

**HISTORICAL AND GEOGRAPHICAL ASPECTS OF THE
EPIDEMIOLOGY OF POLIOMYELITIS***

Some of us who have been concerned with the epidemiology of poliomyelitis for a decade or more have finally come to realize that the behavior of this disease differs in different places and has differed in different times. Prior to this realization vain attempts had been made to make this disease conform in its behavior to a universal pattern, which could be presented as simple truths in a concise and digestible manner in textbooks of medicine and pediatrics. Today it is borne in upon us that as in many other situations, each truth can be related only to the circumstances—or the setting in which it has been observed. Thus the behavior of poliomyelitis under different environmental or sociological conditions varies from that of an “endemic disease” in infants as in Cairo, Egypt, to an “epidemic disease” of school children or young adults such as one might find in a North American city.† Similarly the *infantile paralysis* of our grandparents’ time is somewhat different from our polio of today, in the creation of which even the impact of the newspapers has been felt. In fact, ideas about poliomyelitis have had to be constantly revised and probably will continue to undergo revision. This should explain, perhaps, the title chosen for this lecture: *Historical and Geographical Aspects of the Epidemiology of Poliomyelitis*, and in this choice of a title I have shown no great originality, for I have merely taken a page from a well-known book by August Hirsch: “*Handbuch der historisch-geographischen Pathologie*,” first published almost a hundred years ago.¹¹

Historical. Acute paralysis in infants as a disease entity goes back to biblical times, perhaps earlier, but it does not seem to have attracted the attention of physicians prior to the late eighteenth century. Nevertheless, by the early nineteenth century, infantile paralysis was widespread geographically, for the earliest clinical descriptions of it came from several diverse areas: England, 1795; Italy, 1813; India, 1823; and U.S.A., 1830.²⁰

* The Fifth Annual Don W. Gudakunst Memorial Lecture presented on May 25, 1954 at the School of Public Health, University of Michigan, Ann Arbor, Michigan.

† The terms: *endemic* disease and *epidemic* disease here have been used in an arbitrary sense; endemic meaning frequent sporadic cases without epidemics, and epidemic meaning both sporadic cases and periodic epidemics.

Received for publication August 3, 1954.

The disease was regarded at that time as ubiquitous and due either to "teething," "foul bowels," or to a "fever." There was no mention of contagion, or of epidemics, nor was the condition regarded as a medical problem of any magnitude. As to how common or how prevalent endemic poliomyelitis was during the greater part of the nineteenth century, this is a matter for speculation; but Heine,⁹ an orthopedic surgeon in Germany, was able to collect a series of 30 late paralytic cases and publish a monograph in 1840 describing them. And coincidentally in this country, "orthopaedic surgeons" of the 1830's made occasional mention, without comment as to rarity, of cases of club foot due to paralysis, acquired in infancy as a result of teething or fever.¹⁰ Apparently then, the general situation in our grandparents' or great-grandparents' time was similar to that which can be found today in certain countries where substandard sanitary conditions exist and where this disease is endemic, limited to infants, where epidemics in local inhabitants are unknown, and where it is not regarded as a problem of major importance.

The first indication of anything which might be called an epidemic appears in a report by Sir Charles Bell, the anatomist and experimenter² in 1836. He stated that a "group of cases" of paralysis in children had occurred in an isolated community, namely, on the Island of St. Helena. Dr. Bell believed that the situation "deserved looking into." In the same year, Badham in England reported four cases of acute paralysis which had occurred more or less simultaneously in the small community of Worksop.¹ He also regarded this episode as ominous, although he hesitated to contend that any real element of contagion existed in this situation, perhaps because contemporary authorities were not familiar or sympathetic with any such idea. It was the reading of Badham's paper that prompted Heine, in Germany, to assemble his series of some 30 late paralytic cases. Here he firmly states his belief that the trouble is in the spinal cord, but he did not mention the epidemic character of the disease. A few years later there appeared Colmer's report, which has often been quoted, though erroneously interpreted as being the first description of contagion in poliomyelitis. This item which was printed in the *American Journal of Medical Science* in 1843⁸ is quoted herewith:

Medical Notes. By George Colmer. *Paralysis in Teething Children.* Whilst on a visit to the parish of West Feliciana, La., in the fall of 1841, my attention was called to a child about a year old, then slowly recovering from an attack of hemiplegia. The parents (who were people of intelligence and unquestionable veracity), told me that eight or ten other cases of either hemiplegia or paraplegia, had occurred during the preceding three or four months within a few miles of their residence, all of which had either completely recovered, or were decidedly improving. The little sufferers were

invariably under two years of age, and the cause seemed to be the same in all—namely, *teething*.

