to press its just claim for recognition on the curriculum of the undergraduate school. As a matter of fact, a considerable number of these schools do avail themselves at present of the services of the gastro-intestinal specialists in providing instruction in this field. More than 50 per cent of the answers to the questionnaire made frank acknowledgment of this fact. I agree fully, however, with the prevailing sentiment of the authorities on medical education that a certain concentration of authority in the major clinical branches, three or four at the most, is necessary in order to conform with the real intent of undergraduate study; namely, to turn out general practitioners of medicine. Each special subject, though treated as a distinct subdivision, should be brought under the centralized control of the departmental chief.

In the ideal type of curriculum set up by the Association of American Medical Colleges, general medicine has been made to include the following subdivisions: pediatrics, neurology, psychiatry, dermatology and syphilis. Gastro-enterology is neither mentioned nor apparently considered in this classification as an essential subdivision of medicine. Nonetheless, the fact remains that this branch is fairly entitled to recognition in the plan of undergraduate teaching, and it is equally undeniable that instruction in the subject is best given by those possessing special training and experience in this particular field of work. The omission, therefore, of gastro-enterology from the list is not, I believe, a mere oversight and should call for immediate correction.

After many years of service and personal observation in the ranks of the undergraduate school, I am of the opinion that the interest of the student in respect to the teaching of gastro-intestinal and allied subjects would be best conserved by a plan such as follows: For the first and second, or preclinical years, correlation in the study of the basic sciences should be carried out in connection with the presentation of simple clinical data relating to disturbances of function of the digestive organs. A general plan of this character has been successfully employed and its merits highly commended by Pepper, Joseph and Emerson,[1] at a recent meeting of the Association of American Medical Colleges. Throughout the entire third or junior year, one hour a week should be allotted for the presentation of the various principles and methods underlying the modern concepts of gastro-enterology. This course should include a series of discussions and demonstrations on the technic of passing the stomach and duodenal tubes, duodenal siphonage, nonsurgical biliary drainage, and proctoscopy, as likewise the clinical interpretations to be placed on gastric and duodenal functional tests, feces and other laboratory examinations, and roentgenology of the gastro-intestinal tract. The way would thus be prepared for a close up clinical contact with the digestive patient in the fourth year. Here, the class should be taught, in small groups only, the ways and means of correlating all the clinical data obtained at the bedside and in the laboratory, along with the proper evaluation of the material thus obtained. The so-called overcrowded curriculum of the third and fourth year could be made to provide a limited, though sufficient time, I believe, for the plan as outlined above, even at the expense, if necessary, of slight encroachment on other fields.

1. Pepper, Joseph and Emerson: Proc. Thirty-Fourth Annual Meeting, Association of American Medical Colleges, March, 1924.

THE EPIDEMIOLOGY OF POLIOMYELITIS

WITH REFERENCE TO ITS MODE OF SPREAD*

W. LLOYD AYCOCK, M.D.
BOSTON

The most widely accepted theory concerning the mode of spread of poliomyelitis is that of direct contact through the upper respiratory passages. This theory, brought out by Wickman, has received experimental support in the detection of the virus in the upper respiratory passages in active cases, abortive cases and healthy contacts, and in the infection of animals through the nasal mucosa. However, epidemiologic evidence of direct contact is scant. The proportion of cases ascribed to direct contact, made up largely of multiple cases in families, has been stated at around 5 per cent. It has been observed that the onsets of multiple cases in families as a rule so nearly coincide that they probably represent in the majority of instances simultaneous infection.[1] When allowance is made for this, the proportion of direct contact cases is reduced to an extremely small figure.

The apparent discrepancy between the theory of spread through direct person to person contact and observation in the field has been met by the assumption of a comparatively large proportion of mild forms of the disease which escape recognition, and of healthy carriers, which together are largely responsible for the spread of the virus. Grounds for this assumption are found in the well known occurrence of paralyses so slight that they often pass unnoticed, and of the abortive type of the disease, as well as by the occasional detection of the virus in the nasopharyngeal secretions of healthy persons.

