

1940

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THE EPIDEMIOLOGY OF POLIOMYELITIS

by Thomas W. Deakin

A Senior Thesis Presented

to the

College of Medicine

UNIVERSITY of NEBRASKA

Omaha 1940

INTRODUCTION

Ever since its recognition, the disease called infantile paralysis has raised a controversy, which, it must be admitted, is kept as moulder by the broad retrospective views on one hand directly opposed by the more concise scientific attitude on the other. For the former group it has existed from the earliest recorded days and perhaps before, while for the others it is practically a new disease. Regardless of which view is the most reasonable, its increasing incidence throughout the world in epidemic form is an admitted fact. It is for this reason that the following pages are devoted entirely to the epidemiology of poliomyelitis; not only because it is an epidemic disease, but because of the puzzling nature of its epidemiology.

.....

The earliest cases of poliomyelitis, of necessity, rely upon retrospective diagnoses made upon archeological material. Although there is not sufficient factual evidence in such studies to offer them here as definite data, they are not entirely

A skeleton, dating from about 3700 B.C., was found at Cairo by Flinders Petrie. He noted that one leg was shorter than the other and assumed that the thigh had been broken. J.K. Mitchell, upon studying this mummy later in the archeological museum of the University of Pennsylvania, and making careful measurements of the bones, found no evidence of fracture. Instead, he reports,

"...the left leg is considerably shorter than the right, and at first sight the femur of the left side seems heavier and thicker than the right one. However, measurement makes it evident that the left femur's greater circumference is only due to its shortening and to the strongly marked ridges at the site of the muscular attachments, which suggest that the muscles may have even been hypertrophied on that side. With this exception the left femur is imperfectly developed in all directions. There is no sign of fracture or injury to the bone of the left thigh."

After considerable surmising as to the course of the man's life, he goes on to add, "The impossibility of cross-examination of the patient on his early history leaves free to conjecture as to the cause of the difficulty, but nothing seems so probable as that the defect of growth is due to an attack of poliomyelitis, perhaps even an intra-uterine one..... The subject is one for larger discussion and study -- and I have been concerned only with the presentation of what is, if my supposition of the causation is correct, the earliest known case of infantile palsy..."

Although this account has often been presented as evidence of the early occurrence of poliomyelitis, Mitchell himself would no doubt grant that, "...in the absence of other evidence of poliomyelitis than the shortening of the femur, the disease might equally have been of congenital origin (Mayer, 1917)."

An Egyptian stele of the eighteenth dynasty (1580-1350 B.C.) now in the Carlsberg Glyptothek at Copenhagen has often been pre-

sented as evidence of the antiquity of infantile paralysis. This example of Egyptian art has been analyzed by Ove Hamburger, both a physician and a student of ancient Egyptian art. His article, which appeared in 1911, has been translated into English since by Ejnar Hansen. Upon describing the three figures on the stele, a priest, a woman, and a child, he observes;

"When you look closely you will see that the figures are cut very distinctly by the artist, with precision and delicacy, but also that there is something wrong with the man's leg.

Of course this abnormality has been noticed, and in the catalogue you read; 'The drawing is not especially good. The man's one foot and leg is absolutely deformed; the stele is possibly from a later period, when the Egyptian art was decadent.' If the aforequoted Egyptologist had been a physician, he would surely not have made that statement. In this case there is undoubtedly no misdrawing. The artist has produced a man with a 'withered' leg. The foot is in the typical equinus-position. The slight flexion of the hip and knee joints is not enough to raise the heel so high from the ground. There is a shortening of the femur as well as the tibia and fibula. The whole leg is diminished in size. Another thing that speaks for the correctness of the drawing is the way Ruma is carrying his staff. It is originally the kind of cane Egyptians of quality used to carry, but in old pictures we always see them carrying it in front of them and parallel with the body. Ruma is carrying his cane in an unusual manner,

crosswise from the shoulder, in the bend of the elbow and alongside the withered leg, apparently as a support. If the artist has drawn the man as he was in life, it seems natural to think of either infantile paralysis or coxitis as being the cause of the deformity, and of these two infantile paralysis seems the most probable." (Hansen, 1913)