It seems clear that Dr. Colmer did not regard this situation as an epidemic of an infectious disease.

But in spite of these limited and scattered outbreaks in the mid-nineteenth century, poliomyelitis was not destined to become quickly recognized as a disease entity with infectious or epidemic potentialities. Not until 1868 were groups of cases reported in Norway⁴; and it was still later when the pediatrician, Medin,¹⁷ and others described outbreaks in Sweden, that the epidemic character of poliomyelitis began to be taken seriously. Furthermore, recognition of this new trend, if so it can be called, was for some 25 years limited largely to northern Europe, for textbooks articles on infantile paralysis which appeared in this country during the 1870's and 1880's fail to make mention of it, other than to state that various infectious diseases might be the immediate causal antecedent of paralysis in childhood.¹⁸ However, by 1895 ideas about the disease had entered a new phase; its epidemic potentialities, were recognized, and it had temporarily acquired a new name: Heine-Medin Disease.*

It was after the turn of the century that Medin's pupil, Wickman, in Sweden,³⁰ began his classic work. This challenged the emphasis upon paralysis, which had previously dominated all thought in this disease, and became the basis of more modern epidemiological concepts, with an appreciation of its infectious nature, its spread through human association, or possibly through the agency of milk, and the importance of mild, nonparalytic cases as carriers. Not only did Wickman recognize the mild cases, but he recognized their epidemiological significance—for by including them in his epidemiological studies he was better able to trace the comings and goings of the disease in small villages and communities than had his predecessors. Indeed, he had some inkling that nonparalytic poliomyelitis was actually the "typical form" of the disease with paralysis as a "sort of complication," albeit a spectacular one. It was *clinical epidemiology* at its best, in spite of the fact that he was working in the dark as far as knowledge of the etiological agent was concerned, for all his field work was done prior to the discovery of the virus of poliomyelitis by Landsteiner and Popper in 1908.³⁵

If one were to continue to search for names (appropriate in 1910) to add to the title of this disease, one could compose the following: Heine-Medin-Wickman-Landsteiner Disease.

* In the meantime other names had crept in, some of which today sound singularly cumbersome and inept. Among these were: acute fatty atrophic paralysis; essential paralysis of childhood; tephromyelitis, etc.

But let us not forget that a decade before Wickman, Caverly, a physician in Vermont, first described nonparalytic poliomyelitis.⁸ The basis of his description was an epidemic in Otter Creek Valley near Rutland, Vermont. It reminds us that only when fairly large numbers of cases were seen together as in an epidemic that observations on the variegated clinical picture of this disease were at last made possible.

Since 1900, and with increasing rapidity, epidemics of poliomyelitis appeared in Europe, North America, and elsewhere. Areas and approximate dates where and when this transition began are: Scandinavia, 1860-1890; western Europe, 1880-1890; the northeastern section of the U.S.A. in the 1890's; and the southeastern section two decades later. From a comparative curiosity, infantile paralysis had become a periodic scourge. And once epidemics of poliomyelitis have begun to appear in a given region, the pattern has not been reversible. Furthermore, there has generally been a sharp or gradual progressive increase in prevalence, as measured over ten-year periods. In some countries the annual number of cases has risen over the past 20 years, as in the U.S.A., Canada, and France; in others, the number of cases has maintained itself at fairly high levels during this period with widely irregular swings.

