, and on inquiry I found that this occurred on June 27 and 29, and that the strawberries came from two farms, one of them at Halstead.

Here was a definite connexion with the other area which had been affected from July 1 onwards, and an event that had occurred sixteen and eighteen days before the four cases developed simultaneously in the only house where the strawberries were eaten. But as all the boys in the house had no doubt eaten them, could one reasonably speculate on anything about strawberries which would

pick out four boys only? Strawberries grow very near the earth, and are usually contaminated with it, as well as the common garden pests and their excreta. If it were conceivable for the virus to be present in such contamination, then accident would no doubt account for its being

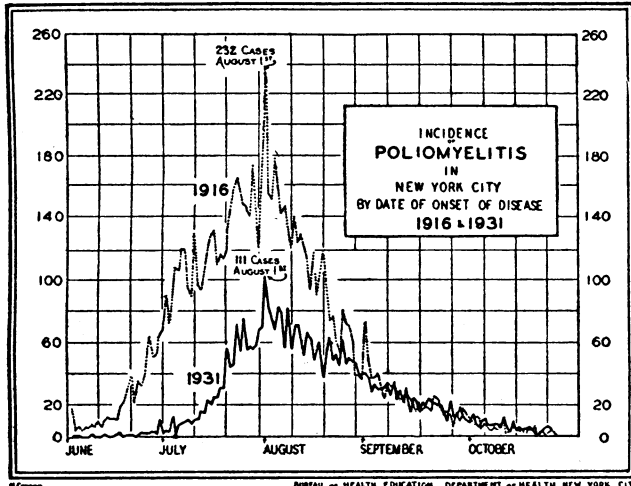


CHART II.—Curves showing the course of the epidemics of 1916 and 1931 in New York. The similarity of the two is remarkable: note that the highest count in each year occurred on the same day, August 1. (Reproduced by courtesy of the New York City Department of Health.)

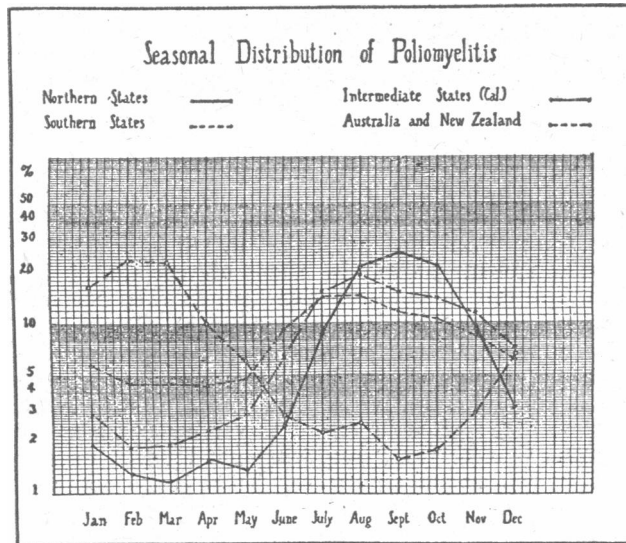


CHART III.—Seasonal distribution of infantile paralysis in different climates. Northern and Southern States, 1912-27; intermediate States, 1913-27; Australia and New Zealand, June, 1924, to August, 1927. (From Aycock, *Journal of Preventive Medicine*, 1929, 3, 245.)

ingested by a small number only of those eating the fruit. The simultaneous onset certainly resembles a food-poisoning outbreak rather than an epidemic spread by droplet infection.

Resemblance to Food Poisoning

This line of thought, though apparently far-fetched, could not be lightly dismissed when one considers how easily it would explain the majority of the well-known puzzling features in the epidemiology of the disease—namely:

1. The marked seasonal and climatic incidence (see accompanying graphs from the New York Health Bureau) agrees closely with the seasonal increase in the consumption of fresh garden produce and its climatic maximum production.

2. The epidemiological distribution of the disease does not resemble that of respiratory infections; it resembles that of food-poisoning and typhoid.

3. It is definitely *not* highly infectious. Until recently, cases were nursed from the start in general wards of general hospitals, and there have been no well-authenticated cases of infection to contacts. Certain of the cases in this recent outbreak occurred in crowded families, and were not reported until the illness had been in the paralytic stage for several days. During this time other children had been sleeping every night in the same bed as the paralysed child, and in no case was one of these contacts affected later.

4. The virus is said to be recoverable from the faeces of patients, proving that it is present in the alimentary canal.

5. In the classical case recorded by Draper a carrier was definitely postulated from the evidence as being a Greek fruiterer. All the cases were in contact with him as business associates, relations, or customers, and there is nothing in the evidence to point to the infection being carried by the man himself other than by the fruit he supplied.

This possible route of infection has already been discussed by Kling in Sweden on rather different evidence. The present account is given with the object of suggesting to virus workers a line of investigation that might be worth pursuing. It might then be thought worth while to experiment, for example, on passing the virus through some of the many possible contaminants of fresh garden produce—namely, earthworms, slugs, snails, insects, frogs, birds, and domestic animals and their excreta—or at least to determine the period of survival of the virus in fruit and vegetables.

Should anything of value result it would of course point the way to the prevention of the disease, as well as cause a radical alteration in the present attitude towards isolation and quarantine.

Summary

The history of an outbreak of anterior poliomyelitis in a boys' boarding school is recorded, with details of cases.

An account is given of an investigation into the cause of the epidemic.

The simultaneous onset resembled a food-poisoning outbreak, and is discussed from this aspect.

A line of investigation is suggested to virus workers.

Charts II and III are reproduced from Dr. George Draper's *Infantile Paralysis*, published by the D. Appleton-Century Co.

BIBLIOGRAPHY

Aycock, W. Lloyd (1929). *J. prevent. Med.*, 3, 245.
 Burrows, M. T. (1931). *Arch. intern. Med.*, 48, 33.
 Draper, G. (1935). *Infantile Paralysis*, D. Appleton-Century Co., N.Y. and London.
 Fairbrother, R. W., and Hurst, E. W. (1930). *J. Path. Bact.*, 33, 17.
 Flexner, S., and Lewis, P. A. (1909). *J. Amer. med. Ass.*, 53, 1639.
 Jungeblut, Claus W., and Spring, W. J. (1930). *Proc. Soc. exp. Biol.*, N.Y., 27, 1076.
 Kling, C. (1937). *Bull. Off. int. Hyg. publ.*, 29, 2137.
 Landon, J. F. (1938). *N.Y. St. J. Med.*, 38, 1.
 MacNalty, A. S. (1936). *British Medical Journal*, 2, 57.
 Paul, John R., Salinger, R., and Trask, J. D. (1933). *Amer. J. Hyg.*, 17, 587.
 Sabin, A. B., and Olitsky, P. K. (1937) *J. Amer. med. Ass.*, 108, 21.
 Toomey, John A. (1934). *Proc. Soc. exp. Biol.*, N.Y., 31, 680.
 Walshe, F. M. R. (1927). *Lancet*, 1, 326.