In a more conservative vein, Rurah rejects this as evidence of the early occurrence of poliomyelitis with the comment that, "... the deformity might, however, be due to some other lesion in the nervous system or to a disease of the hip joint contracted in infancy." (Rurah, 1932)

The ruins of a medieval Norse colony, Herjolfsnes, were found in South Greenland by an archeological expedition in 1921. A cemetery yielded about twenty-five skeletons dating from the fifteenth century. In studying these skeletons Hansen found, "A striking number of the individuals examined (6 out of the 25) have had diseases involving physical deformities." It is the belief of Aycock that such bony deformities as described in the Herjolfsnes skeletons are very probably to poliomyelitis suffered in childhood. (Aycock, 1929)

Although there is doubtless sufficient foundation for the view that such conjectural cases are only of interest as historical curiosities, there is also a tendency to give them some credence in the consideration of the epidemiology of the disease, and to include them in explanations of its puzzling epidemicity.

Though not brought forward until recent epidemics created interest, Hippocrates' account of an epidemic of paralysis on the island of Thasos is now thought to have been an epidemic of poliomyelitis. Hippocrates has recorded that, after a summer of severe droughts and an autumn of heavy rains, "during winter, paraplegia set in, and attacked many, and some died speedily; and otherwise the disease prevailed much in epidemic form, but persons remained free from all other diseases."

In his monograph of 1913, which still stands as a monumental work on poliomyelitis, Wickman gives credit to Bergenholtz for observing the first true epidemic. Of him he says, "He recorded in the Swedish Public Health reports eighteen cases of infantile paralysis which occurred in North Sweden in 1881." In further establishing poliomyelitis as an epidemic disease, Wickman states that, "Medin's lecture at the Tenth International Medical Congress at Berlin in 1890, upon his observations during the first epidemic in Stockholm (44 cases), convinced everyone that spinal infantile paralysis could appear in epidemic form."

Whether the instance described by Hippocrates was an epidemic of poliomyelitis, and whether epidemics have occurred unrecognized through the intervening years until the time of the report of Bergenholtz, are undoubtedly questions of significance in the broadest studies of the epidemiology of poliomyelitis. But, since it is definitely established that it is an epidemic disease at the present time, it is not the purpose here to enter

this controversy. It is, rather, to present the later developments in epidemiology and, if possible, to impress upon the reader the importance of the recognition of poliomyelitis as one of the true epidemic diseases. For surely, great progress may be made toward reducing the incidence of this disease, sensible preventive measures being our only weapon until an effective method of treatment is discovered.

PART I
EPIDEMIOLOGY OF THE DISEASE

There has undoubtedly been an increase in the incidence of poliomyelitis in the past fifty years along with other infectious diseases of the nervous system. But whether this increase is real or only apparent is sometimes questioned. In regard to this an editorial in the Medical Officer for May 14, 1932 says, "It is well to consider whether this increase is genuine and, if it is, to what it may be due. Special attention directed against any disease always results in an immediate rise of apparent incidence, followed by a fall which, however, does not reach the low level the disease appeared to have had before it was specially scrutinized. If the attention results in some satisfactory means of prevention, a further fall occurs which may reach any level short of zero. The bulk of the preliminary rise is naturally due to unearthing missed and trivial cases, but also to the inclusion of doubtfuls, which formerly were attributed to other conditions. The fall following the rise is due to stabilization of the dividing line which cuts across the doubtfuls. Whereas, before the special attention all doubtfuls are excluded, and in the first phase of attention all doubtfuls are included, experience teaches us to separate this class more accurately into positives and negatives. In the past poliomyelitis was synonymous

with infantile paralysis, now it is not, for we know that the disease does occur without the production of paralysis. Perhaps one third of the cases now rightly diagnosed as poliomyelitis do not end in paralysis, and so we should expect the incidence of the disease to have increased, apparently by one-third. The increase has, however, been much greater than this, suggesting that the disease is actually more frequent than it used to be."