There are certain facts which seem to indicate that the distribution of the virus is indeed much more widespread than is indicated by recognizable cases; namely, age distribution and serum neutralization of the virus. The age distribution of poliomyelitis corresponds closely to that of the common contagious diseases, measles, scarlet fever and diphtheria. That this is probably a function of exposure and immunization is indicated by the difference between urban and rural age distribution. In concentrated populations, the peak of incidence is at from 2 to 3 years of age, after which the decline is so marked that only a small proportion of cases occur after 10 years of age. On the other hand, the peak in rural sections occurs somewhat later, and is not as pronounced as in cities. However, the decline after the peak is more gradual, so much so that after 5 or 6 years of age the incidence is relatively greater in rural sections.

This difference in age distribution, notwithstanding the fact that the incidence of poliomyelitis is as a rule greater in small communities and in rural sections than in large cities, suggests that with concentration of population and, as has been assumed, greater person to

* From the Department of Preventive Medicine and Hygiene, Medical School of Harvard University, and the Research Laboratory, Vermont Department of Public Health, Burlington.

* Read before the Section on Preventive and Industrial Medicine and Public Health at the Seventy-Seventh Annual Session of the American Medical Association, Dallas, Texas, April, 1926.

* This work was done with the support of the Harvard Infantile Paralysis Commission and the aid of a special fund privately donated to the Research Laboratory of the Vermont Department of Health.

1. Aycock, W. L., and Eaton, Paul: The Epidemiology of Infantile Paralysis: The Relation Between Multiple Cases in the Same Family, Am. J. Hyg. 5:724-732 (Nov.) 1925; The Epidemic of Poliomyelitis in New York City in 1916, Monograph, Department of Health, New York City, 1917, p. 121.

person contact, there is a widespread distribution of the virus, resulting not in greater incidence than in rural sections, as is true of the common contact diseases, but in a widespread immunization. In view of the fact that there is no theoretical reason why persons in rural life should be more prone to exhibit the paralytic form of the disease, it would seem perhaps more reasonable to believe that the immunization in concentrated populations may be due to subinfective doses rather than to a mild attack of the disease. Another point in favor of this immunizing process is the viricidal property of serum from a considerable proportion of normal individuals tested. While these tests have not been done on a large scale and the limits of serum neutralization are not yet clearly defined, the suggestion is that such individuals have undergone a process of immunization. This is evidenced by the absence of viricidal action of normal monkey serum, this property being acquired only after an attack of the experimental disease or after artificial immunization.

Under this conception of the distribution of the virus, the paralytic case has been considered as a relatively infrequent form of the disease, the lack of traceable relationship between recognized cases being accounted for by the supposition of intervening missed cases or healthy carriers.

The extent of the occurrence of the abortive form of poliomyelitis is not known, and the same is true of healthy carriers. The detection of the virus has been accomplished in such a small proportion of persons tested that, from an experimental point of view, the widespread distribution of abortive cases and healthy carriers can only be hypothesized. Epidemiologic evidence does not reveal the presence of any considerable number of cases of the abortive type of the infection. In a number of instances in which there were good opportunities for the detection of such cases, little evidence of their existence was found. For example, in a camp of approximately sixty boys, three paralytic cases occurred within a week. There was no increase in the sick-call, and an examination of the entire population of this camp did not reveal any evidence of sickness that could be suspected as having been abortive poliomyelitis. In a number of outbreaks in schools, we were not able to find any trace of mild illness that could be suspected. In several instances in which mild illness prevalent in the vicinity was regarded as poliomyelitis, investigation revealed other conditions.

Observations of the occurrence of poliomyelitis in Vermont over a number of years has borne out the idea that recognizable cases seldom occur in such relation to one another that they could be considered as resulting from direct contact; and yet, on the other hand, the time and space relationships between such cases suggest a more definite relationship than is implied in the abortive case healthy carrier theory of transmission.