Still more recently, i.e., within the past 20 years, has this epidemic evolution of the disease come to pass in tropical and semi-tropical areas such as Puerto Rico, Hawaii, Malta, El Salvador, and the Island of Mauritius. In keeping with the idea that poliomyelitis has not usually been considered to be a disease of the tropics, the thought usually was, when these tropical epidemics appeared, that a "new disease," or at least a new strain of virus, had suddenly been introduced into the community. This may well have been the case in some places; but, on the other hand, many tropical populations had not been previously aware of the endemic character of their own local poliomyelitis or of how common it was. It certain places of this type, such as the Philippine Islands, the native disease only came to the surface when groups of foreigners, such as U. S. soldiers, entered the country as "susceptible immigrants," and there these military populations acquired poliomyelitis at far higher rates than those reported locally in native civilians and at far higher rates than would have been expected in their homelands. This was noted in the Philippines as early as 1936 and subsequently,¹⁰ i.e., before, during, and after World War II.²⁸ Documented examples have also occurred in Cairo, Egypt²¹; in India¹⁰; and in French Morocco.²⁸

Another evolutionary change which followed the advent of epidemics has been a gradual shift in *age incidence*. During the "pre-epidemic era," of the

nineteenth century and before, poliomyelitis seems to have been almost entirely a disease of infants under five years of age. Indeed, one might term the *primitive form* of poliomyelitis as the true "infantile paralysis." This concentration of 90 per cent of the paralytic cases within the infantile age group is seldom seen in north Europe or Scandinavia today, or in the U.S.A., Canada, or Australia; for, whereas 40 or 50 years ago maximum attack rates were nearly always recorded in the 0-4 age group, this soon shifted to the 5-9 age group, or, as in Sweden at present, to even older age groups.¹⁹

It is obvious that the factors which wrought this change were multiple: The composition of the population as far as age groups are concerned, the curtailment in the size of the families, and perhaps others. But important among these factors has been a shift in the opportunities for exposure early in life. It had long been appreciated that the age incidence of poliomyelitis, like that of measles a generation ago, varied with the concentration of population; i.e., the more densely populated the community, the younger the age of those affected. It was not until about 1930 that it became evident that urban cases were becoming older and that a general or world-wide trend towards an *increase in the age* of poliomyelitis patients, was taking place. Burnet⁵ was a pioneer in placing emphasis on the epidemiological significance of this trend. Today in the northeastern part of the United States as many as 35 per cent of the patients may be 15 years of age or older (see Fig. 1). This is a phenomenon of *epidemic* as opposed to *endemic* poliomyelitis and, as adult cases are apt to be more severe, with this rising age incidence cases are more readily diagnosed and reported, and therefore attack rates increase accordingly. For these reasons alone, it is easy to see how the *epidemic* form of poliomyelitis has attracted much more attention, more alarm, and more study than has the endemic disease.

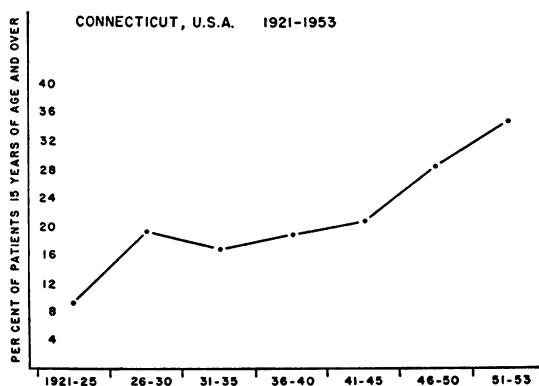


FIG. 1. The per cent of cases of poliomyelitis which occurred in Connecticut patients who were over the age of 15. Annual lists of such cases have been averaged for five-year periods, for the period 1921-1953, inclusive. A progressive rise on this percentage is apparent. (Data obtained through the kindness of Dr. Mila Rindge, Bureau of Preventable Disease, State Department of Health, Connecticut.)

For a number of years this shift from an endemic to an epidemic disease, from an infantile to a childhood disease was without explanation.¹³ But most of the remainder of this article will be concerned with a review of evidence as to what has been responsible for this evolution. It will also be concerned with newer measurements on which the evidence is based and which now have made it possible to estimate the *immunity status* against poliomyelitis existing within certain populations and communities.