The fact that poliomyelitis became a reportable disease in most countries at different times, most of them between 1910 and 1920, makes any comparison of incidence before and since that time valueless. In spite of these difficulties of comparison it is generally agreed that there has been a true increase in incidence. It is concluded that greater interest, better diagnosis, and compulsory reporting are actually the effects of the increase, rather than the cause.

Despite this greater interest, and much speculation, this increase is still without an adequate explanation. However, we are not without theories as to how poliomyelitis, which is known to have occurred in sporadic form throughout the world for many years, should suddenly become epidemic in its method of attack. Of the theories, the foremost are concerned with the three basic variants of epidemiology -- the virus of the disease, the host, and the transmission factor. In brief, these theories are;

(1) Changes in the Virus. An increased infectivity due to mutation of the virus is advanced in explanation by some observers.

In this respect, a strain has been reported which exhibited adaptation for monkeys through repeated passage; lost this quality for a time; and regained it later (Flexner and Amoss, 1924.)."

Others attribute the incidental rise to an increased virulence. In regard to this the human strains have not shown a rise or fall of virulence which is in any way relative to the epidemics, and, from the work of Flexner and others, it can be said that the viruses from the successive epidemics have been of equal virulence for monkeys. However, Park has encountered one strain which increased in virulence when passed very rapidly through several animals. Some epidemiologists hold that poliomyelitic virus is exhibiting an increased affinity for the central nervous system. The following hypothesis comes from Australia, "Many organisms exhibit strains that are biologically distinguishable. They must have evolved from a common progenitor, and that evolution must be continuous..... It is not far-fetched to think that the occurrence of epidemic poliomyelitis is due to the development of a more definite neurotropic tendency by a strain of an already wide spread virus. The march of that particular strain, unnoticed before, would then become evident as a prevalence of poliomyelitis (Dale, 1928)."

(2) Changes in the Host. Some investigators think that there has been an alteration in the resistance of the population at large. MacNalty attributed this change to the change in our style of living, and claims that the faster tempo of modern life is responsible

for an increased vulnerability of our nervous tissue which leads to an increase in incidence of nervous system infections (MacNalty, 1927). This is certainly not a new viewpoint, since Chas. Taylor, as early as 1867, presented the hypothesis that the increasing incidence of infantile paralysis in the United States at that time was due to the nervous strain associated with the efforts of pioneering in this relatively new country. The nervous system vulnerability which these pioneers passed on to their progeny made them the easy prey of infantile paralysis. (Taylor, 1867)

The various factors concerned in susceptibility will be considered in another part of this work.

(3) Changes in Transmission. The method of transmission of this disease is certainly not a settled question. But, if contact spread be conceded, direct or indirect, it is not unreasonable to assume that the tremendously increased rate and amount of transportation in the last fifty years, along with the trend toward urbanization, may be one of the principal causes of increase in a disease spread in this manner. While this increases the possibility of infection, it also adds to the difficulty of tracing routes of infection. This is one reason why some of the earliest epidemics are the best evidence we have to support the theory of contact transmission. It was much simpler for Wickman to trace an unbroken chain of infection in small rural groups in Sweden in 1905 than for Murphy to track down possible routes in the Omaha epidemic of 1937. The daily contacts of the modern city-dweller

are so numerous that they defy tracing. The effect of modern communication and travel on the incidence of poliomyelitis may be estimated by considering the following law of epidemiology; "Given the precedent conditions for infection to occur, then the probability of transmission increases proportionately to the extent to which aggregation and dispersal occur." (Stallybrass, 1931)

It is interesting to note that this argument has been encouraged by experimental work. Topley and co-workers have now shown that within isolated mouse communities, an epidemic tends to die out, leaving the survivors and the virus in a state of apparent equilibrium. The infection will flare up again on the addition to the community of fresh susceptibles, not only the latter being affected but also the survivors of the previous outbreak. The regular importation of fresh susceptibles will maintain the infection indefinitely, the disease waxing and waning.