In a study of multiple cases in families [1] it was found that, in the majority of instances in which more than one case occurred in the same family, the onsets were so close as to suggest simultaneous infection. More rarely, some patients were found attacked at a later date—after an interval of from ten to eighteen days—indicating that they were probably secondary infections.

REVIEW OF CASES

EXAMPLE 1.—Two cases suggesting simultaneous infection from thirteen to sixteen days before onset occurred in Margaret W., aged 16 (onset, Aug. 22, 1924), and Elizabeth W., aged 12 (onset, Aug. 25, 1924), sisters, who were not in contact with each other from August 9 to August 25, Margaret being away for a visit, where she developed poliomyelitis, August 22; she was brought home, August 25, the day Elizabeth developed symptoms. There was no history of contact with other cases. Although poliomyelitis was prevalent in both places, it is believed that the chances of independent origin of the two cases are slight, in view of the low incidence of this disease.

The idea of simultaneous infection is further borne out by the intervals between so-called contact cases. In New York City, in 1916, it was found that additional cases occurring in the same house but not in the same family likewise occurred in the majority of instances within such a short time of each other as to be considered coincident infection.[2] In Massachusetts, in 1916, the majority of so-called contact cases (not in the same family) had practically simultaneous onsets.[3]

TABLE 1.—*Data in Simultaneous Onsets of Poliomyelitis from Common Source Infection*

Year	Place	Name	Age, Years	Sex*	Onset	Time Interval, Days	Comment
1921	Woodstock....	H. L.	4	♂	Oct. 23	4	Within 2 miles; no history of contact
	Woodstock....	B. G.	25	♂	Oct. 27		
1921	Colchester.....	D. W.	5	♂	Aug. 26	1	Adjacent houses, daily contact
		F. M.	35	♂	Aug. 27		
1922	Rutland.....	W. H.	..	♂	July 23	0	No history of contact; same city
		D. C.	..	♂	July 23		
1923	Huntington....	J. W.	9	♀	Aug. 8	2	Ten miles apart; no other case in vicinity; within 4 miles of preceding case, but no known connection
	Shelburne.....	C. G.	3	♂	Aug. 10		
	Shelburne.....	D. W.	14	♂	Oct. 2		
1924	Rock Island, P. Q.	Y. J.	..	♂	July 25	0	Cases 1 and 2, 20 miles apart; cases 2 and 3, 12 miles apart; no history of contact, but all families had done considerable motoring in same territory; no known connection with foregoing cases
	Brighton.....	D. G.	20 mo.	♂	July 25		
	W. Burke.....	H. P.	4	♂	July 25		
	W. Burke.....	.....	..	..	Oct. ?		
1925	Elmore.....	I. H.	13	♀	Aug. 17	0	First case, one family; 2 and 3 in adjacent family; all in daily contact
	Elmore.....	J. R.	17 mo.	♀	Aug. 17		
	Elmore.....	R. R.	4	♂	Aug. 17		

* In this column, ♂ indicates male; ♀, female.

The following are instances of simultaneous occurrence in contacts not in the same family:

EXAMPLE 2.—In Elmore, three cases, with the onset, Aug. 17, 1925, occurred, two in one family and one in the next farmhouse less than one-fourth mile away. The families were relatives and in daily contact. The nearest previous case, about 4 miles away, had occurred, August 3, fourteen days before. A visitor was at this home on the day of onset, and later visited the two families in which the three simultaneous cases occurred.

EXAMPLE 3.—D. W., a boy, aged 5 years, was taken ill, Aug. 26, 1921. F. S. M., a man, aged 35, in whom the onset occurred, Aug. 27, 1921, kept a store in the small village, and the boy, living two or three doors away, was a regular visitor at the store. No other case occurred in the neighborhood.

2. The Epidemic of Poliomyelitis in New York City in 1916, Monograph, Department of Health, New York City, 1917, p. 153.
3. Infantile Paralysis Epidemic in Massachusetts, 1916, Massachusetts State Department of Health, 1919.