Geographical. We have already mentioned that poliomyelitis like many another disease spread by human association (i.e., like measles and mumps) is world-wide in distribution, and there is no reason to believe that there is any part of the inhabited globe to which the disease will not penetrate. Nevertheless, the prevalence of reported cases of poliomyelitis varies enormously in different places, and it can be cautiously stated, as of 1954, that those countries which pride themselves on having advanced standards of sanitation and low infant mortality rates have the highest poliomyelitis rates. Furthermore, the epidemiological behavior, and the clinical behavior of poliomyelitis, vary in other ways in different places as they have in different times: from that of extreme endemicity with low rates and a disease confined to infants to that of recurrent epidemics with involvement of older age groups and high rates when computed over a ten-year period. As to the mechanisms of spread involved, granted that poliomyelitis is an infectious disease which is easily transmitted from person to person, one may well ask about current ideas as to the type of human association needed for transmittal. Although one cannot be dogmatic on this point, it is my belief that most cases of poliomyelitis, though not necessarily all, are acquired as a result of a susceptible person coming in contact with an individual in the infectious or infective stage, regardless as to whether he or she be suffering from an overt, (paralytic or nonparalytic) *abortive case*, or an *inapparent* infection. There is some evidence to believe the severe paralytic cases are the most dangerous spreaders of infection but it is the inapparent form of infection which makes up the bulk of so-called "healthy carriers."

As to the nature of the infectious stage, it appears that early in the disease the virus appears simultaneously in the blood stream, the throat, and the intestinal tract. Subsequently it persists for varying periods of times in the intestinal tract alone. Recognizing these two possible portals of exit for the virus, it is easy to imagine that children can readily contaminate their playmates and their immediate environment. That contamination of the environment also plays some part seems likely, for reasons to be mentioned presently. In any event poliomyelitis is apparently one of those

diseases which, like measles, is ever ready to spread into a susceptible population; and it will do so whenever the virus has access (during the appropriate season) to such a susceptible population, unless the mechanism or spread is removed by artificial or special circumstances. Today, there are few communities no matter how small or remote, how sanitary, or how modern, which can hope to escape periodic contact with this virus forever, and, if the inter-epidemic period is long, the risk of a potentially severe epidemic increases annually, as a population composed of susceptible children, adolescents, and even young adults is built up. This susceptible population therefore represents a kind of vacuum of growing size into which the virus may surge when the opportunity offers, and the older the susceptibles are, the more severe is the epidemic likely to be. Few more graphic examples of this are at hand than that of the poliomyelitis epidemic among Eskimos in the Canadian Arctic in 1948-49,³⁷ and again in 1953.³⁴ Here in the fall of 1948 the disease was first introduced into settlements along the west coast of Hudson's Bay and spread rapidly northwards, reaching the remote settlement of Chesterfield Inlet in mid-winter, where the epidemic involved almost 20 per cent of the entire population with paralytic disease, attacking all age groups, the lowest clinical attack rate being in the infants!³⁷ The same situation was encountered in another village in 1953.³⁴ This fact reminds us that, as in measles, the age distribution of cases of poliomyelitis within a community or population can also be taken as an indication of the state of immunity of that population, which in the case of these Eskimos must have been exceedingly low.

Today, however, measurements besides those based on age specific case rates can be made which may enable the epidemiologist to evaluate the immune status of a given population in advance of an epidemic. This has been done in many areas by determining the per cent of those healthy individuals in the chosen population who possess antibodies capable of neutralizing one or more types of poliomyelitis virus. Such neutralizing antibodies persist after infection probably for life and indicate whether the individual who possesses them has been infected either in the recent or distant past. In other words, although the presence of antibodies and immunity is not one and the same thing, neutralizing antibodies represent the footprints of disease, whether they be clear or faint, which indicate that an infection has passed that way. And again, although the significance of various antibodies differs, a general pattern can be found which allows one to use this method to determine roughly the "immune status" of a given population not only to poliomyelitis but to a variety of different infections.³⁸

Originally such surveys on the serological epidemiology of poliomyelitis were carried out in various parts of the world with very crude and ex-