Geographic Distribution. Following an epidemiologic study of poliomyelitis in New York City and surrounding territory in 1918, Lavinder, Freeman, and Frost concluded that, "Poliomyelitis has become so widespread as to make it evident that this disease is independent of any climatic or other conditions which are peculiar to any restricted part of the globe." (Lavinder, Freeman, and Frost, 1918) While this is an unassailable statement it must also be admitted that all of the large epidemics have been confined to those areas having cold weather for at least 3 months

POLIOMYELITIS CASE RATES
United States, 1915 - 1929



The upper figures represent the mean, the lower the median case rate per 100,000 for these years.

CHART I



(After Wells, from USPH Reports, 1915,- 1929)

each year. However, it does exist in every country for which records are available.

Latitude seems to have some effect on the endemic rate and epidemicity of this disease. In general, both the incidence in endemic form and the frequency and severity of the epidemics increase with distance from the equator, both in the northern and southern hemispheres. Most of the cases have been reported from northern United States and Canada, and northern Europe. The zone corresponding to this in the southern hemisphere is for the most part occupied by water, but the rates in Australia and the most southern parts of Africa and South America are very comparable. It must be admitted that a vast portion of China should be included with the group in the northern hemisphere. However, in spite of the similarity of climate, very few cases of poliomyelitis have been known to occur there according to Zia, who made a study of the disease in China in 1930. No epidemics have been reported from there.

Small epidemics have been to occur even in the tropics, but they are so rare that they amount to curiosities. Lebrede and Recio reported an epidemic of 140 cases in the Province of Santa Clara, Cuba in 1909; Morales reported an outbreak of ten cases in Porto Rico in 1928.

The case rates for the United States show the effect of latitude even in the short distance between the northern and southern borders. Not only is the endemic rate higher in the northern

states, but the frequent epidemics are superimposed while epidemics are practically unheard of in the South (see Chart I).

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Even the countries reporting the most cases of poliomyelitis have low rates for the disease. It is a disease of low incidence, compared to most diseases. In rare instances, as much as one per cent of a total population has been attacked, but these are the exception and usually confined to villages or small rural areas. In a large group of people, such as a city of 100,000 inhabitants or a county or state, a severe epidemic seldom attacks more than one in a thousand and usually not more than one in three or four thousand. In the most devastating epidemic known, in New York City in 1916, the incidence was less than three per thousand of population. From these rates it may be seen that the incidence of poliomyelitis, even during an epidemic, is usually less than the annual expectance of several of the more common endemic infectious diseases. One should not, however, allow this evidence to minimize the importance of the disease. Lavinder, Freeman and Frost stress this point as follows; "While the incidence among a population affected by poliomyelitis, even in its severest epidemic form, is usually not high, yet this disease possesses not infrequently the power to spread widely -- in fact to become endemic in a country. It has been suggested that an epidemic of infectious disease may be viewed as the resultant of two excursions, a vertical one representing the heaping up of cases in any locality, as usually shown plotted on any chart, and a lateral one

representing the extent of territory covered -- the geographic distribution. Epidemic diseases not infrequently show decided variation in these two movements. Certain of them, like dengue for example, show a most striking vertical movement along with a very limited lateral movement. Poliomyelitis has exhibited a significant power of lateral movement and, up to the present time, in much greater degree than its power of vertical excursion."

Dale calls this lateral spread "creeping tendency". In the opinion of most epidemiologists, it is suggestive of a host resistance which limits "heaping up", a small and widely spread population of susceptibles, and a widespread virus.