EXAMPLE 4.—Mrs. G., aged 31, was taken ill, Aug. 15, 1923; Bernice C., aged 9, Sept. 1, 1923, and Dorothy C., aged 15, Sept. 1, 1923. Bernice C. lived about 3 miles from the village on an isolated farm. Dorothy C., living in a city 20 miles away, visited her for about a week, returning home either one or two weeks before the two girls fell sick. Unfortunately, neither family could be perfectly sure whether the visit between these two girls ended one week or two weeks before the onset of sickness. There was no direct or indirect contact with Mrs. G., who lived in the village about 3 miles from Bernice's home. The nearest case to this group was more than 20 miles away.

These examples illustrate common source infection in contacts, as well as the interval of approximately fourteen days between cases within a short distance but not in contact.

The accompanying tables represent instances of the occurrence of two or more cases within such a short distance of each other, and at the same time so far

TABLE 2.—*Data in Simultaneous Onsets of Poliomyelitis in Common Source Groups with Interval of from Ten to Eighteen Days Between Cases*

Year	Place	Name	Age, Years	Sex	Onset	Comment
1921	Grand Isle.....	F. B.	16	♂	July 14	All within radius of 3 miles; two sets of coincident cases separated by fourteen-day interval
	Grand Isle.....	H. B.	16	♀	July 18	
	Grand Isle.....	A. L.	49	♀	Aug. 1	
		M. B.	1	♀	Aug. 1	
		J. R.	14	♂	Aug. 6	
1923	Athens.....	R. H.	7	♂	Aug. 1	Two September 1 cases in contact; no contact between other cases in the group; 14 day interval between 1st and 2d cases; 17 days between 2d and 3d, and 11 days between 6th and 7th should be noted
	Wilmington....	L. G.	31	♀	Aug. 15	
	Wilmington....	B. C.	9	♀	Sept. 1	
	Wilmington....	D. C.	15	♀	Sept. 1	
	Bellows Falls..	S. A.	4	♂	Oct. 22	
	Putney.....	K. H.	18	♂	Nov. 22	
	Dummerston...	E. G.	12	♀	Dec. 3	
1923	Rupert.....	A. L.	8	♀	Sept. 11	First two cases, same family, third across street; daily contact; fourth case some distance away; no contact
	Rupert.....	F. L.	12	♂	Sept. 14	
	Rupert.....	G. M.	7	♂	Sept 17	
	Rupert.....	P. T.	8	♂	Sept. 25	
1924	Sharon.....	C. B.	4	♂	Sept. 11	Second and third cases same house; daily contact; no other contact in group
	Randolph.....	G. A.	21	♂	Sept. 17	
	Randolph.....	J. E.	Adult	♀	Sept. 29	
	Randolph.....	K. B.	21	♂	Oct. 1	
1925	Calais.....	E. B.	17	♂	Aug. 3	First case 8 miles distant from group of three, and was nearest previous case; indirect contact through relative who visited first patient day of onset, and 1 week later visited other three; at no time did she show symptoms, but at this time she also visited a patient with scarlet fever, which she contracted and carried to other members of her own family
	Elmore.....	I. H.	13	♀	Aug. 17	
	Elmore.....	J. R.	17 mo.	♀	Aug. 17	
	Elmore.....	R. R.	4	♂	Aug. 17	
1925	Stowe.....	C. L.	25	♂	Sept. 19	No contact direct or indirect; first case in village; second case within a few blocks; third, about a mile away
		C. P.	15	♀	Oct. 1	
		R. P.	6	♂	Oct. 4	

removed from other cases that it seems reasonably certain that they constitute small localized outbreaks. In these tables multiple cases in the same family have not been included, except when such a family happens to be in a group with other adjacent cases.