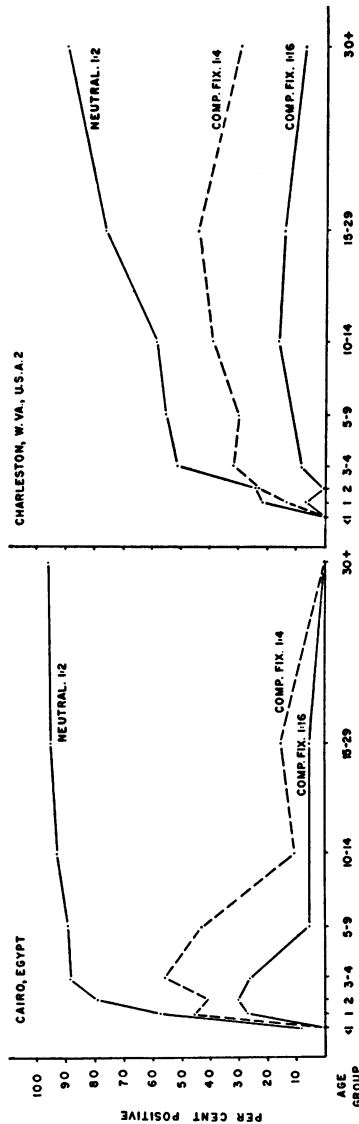


FIG. 2. Age distributional antibody patterns to Type 2 poliomyelitis virus in the populations of two cities (Cairo, Egypt, and Charleston, W. Va., U.S.A.) which differ widely as far as local environmental conditions are concerned. The neutralization tests (done with a serum dilution of 1:2) indicate a rapid rise in infancy of these antibodies in Cairo (on the left), as compared with Charleston (on the right). The complement fixation tests done with two serum dilutions 1:4 and 1:6 indicate a concentration of Type 2 poliomyelitis infections in the age group under 10 in Cairo, which is not apparent in Charleston. (Cairo data from Paul *et al.*²⁸; Charleston data obtained through the kindness of Drs. Mary Walton and J. L. Melnick.)

pensive tests in monkeys, later in mice, but today, thanks to discoveries by Enders and his co-workers, in tissue cultures. This special approach has allowed measurements on poliomyelitis antibodies to be made on a large

scale. The serological survey can now take its place along with tuberculin test surveys or Schick test surveys. In other words, it aims to differentiate the immunes from the susceptibles within a given population.

This survey method has been most useful in tracing the presence of infection in remote areas. Thus in two remote Eskimo communities, North Alaska,²⁸ and Baffin Island,⁷ it has been found, as one might have expected, that there was a dearth of neutralizing antibodies of all three types of poliomyelitis in over 95 per cent of the young adult population. In contrast, with-

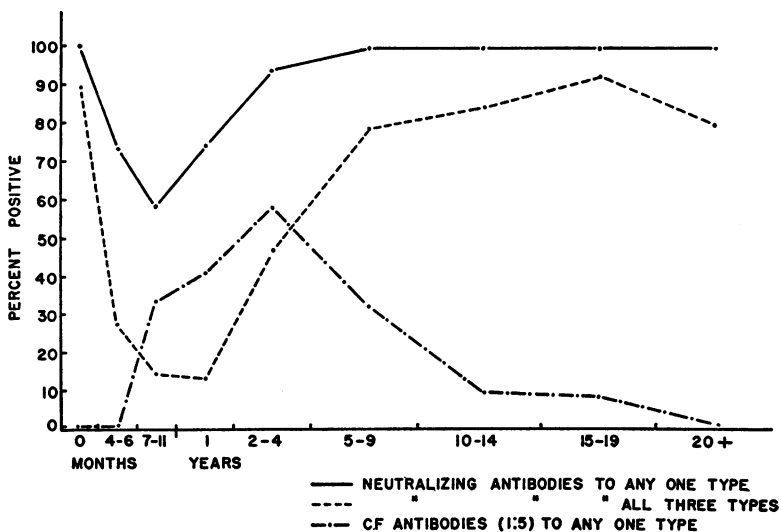


FIG. 3. Neutralizing and complement-fixing antibodies to all three types of poliomyelitis virus as determined in a group of the juveniles and adolescents from Northwest Africa. Heavy infection in early infancy is apparent with a good deal of such infection occurring within a very brief period after the loss of maternal neutralizing antibody. After the age of 10 years, the curve for complement-fixing antibodies reveals very little evidence of reinfection or new infections in this population. (Data from Paul and Horstmann.²²)

in certain crowded areas both urban and semi-urban, the situation may be quite the opposite, for surveys made in areas with substandard sanitation, many of them in the tropics, have shown that almost all healthy children over the age of four or five years possess Type 2 neutralizing antibodies, indicating that they had already been infected with this strain and gained immunity,^{28, 24} see Figures 2 and 3. Thus in Northwest Africa and in Cairo, Egypt, there is evidence of heavy exposure in early life to poliomyelitis virus. The infants there are very busy making antibodies during the earliest years of their life. Actually, by the time they have reached the age of five or six years they have reached immunological maturity as far as this

disease is concerned, thus eliminating the subsequent threat of adult poliomyelitis. This fact is also borne out by the extreme dearth of adult clinical cases in locally born residents of these areas.