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Early in the study of epidemic poliomyelitis, Wickman has pointed out that this disease seemed to attack rural groups in preference to urban communities. It seemed to him that it even evaded the cities. This observation has been corroborated many times since; once an epidemic becomes established, even if initiated in the city, the rural incidence gradually comes to exceed that of the city of origin. This has been found to be true not only in isolated areas but in widespread epidemics. From a comparison of the rural and urban rates of a whole state this rural prevalence in epidemics is evident and is certainly not mere chance, but due to some unknown peculiarity of the spread of the virus. Not only in times of epidemics is this higher rate in the sparsely settled areas evident, but also the endemic rates for a twenty-year period in the northeastern United States were shown

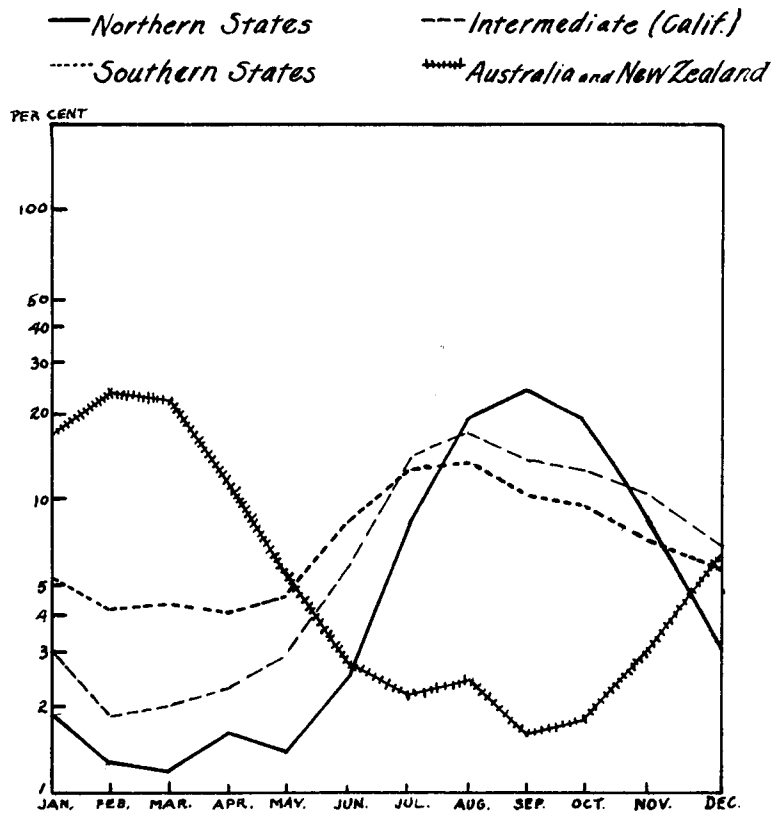
to be higher for communities of 5,000 or under than for those with a greater number, with the exception of the rates for New York city. The rates in this case were, of course, thrown clear out of balance by the epidemics of 1916 and 1931. This high rural case rate is plainly compatible with the "creeping tendency" of the disease, mentioned above. This type of spread should explain, to a certain extent, why the disease tends to spread from an urban focus and lead to a higher case rate in the surrounding rural districts, instead of "heaping up" in the city where the epidemic was first established.

Seasonal Incidence. Poliomyelitis is characteristically a disease of summer and fall, in both endemic and epidemic forms. Although sporadic cases, and even epidemics, may occur in any month of the year, the maximum rate of incidence occurs between July and October in the northern hemisphere and between January and April in the southern hemisphere, the seasons which are comparable in conditions of weather for these two parts of the world. Rarely do epidemics begin before summer, and, with very few exceptions, they disappear with the advent of moderately cold weather. This prevalence during the warmer months was first pointed out in 1875 by Sinkler. During a period of four years he observed eighty-six cases in Philadelphia. Of these, 89 per cent occurred in the interval from June to October.