These groups of cases have been divided for convenience into three tables: (1) cases with simultaneous onset; (2) those including instances of simultaneous onset as well as intervals of approximately fourteen

TABLE 3.—*Data in Instances of Poliomyelitis with Interval of from Ten to Eighteen Days Between Cases*

Year	Place	Name	Age, Years	Sex	Onset	Time Interval, Days	Comment
1921	Jericho.....	G. W.	28	♂	Oct. 1		Within two mile radius; late in season; no contact; intervals should be noted
	Underhill.....	M. N.	9 mo.	♀	Oct. 18	17	
	Jericho.....	D. P.	7	♂	Nov. 4	16	
	Underhill.....	R. T.	10	♀	Nov. 18	14	
1921	Hinesburg.....	J. M.	32	♂	Oct. 11		Last three same family; about 5 miles from first patient; first and third patients in contact
	Starksboro....	S. R.	24	♂	Oct. 20	9	
	Starksboro....	G. R.	22	♀	Oct. 27	7	
	Starksboro....	G. R.	15	♀	Nov. 15	19	
1921	Bennington....	R. P.	14	♂	Sept. 3		Three miles apart; no contact
	Bennington....	A. J.	21	♂	Sept. 15	12	
1922	Waterbury.....	H. H.	..	..	Oct. 12		No contact, direct or indirect
	Waterbury.....	E. M.	8	♀	Oct. 22	10	
1923	Bennington....	G. S.	5	♂	July 29		No contact, direct or indirect
	Bennington....	J. M.	13	♂	Aug. 17	19	
1925	Granville.....	O. P.	16	♂	Oct. 17		On day of onset first patient passed house of second patient and held few minutes conversation with cousin of second patient; no other case within about 25 miles
	Granville.....	C. W.	11	♀	Oct. 30	13	
1925	Middlebury....	E. B.	12	♀	Aug. 24		No contact, direct or indirect
	Cornwall.....	H. N.	20	♂	Sept. 18	18	
1925	Shaftsbury....	A. F.	10	♂	Sept. 20		Cases about 12 miles apart, but along main highway; no direct or indirect contact
	Manchester....	F. D.	6	♂	Oct. 1	11	
	Danby.....	N. W.	..	♂	Oct. 15	14	
1925	St. Albans.....	Z. L.	27	♀	Oct. 12		At time of onset of first case, second patient was visiting within a block, but returned home—14 miles—ten days before onset; no direct or indirect connection
	Milton.....	H. B.	31	♂	Oct. 30	18	
1926	Bradford.....	L. T.	13	♀	Feb. 14		No direct contact, connection being as follows: *
	Bradford.....	H. S.	13	♀	Feb. 25	11	

* Father of first patient killed a beef about day of onset of this case, of which he sold a quarter to father of second patient, who called for it and delivered some to his older children, who lived in a village 3 miles away, in one of whom a second case occurred—a child, aged 6, in family of second patient lived at home near that of first patient and attended same school.

days; (3) instances in which an interval of from ten to eighteen days elapsed between cases.[4] In this respect, the time intervals between cases in these small localized outbreaks are similar to those of family outbreaks previously referred to.

In table 1 are given five instances, in addition to that cited in example 2, in which neighboring cases have occurred practically simultaneously.

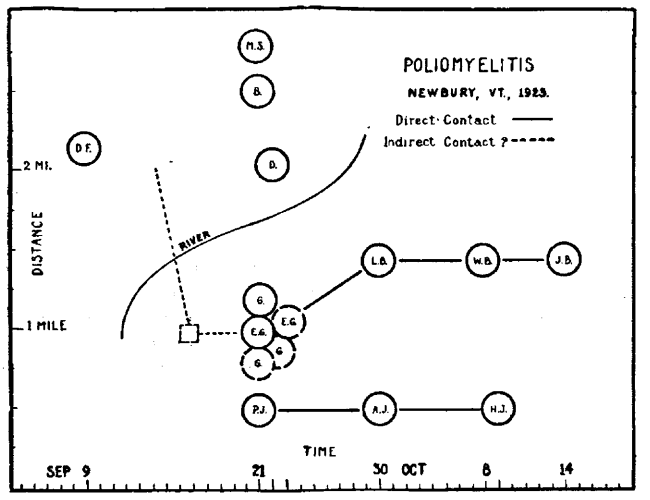
Table 1 gives examples involving, it is believed, common source infection, while table 2 shows cases in

4. This variation of eight days corresponds to the variation in the incubation period noted in experimental disease in monkeys (footnote 1, first reference).

which there were common source groups, as well as an interval ranging from ten to eighteen days.