Although the neutralizing antibody response recorded in Figure 2 is only that of one strain, i.e., Type 2, it appears that this antibody is a fairly good index of the immunity status of poliomyelitis in general.²⁴ The basis of this statement rests on the fact that in a number of widely separated areas, there is fairly good correlation, i.e., if the ages at which Type 2 *poliomyelitis neutralizing* antibodies have been acquired are compared with the ages at which *clinical poliomyelitis* (presumably due to any type) was acquired.

As to the speed with which these Type 2 poliomyelitis neutralizing antibodies are acquired, in contrast to the Cairo situation where it is about as fast as can be, one may choose a typical U.S.A. situation such as that in Charleston, West Virginia, where the curve in Figure 2 reflects a delayed rise in neutralizing antibodies which does not reach 90 per cent until the age of 30, indicating that adolescent and adult poliomyelitis is common, and immunological maturity has been postponed.

The above results record findings concerned with only one strain of the virus. With the introduction of tissue culture methods it is now possible to carry out neutralization studies with all three types. In general, the results of these triple surveys are the same as those done with Type 2 virus alone, indicating that in some populations infants are infected two, and sometimes three times in the early years of their life, so that antibodies to one, two, or three types are present in about 80 per cent of the population by the time they have reached five years (see Fig. 3).²⁵

It has also been possible recently to carry out *complement fixation* tests on sera from a given population.⁸ From analyses of the age specific rates at which these more transient antibodies are acquired or lost one can determine not only what the story has been with regard to poliomyelitis infections in the past but also those which have occurred recently, i.e., within three to five years. This is based on the assumption that complement fixing antibodies as measured in these studies do not remain elevated for longer than three to five years (see Figs. 2 and 3).

By including both neutralizing and complement fixation results in the juvenile population studies of Cairo, Northwest Africa and Charleston, West Virginia, one can compare the age incidence of recent versus old infection. Here we also have an opportunity to get some answers on the puzzling question as to the duration of immunity in poliomyelitis. Is it lasting and solid? Do type specific reinfections occur? Actually there appear to be at least two answers (see Fig. 2) to this, for one can find two different

types of complement fixation responses in different populations: one indicates (see left side Fig. 2) that in the Cairo population there is evidence that recent infections (whether new infections or re-infections) are all concentrated in childhood; whereas another in the city of Charleston, West Virginia, (see right side Fig. 2) indicates that these events are spread over a 30-year period, suggesting that immunity as acquired there was not so solid, and type specific reinfection may occur. This phenomenon calls for far more investigation, for it is clear that the work is in its infancy and that older qualitative measurements of age specific antibodies now deserve to be replaced by quantitative measurements.

In summary, therefore, it appears that both the disease, poliomyelitis and epidemiological thinking about it has undergone revision in the past generation. From an *endemic* disease it has become *epidemic* in many places, and indeed has become a common periodic scourge. From a disease originally regarded as being limited to infants, it is no longer confined to infancy. From a disease originally considered as mildly contagious or infectious, it is now regarded as highly infectious. From a disease in which it has been claimed that environmental sanitation played no part in its spread it has become one in which it is now clear that this factor must play some part.

A number of methods which utilize greatly improved techniques for the determination of antibodies against poliomyelitis virus have clarified some of the previously existing epidemiological puzzles by giving us some comprehension of the relative immunity status of various populations. In its new perspective, poliomyelitis now appears as a disease, spread not only through *direct contact* between individuals, but one in which contamination of an infected individual's *immediate environment* cannot be ignored. This is brought out by its varied behavior in different urban areas where sanitary arrangements vary. In communities where infant mortality is high, sanitation is primitive, and living conditions are crowded and poor, facilities for the spread of such a virus are better than elsewhere. Consequently, within such primitive communities, infants have the opportunity to come in contact with all three types of poliomyelitis virus early in life. Few of them reach the age of three or four years without having been infected at least with one strain, and most of them with two, although clinically, and officially the infection to a very large degree is inapparent. In any event, immunity is acquired rapidly and silently in such areas as far as local official morbidity statistics are concerned and no large group of susceptibles is built up. Within such communities the disease smoulders, and they are not liable to have epidemics of poliomyelitis. In countries where sanitary arrangements are no longer primitive, the risk of contact with the virus at an early age