While various explanations have been offered to explain the

CHART II

POLIOMYELITIS - SEASONAL DISTRIBUTION



(From Aycock, J. Prev. Med. 3: 245, 1929)

summer prevalence on some other basis than the seasonal factor, the most recent figures still support the idea that there is a seasonal influence, climatic or otherwise, upon the incidence of this disease. Nevertheless, Aycock and Eaton believe that this seasonal variation of poliomyelitis morbidity is definitely accentuated by seasonal expectancy on the part of physicians, leading to less prompt reporting in the months with lowest expectancy; and, that although predominantly a summer disease, it prevails to a greater extent throughout the year than reported cases would seem to indicate. They call attention to a comparison of the morbidity and mortality rates by months for poliomyelitis, and point particularly to the fact that the case fatality is noticeably higher in those months with the fewest cases. They attribute this to the necessity of reporting deaths at all times, and also to the hesitancy of many physicians to label cases as poliomyelitis in the winter. (Aycock and Eaton, 1924)

By a comparison of the incidence of poliomyelitis over a sixteen year period by months in the northern United States, southern United States, intermediate states, and Australia and New Zealand it is evident that seasonal variation bears a relationship to geographic distribution. The chart on the opposite page (Chart II) has been prepared by Aycock to illustrate this relationship. In order that the difference in total incidence for the various locations should not distort the comparison, the values on this chart are the percentages of the total cases in these years which have

occurred in each month. The resultant curves are therefore representative only of seasonal fluctuation.

"It will be noted that the most marked seasonal variation takes place in the Northern United States and in Australia and New Zealand, the latter curve being almost exactly the reverse of that for the Northern United States with its peak in February and March, the season in the Southern hemisphere which corresponds to August and September in the Northern hemisphere. It is further shown that the disease tends to occur more evenly throughout the year in the Southern United States." (Aycock, 1929)

With better diagnosis, greater vigilance on the part of the health departments, and the recent publicity acquired by poliomyelitis, the errors of poor reporting should now be discounted. The latest epidemics have all occurred in the late summer and early fall; no change has been noted in the seasonal variation. It must be admitted that the season is a factor in the occurrence of this disease, at least in its epidemic form, suggesting that climatic conditions may influence the transmission of the virus in some manner as yet unknown.

In spite of the foregoing evidence, winter epidemics are more frequent than was formerly suspected, and, though the facts may seem to refute much of the above material, they should be presented here.

In the laboratory of Flexner and Lewis a specimen of cord from a human case retained its virulence for 40 days at -2° C to -4° C. They comment; "These experiments have a bearing on the

epidemiology of the disease, and indicate that the reduction in cases which occurs with the onset of cold weather does not depend on the destruction of the virus, although it may have to do with an effect on its multiplication." (Flexner and Lewis, 1910)

Leake, Bolten, and Smith who investigated an outbreak of poliomyelitis in Elkins, West Virginia, in December 1916 and in January 1917, say in part; "Particular attention was attracted to the West Virginia epidemic of poliomyelitis because it occurred in the winter. At the time it was thought to be the first winter outbreak in the United States; indeed it was the first to assume anything like epidemic proportions, but a search of the records has revealed that other outbreaks in this country have occurred during cold weather. Scandanavian observers had called attention to a few winter outbreaks in Sweden and Norway although no definite study of the temperature conditions had been made..... A sort of criterion must be adopted as to what constitutes a winter outbreak. Therefore, it is here assumed that to be considered a winter outbreak two or more connected cases must occur after the temperature has remained below freezing for twenty-four hours. A study of the literature reveals 38 such outbreaks not including this one..... Some of these outbreaks occurred far north of the Arctic Circle..... In 15 of the outbreaks the thermometer went below 0° Fahrenheit, the lowest temperature recorded being -29° Fahrenheit at Misvar, Norway." (Leake, Bolten, and Smith, 1917)

A study of the 39 outbreaks cited by these writers shows that,

It has been said that if a community has an epidemic of infantile paralysis one year, it may be expected to be relatively free of many cases for a varying number of years.