Table 3 shows instances in which there was an interval of from ten to eighteen days between cases.

As will be seen in table 3, in none of the cases is there a history of direct contact. It would seem, then, that these cases, occurring as they do within very short distances and removed from other cases and within an apparently definite time, constitute primary and secondary cases.



Time and distance between cases in a localized outbreak: solid line circles, paralytic cases; broken line circles, nonparalytic cases; the "G" cases are all in one family.

Table 4 and the accompanying chart represent an isolated outbreak comprising fifteen cases, occurring within a radius of about 3 miles. Nine of these cases with no history of contact occurred practically simultaneously, twelve days after a previous case in the same vicinity. This suggests that the earlier case was the source of the outbreak, although we were unable to establish any relationship. From September 30 to

TABLE 4.—*Data in Isolated Outbreak of Poliomyelitis*

Year	Place	Name	Age	Sex	Onset
1923	Piermont (N. H.)	D. F.	..	♂	September 9
	Piermont (N. H.)	M. S.	50	♀	September 21
	Haverhill (N. H.)	B.	11	♂	September 21
	Newberry	G.	5	♂	September 21
	Newberry	E. G.	8	♂	September 21
	Newberry	G.	15	♂	September 21
	Bradford	P. J.	4½	♂	September 21
	Piermont (N. H.)	D.	5	♂	September 22
	Newberry	E. G.	14	♂	September 23
	Newberry	G.	12	♂	September 23
	Newberry	L. B.	12	♂	September 30
	Bradford	A. J.	27	♂	September 30
	Newberry	W. B.	13	♂	October 8
	Bradford	H. J.	27	♀	October 9
	Newberry	J. B.	10	♂	October 14

October 14, five additional cases occurred, three of these in the B family who lived next door to the G family and were in daily contact with them. The two latter cases occurred in families in which there were previous cases.

Tables 1-4 comprise approximately one third of all the cases of poliomyelitis that have occurred in the state of Vermont in the period represented, from 1921 to 1926. These cases occurred in small localized groups which, according to the space and time intervals, for the most part fall into classes 1 and 3 in table 5, which is intended to show different means of transmission as indicated by time and space relationships between cases.

This study, it is believed, suggests that paralytic poliomyelitis is not infrequently transmitted from a given person to other persons within a definite range,

TABLE 5.—*Time and Space Between Cases in Relation to Transmission*

	Space	Time	Mode of Transmission
1.	Immediate associates or within limited distance	Simultaneous onset	Common source
2.	Immediate associates	Specific interval	Direct contact
3.	Within limited distance	Specific interval	Transmission through indirect means
4.	Random distance	Random time	Transmission through missed cases and healthy carriers
	Sporadic cases		

but that transmission in such instances is not usually through direct contact between the individuals, nor through the intervention of missed cases or healthy carriers, but through some indirect means. This is illustrated by a recent outbreak, the epidemiologic evidence of which pointed to milk as the means of transmission.[5]

ABSTRACT OF DISCUSSION

DR. J. P. LEAKE, Washington, D. C.: Dr. Aycock's plan seems of great promise in its probability of advancing our knowledge of the spread of poliomyelitis, particularly in cases such as these, which can be followed much better than in a crowded community. I wish to call attention to the following of a somewhat similar plan in the Elkins, W. V., epidemic of the winter 1916-1917, the report on which is buried in the literature and somewhat forgotten. Dr. Smith, Dr. Bolten and I were able to prove definitely that insect transmission cannot be a necessary factor in the spread of infantile paralysis. We also showed, somewhat in line with some of Dr. Aycock's work, that in cases which might be called abortive, the symptoms tend to be referred to the digestive tract. So much for real evidence. There are other suggestions which the following, out of a series of such more or less isolated epidemics, does give one. Dr. Aycock has mentioned the failure to find abortive cases in many instances. I think any one who has studied intensively many epidemics has found that to be true; certainly, in some epidemics abortive cases are very difficult to discover and one cannot by means of such cases account for the spread of the disease. We must either assume healthy carriers or some other intermediary. The suggestion Dr. Aycock has given here and in other papers about the possibility of invasion by way of the digestive tract is worth bearing in mind. I was also interested in his last diagram of cases in which the father from the family across the river did the visiting and was presumably the healthy carrier. Repeatedly there have been suggestions, in studies that I have had occasion to make, of this very thing, that the adult male member of the family is likely to be more dangerous than others.