is diminished. A large percentage of the juvenile population reaches the age of six, eight, or ten years without having acquired any infection or any immunity, and consequently such populations are ripe for epidemics, which sooner or later seem to appear. It might seem questionable whether this latter situation is desirable. This characteristic difference in the behavior of poliomyelitis in different places is the strongest evidence that the spread of this disease is influenced by environmental factors, a feature which for years has been controversial.*

Other and crucial questions here are whether poliomyelitis is really a much milder disease in infancy than it is later in life and whether the infantile ratio of apparent to inapparent infection is of the order of 1 : 200, in contrast to a childhood ratio of nearer 1 : 100. This may be based on the fact that the recent presence of residual maternal antibody, or as better stated, "resistance inherited from the mother," may equip the infant quite well to sustain a modified attack or attacks of poliomyelitis, if he is fortunate enough to be heavily exposed in infancy.

If there are any morals or lessons to be learned, it would appear that if one is to be concerned with the prevention of poliomyelitis, then the whole situation in this country is one which cries out especially for some means of artificial immunization as a far more hopeful approach than further attempts to rid the environment of poliomyelitis virus.

REFERENCES

- 1 Badham, J.: Paralysis in childhood. Four remarkable cases of suddenly induced paralysis in the extremities. Lond. M. Gaz., 1835, 17, 215.
- 2 Bell, C.: *The nervous system of the human body, as explained in a series of papers read before the Royal Society of London*. 3d ed. London, 1836, p. 434.
- 3 Black, F. L. and Melnick, J. L.: Complement fixation tests in poliomyelitis. Yale J. Biol., 1954, 26, 385.
- 4 Bull, cited by Leegard, C.: Poliomyelitis with demonstration of microscopical preparations. Abstr. Neur. Zbl., 1890, 9, 760. (Norske lægeböde i Bergen, Aug. 1889); see also Bergenholtz. Cited by *Medin*.¹⁷
- 5 Burnet, F. M.: The epidemiology of poliomyelitis with special reference to the Victorian epidemic of 1937-38. Med. J. Australia, 1940, 2, 325.
- 6 Caverly, C. S.: Notes of an epidemic of acute anterior poliomyelitis. J. Am. M. Ass., 1896, 26, 1.
- 7 Clark, E. M. and Rhodes, A. J.: Poliomyelitis in Canadian Eskimos. Laboratory Studies, III. Lansing antibody levels in Baffin Island Eskimos. Canad. J. M. Sc., 1952, 30, 390.
- 8 Colmer, G.: Paralysis in teething children. Am. J. M. Sc., 1843, n.s.5, 248.
- 9 Heine, J.: *Beobachtungen über Lähmungszustände der unteren Extremitäten and deren Behandlung*. 1st Ed. Stuttgart, F. H. Köhler, 1840.
- 10 Hillman, C. C.: Poliomyelitis in the Philippine Islands. Mil. Surgeon, 1936, 79, 48.

* Space does not allow a discussion of the question as to whether the dominant effect of poor living conditions consists in crowding. By comparing different environmental conditions on another contact disease, namely, mumps, poor living conditions do not exert as much effect as in poliomyelitis.¹⁸