Wernstedt made a study of the Swedish foci of 1905 and 1911 to determine if one epidemic conferred any amount of immunity to subsequent attacks. He found that in none of the five principal centers of the 1905 epidemic wave did the disease appear epidemically in 1911, though the immediately surrounding localities registered a very high incidence in the later attack. Conversely, he found that those counties which suffered the least in 1905 were the ones most severely ravaged in the 1911 epidemic. (Wernstedt, 1912)

This work of Wernstedt's has been corroborated by Dubois in Zurich and by Lavinder, Freeman, and Frost in New York state. They also found that there is a tendency for the inter-epidemic interval to be longer after the larger epidemics. It has often been observed that the lower the incidence in inter-epidemic years, the higher will be the incidence in epidemic years. Although it is uncertain to what this apparent immunity is due, following an outbreak, it has been assumed that an extensive immunizing wave accompanies the recognized cases.

In this regard Kramer and Aycock presented a report in 1931, based on neutralization tests upon children in the small town of Bedford, Massachusetts, which would seem to indicate that is not due to any increase in specific immunity. In October, 1930, five frank cases of poliomyelitis occurred in Bedford, a town of 1700.

(This shows an incidence of 29⁴ per 100,000; the incidence in New York City in 1916 was 185 per 100,000.) Five months later, they tested 20 Bedford children who had passed through minor illnesses at the time of the epidemic (possibly abortive poliomyelitis), and 28 who had remained well. For comparison, they tested 29 children from Burlington, Massachusetts, where no cases had occurred. The results of the immunity tests in these three groups of children were practically identical and compared favorably with tests made on normal children previously. (Kramer and Aycock, 1931)

It has been pointed out that, though localities which had been the centers of epidemics tended to be free for several years, epidemics are more apt to recur there than in communities which run a more steady endemic rate. Reservoirs of infection, possibly carriers, have been blamed for this tendency, but the above findings of Kramer and Aycock would seem to indicate that some environmental factor in the particular locality either lowers host resistance or else affects the mode of transmission.

The best explanation for the bizarre way in which poliomyelitis exhibits periodicity or, if you wish, a lack of it, is based on the theory that those areas most affected represented a chance accumulation of susceptibles. Susceptible not only in that they lack specific immunity but also because they are lacking in what is termed non-specific resistance. However, if this were the entire explanation, we would expect the age group most affected to be older than usual in epidemics occurring in areas unaffected

for several years, but this is not found to be true. The age incidence of poliomyelitis is probably the most constant factor of a strangely variable disease.

Incubation Period. There has been little accord until recently on the length of the incubation period in poliomyelitis. The early workers were hindered mainly by their failure to recognize multiple cases as common source infections. Consequently, their tendency was to set the interval shorter than is now thought to be right. Since the mode of transmission is still a debatable question in clinical poliomyelitis, the only available figures must necessarily be computed from the time a person is known to have come in contact with a case until this person begins to present symptoms of the disease. There is some question whether this is a true measure of the incubation period. The interval between inoculation and appearance of the disease in monkeys is from five to eight days according to Flexner. This cannot be compared to the human cases but it is interesting to note that many observers claim that this is very close to their findings in clinical work. The average of figures reported in the past 20 years gives us a period of from 6 to 20 days, with the majority of cases falling closer to 6 days than to 20.

Contact Between Cases. One of the most striking features of poliomyelitis is the lack of obvious connection between cases. In the last few years more prompt reporting and better diagnosis (of abortive cases especially) have cleared this up to some extent,

yet it is only in a small percentage of instances that a definite series of cases and contacts may be secured. The New York City Health Department investigated carefully the epidemic in Brooklyn in 1931. Of the first 500 cases, in 31 or only 6.2 per cent was evidence of contact with previous cases established. No proof of association with other cases could be obtained in any of the remaining 93.8 per cent. (New York City Dept. of Health, 1931)

In an epidemic of 100 cases in Glasgow in 1928, in only two cases was it possible to trace any direct connection between the cases.

From these instances it is plain that there is some factor in the spread of this disease which, up to the present, is not understood. Certain it is that many small epidemics are reported in which contacts could be traced in all cases. However, in the light of our present knowledge, we cannot say that the same number of cases would not have occurred and in the same individuals even if there had been no actual contact as we now use the word.