DR. W. L. HOLT, Little Rock, Ark.: I investigated an epidemic of poliomyelitis in a suburb of Newark, N. J., in the summer of 1916, when there was a big epidemic in New York state. It started in Brooklyn. We had twenty cases in our town during the summer. I investigated the best I could and was much surprised that I could trace hardly any cases to personal contact with others, there rarely being successive cases. It was certainly a place in which the population was very evenly distributed over the area. I do not think that

5. Knapp, A. C.; Godfrey, E. S., and Aycock, W. L.: An Outbreak of Poliomyelitis Apparently Milk Borne, J. A. M. A., to be published.

there was more than one case in a family, and in the very small slum district there was one fatal case with many children exposed and no secondary case resulting. Also, the first case was on the side of town toward Newark. There were a great many cases in Newark before we had our first case. The father of this first patient worked in Newark. The child had not been in Newark, nor had any one else in the family, except the father. Of course, we knew very much less about poliomyelitis in 1916 than we know now, but it is the general belief of health officers now that this epidemic was spread a great deal by the healthy adults who became healthy carriers.

DR. I. D. RAWLINGS, Springfield, Ill.: Any one who has had much experience with poliomyelitis is struck by the infrequency, relatively, of the secondary cases among direct contacts. In 1916, in Chicago, we became very much excited over the horrible situation in New York, with so many persons dying of the disease, and we took very stringent precautions through cooperation of the public health service and the local authorities in New York. We were advised of every person who left New York for Chicago who might possibly have been exposed. We immediately isolated these people and kept them under observation. When such persons went beyond Chicago, we notified the health officers of their home towns. Cases occurring in our vicinity, with no direct contact traced from New York, were treated in the same way. Every patient with poliomyelitis was hospitalized immediately and all contacts were put under quarantine for sixteen days. We had about 285 cases of poliomyelitis in Chicago in 1916, and 525 in 1917, and there was an average of three direct contacts to each of these cases. Every patient was taken out of the home and hospitalized, but the contacts under 16 were kept in the home for sixteen days, so there were approximately 1,500 direct contacts, and yet but one possible case occurred among them. Also among the large number of people that came from New York and other infected areas not a single case occurred. One is constantly struck with the fact that there are relatively few contact cases. We thought we had three secondary cases in a family in which there had been a case of poliomyelitis. They were reported as positive cases and were taken to the hospital. The final diagnosis in all four cases was trichinosis. I wonder whether in these rural areas referred to by Dr. Aycock the diagnoses were confirmed. We find in Illinois that quite a number of cases reported as poliomyelitis are not poliomyelitis when carefully investigated. The milk-borne epidemic described is very interesting. The Chicago commissioner of health found that in New York very few cases, if any, occurred among children who were using pasteurized milk distributed through the Nathan Straus infant welfare clinics. That gave him the idea that possibly milk had something to do with the epidemic; so an executive order was issued that all milk distributed in the city of Chicago must be pasteurized, and up to 1922 a large percentage of the milk supply of Chicago was pasteurized.