- 11 Hirsch, A.: *Handbuch der historisch-geographischen Pathologie*. Erlangen, Ferdinand Enke, 1862-64.
- 12 Hutchins, H.: Historical summary. Chap. 1 in *Poliomyelitis*. Baltimore, Williams & Wilkins Co., 1932, pp. 1-22.
- 13 Jacobi, P. Mary: Infantile spinal paralysis. In *A system of practical medicine by American authors*. Edited by Wm. Pepper. Vol. V, pp. 1113-1164, Philadelphia, Lea Bros. & Co., 886. See also Atrophic infantile paralysis. Article xi in *A practical treatise on the disease of children* by F. Meigs and Wm. Pepper 4th ed. Philadelphia, Linsay & Blakiston, 1870, pp. 568-586.
- 14 Johnsen, H. V. and Wood, W. J.: Outbreak of poliomyelitis at Maguse River. N.W.T. Canad. J. Pub. Health, 1954, 66, 16.
- 15 Landsteiner, K. and Popper, E.: Mikroskopische Präparate von einem menschlichen und zwei Affenrückenmarken. Wien. klin. Wschr., 1908, 21, 1830.
- 16 McAlpine, D.: Epidemiology of acute poliomyelitis in India Command. Lancet, Lond., 1945, 2, 130.
- 17 Medin, O.: Ueber eine Epidemie von spinaler Kinderlähmung. Verh. Internat. med. Kongr. (1890), 1891, 3, (Abt. 6), 37.
- 18 Melnick, J. L. and Ledinko, N.: Development of neutralizing antibodies against the 3 types of poliomyelitis virus during an epidemic period. The ratio of inapparent infection to clinical poliomyelitis. Am. J. Hyg., 1953, 58, 207.
- 19 Olin, G.: Epidemiologic pattern of poliomyelitis in Sweden from 1905 to 1950. In *Papers and discussions*, presented at the Second International Poliomyelitis Conference. Philadelphia, J. B. Lippincott Co., 1952, p. 367.
- 20 For a review of this period in the history of poliomyelitis, see Paul, J. R.: Observations on the history of poliomyelitis. Bull. Ayer Clin. Laborat., 1951, 4, 21.
- 21 Paul, J. R., Havens, W. P., and Van Rooyen, C. E.: Poliomyelitis in British and American troops in the Middle East. Brit. Med. J., 1944, 1, 841; see also Caughey, J. E. and Porteous, W. M.: An epidemic of poliomyelitis occurring among troops in the Middle East. Med. J. Australia, 1946, 1, 5.
- 22 Paul, J. R. and Horstmann, D. M.: A survey of poliomyelitis antibodies in French Morocco (to be published.)
- 23 Paul, J. R., Melnick, J. L., Barnett, V. H., and Goldblum, N.: Survey of neutralizing antibodies to poliomyelitis virus in Cairo, Egypt. Am. J. Hyg., 1952, 55, 402.
- 24 Paul, J. R., Melnick, J. L., and Riordan, J. T.: Comparative neutralizing antibody patterns to Lansing (Type 2) poliomyelitis virus in different populations. Am. J. Hyg., 1952, 56, 232.
- 25 Paul, J. R. and Riordan, J. T.: Observations on serological epidemiology. Antibodies to the Lansing strain of poliomyelitis virus in sera from Alaskan Eskimos. Am. J. Hyg., 1950, 52, 202; see also Paul, J. R., Riordan, J. T., and Melnick, J. L.: Antibodies to three different types of poliomyelitis virus in sera from North Alaskan Eskimos. Am. J. Hyg., 1951, 54, 275.
- 26 Paul, J. R., Riordan, J. T., and Kraft, L. M.: Serological epidemiology: Antibody patterns in North Alaskan Eskimos. J. Immun., Balt., 1951, 66, 695.
- 27 Peart, A. F. W.: An outbreak of poliomyelitis in Canadian Eskimos in winter-time. Canad. J. Pub. Health, 1949, 40, 405.
- 28 Sabin, A. B.: The epidemiology of poliomyelitis. Problems at home and among the Armed Forces abroad. J. Am. M. Ass., 1947, 134, 749; see also Hammon, W. M.: Personal communication, 1954.
- 29 Walter, A. G.: Observations of tenotomia and myotomia, for the cure of deformed members; anatomically, physiologically and therapeutically considered. With seventy-four cases. Select Med. Library and Eclectic J. Med., 1840, 4, 385-421. See also Detmold, W.: Report of several cases of successful operation for clubfoot by the division of the tendo achillis, with remarks. Am. J. M. Sc., 1838, 22, 105 and Chase, H.: Report cases of deformed feet, treated by mechanical means, with a description of the apparatus employed. Am. J. M. Sc., 1841, n.s.1, 88.
- 30 Wickman, I.: *Acute poliomyelitis (Heine-Medin's disease)*. Nervous and Mental Disease Monograph Series, No. 16. New York, Journal Nervous and Mental Disease Publishing Co., New York, 1913.