DR. A. C. NICKEL, Rochester, Minn.: I should like to emphasize the fact brought out by Dr. Aycock concerning the occurrence of sporadic cases of poliomyelitis. Last summer, Dr. E. C. Rosenow and I saw about fifty-five cases of poliomyelitis within a radius of 75 miles of Rochester, and frequently we would see a case in a very secluded spot where contact infection was quite unlikely. In such cases Dr. Rosenow was able to get the same positive precipitin reaction from the nasopharyngeal swab as from practically every case in the epidemic zone. By this means Dr. Rosenow feels that he can diagnose the presence of the poliomyelitic streptococcus in the throat, and its presence in the throats of sporadic cases seems to be against the contact theory of infection. For example, in Rochester, during the epidemic, a family came home from a two weeks' vacation. We obtained a nasopharyngeal swab from the entire family the day they returned, and all were negative. The children were not allowed to leave the house, and although the father went out several times he did not come in contact with any families having the disease. However, in less than a week the entire family was positive to the precipitin test, and two weeks after the epidemic their precipitin test was again negative. Such facts seem to suggest possibilities concerning the diagnosis and epidemiology of poliomyelitis.

DR. W. F. DRAPER, Washington, D. C.: I should like to ask Dr. Aycock to outline what measures, if any, he would recommend health officials to take for the control of the spread of the disease and the protection of other people.

DR. W. L. AYCOCK, Boston: Dr. Leake mentioned healthy carriers. In typhoid, a carrier sooner or later comes to be suspected by reason of his being associated with repeated cases. My experience with poliomyelitis has been that frequently a healthy person has been associated with two cases in the same localized outbreak, but we never find the "healthy carrier" associated with repeated cases. He must be regarded more as a transmitter of the infection and not as a chronic carrier, as in typhoid. I think it must be borne in mind that when poliomyelitis occurs in small foci, it is not very difficult to find among the local population the "healthy carrier" pictured. Dr. Holt mentioned diagnosis. Practically every case of poliomyelitis that has occurred in Vermont since 1914 has been verified by the state department of health. We make it a practice to see every suspected case as soon as it is reported. Most of the cases show some paralysis; others, as a rule, are cases in which spinal puncture was done and cell counts were made. Milk was mentioned in connection with the Cortland, N. Y., outbreak. I hope I have not given the impression that it is a major factor in the spread of the disease. Milk can hardly be responsible for any very considerable proportion of cases of poliomyelitis. It may be regarded as one of the several modes of spread. In regard to the precipitin test, some simple test which would give some indication as to presence of the virus of poliomyelitis would further our knowledge of the epidemiology of the disease very much. As to measures for the prevention of poliomyelitis, I think of it now as a disease that may be spread in a number of ways. I prefer to take, perhaps first of all, something like typhoid precautions. Dr. Leake's emphasis on the gastro-intestinal route mode of spread is well worth considering. We should institute precautions against contact spread, upper respiratory secretion spread and the gastro-intestinal, not forgetting such things as house flies and raw food that may be contaminated. As for insect hosts, if we find a few more milk-borne epidemics it will go far to rule out that possibility. It would, indeed, be unusual if poliomyelitis could spread through milk and through insect hosts.

A Sterilizable Dialyzing Membrane.—Tests by medical authorities show that the use, as a sterilizable dialyzing membrane, of a new synthetic cellulose product (used principally for wrapping) will probably make practical a whole field of bacteriology as yet hardly explored. Donna E. Kerr and Dr. H. W. Hill, director of the Vancouver General Hospital Laboratories, made these tests, which are described in part in the *Vancouver Medical Association Bulletin* for February: "In searching for a sterilizable dialyzing membrane, a suggestion came from the Christmas boxes of candy wrapped in glistening sheets of 'paper.' The resemblance of this material to thin sheets of collodion induced me to try its dialyzing powers, which, for silver nitrate, sodium chloride and glucose, proved perfect. The next step was to test its resistance to sterilization. Fifteen pounds of steam for twenty minutes in an ordinary laboratory autoclave left it unchanged in appearance, feel, etc. Its dialyzing powers were still unaffected. The various uses of such a membrane in physics, chemistry, and especially in bacteriology and biology, both scientific and applied, are obvious and need not be enlarged on here. With any household cement, sheets of it may be made into tubes, flat, square or round bags, etc. Diaphragms of it may be cemented across bowls, beakers or glass tubes. So far as we have been able to discover, this material, known as cellophane, has not been previously advocated as a sterilizable dialyzing membrane."