From the foregoing it may be concluded that either the virus is transferred by agencies other than human beings, or else the clinically healthy carrier plays an important role in transmission of the disease. The latter possibility only will be considered in this section.

In a disease of low incidence but with a high carrier rate, the logical explanation would be a widespread virus; a virus that

is either low in virulence or held down by a host resistance of high degree. This should be demonstrable, but such is not the case. True, many have found the virus in a high percentage in small groups, but since negative results are rarely reported, and since the investigation has not been general enough in its scope, it is too early to form an opinion.

The experimental evidence of carrier infection is meager but not contradictory because of this. The fact that there is no animal naturally susceptible to poliomyelitis presents a great difficulty in such a study. The lack of a method of artificial culture which would allow increasing the dose before inoculating animals is another important deterrent.

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If neutralization of the virus by the serum of a suspected carrier is accepted as evidence of previous infection, no further proof is necessary to establish a high carrier rate. However, the possibility has been suggested that this power of the sera may develop without contact with the virus. Until this is settled little weight is carried by experiments along this line.

Fortunately, clinical studies of epidemic poliomyelitis are able to furnish more convincing proof as to the role played by human carriers in the disease. Wickman was the first to recognize and thoroughly study this problem. He worked in isolated areas where the complexity of communication did not muddle his investigations; where contacts were few and people remembered the persons they had met each day. From these investigations, he

came to realize the epidemiologic significance of abortive cases and healthy carriers. He was impressed with the fact that the disease seemed more often to be propagated through the medium of healthy carriers than from case to case. His reports of several small epidemics in Sweden in the early 1900's still stand as the best evidence we have of the contagiousness of poliomyelitis. In several of these groups he was able to trace the course of the virus in its itinerary to include every case occurring in that area.

The length of time that the virus is carried by one host is largely based on experimental evidence, and the times reported by the various observers are all different so apparently this evidence is of little value.

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The only epidemiological evidence as to chronic carriage is based on the above described tendency of epidemics to recur in the same areas. This is not at all conclusive.

There has been some question as to whether actual cases are more infective than healthy carriers. Since the only difference between frank poliomyelitis and an abortive case is that in the former the nervous system has been invaded, there is no reason to believe that one is any more infective than the other. Experience vindicates this assumption.

Even before Wickman's reports were published it had been noted that poliomyelitis spread followed the lines of transportation. This has been evident in all epidemics. Even though it is often

while reported as separate outbreaks, most of them were really the parts of two great epidemic waves, the peaks of which occurred in the summer. Fifteen could be traced as parts of the epidemic of 1904-1906 on the Scandinavian peninsula, and fifteen others were related to the great epidemic of 1910-1913 in the same area. All of the other epidemics could be found to be connected to summer epidemics with a much higher incidence. The intimation here is that that these cases represent merely a more prolonged extension into the winter months of a summer prevalence than is usually encountered.

Periodicity. Epidemic poliomyelitis seems to present no tendency of periodicity in occurrence such as is seen in most of the infectious diseases. A few isolated localities report a tendency of exacerbation at quite regular intervals but this is unusual. In the northeastern United States, where poliomyelitis common, epidemics are usually from three to five years apart. Small communities in this same territory may have only sporadic cases for many years, while others report recurrences spaced by only two-year intervals. Not only the epidemics show this quality, the endemic prevalence is also very irregular. Forsbeck and Luther state that in Massachusetts poliomyelitis is the most variable of the common communicable diseases in yearly incidence. In the other states the same is found to be true. In New Jersey, in 1916, 4055 cases were reported, while in the preceding year only 36 had been reported.

impossible to trace contacts between cases, it is observed that the disease as a whole tends to follow highways and railrad lines. Wickman inferred from this that the spread is effected in this disease by human agencies. Recent work has shown that polio-myelitis tends to spread along water routes and natural drainage contours. This is not contradictory if one will but remember that the railroads and highways have been built along the same water routes and drainage lines, mainly because of less difficulty in construction along these natural contours.

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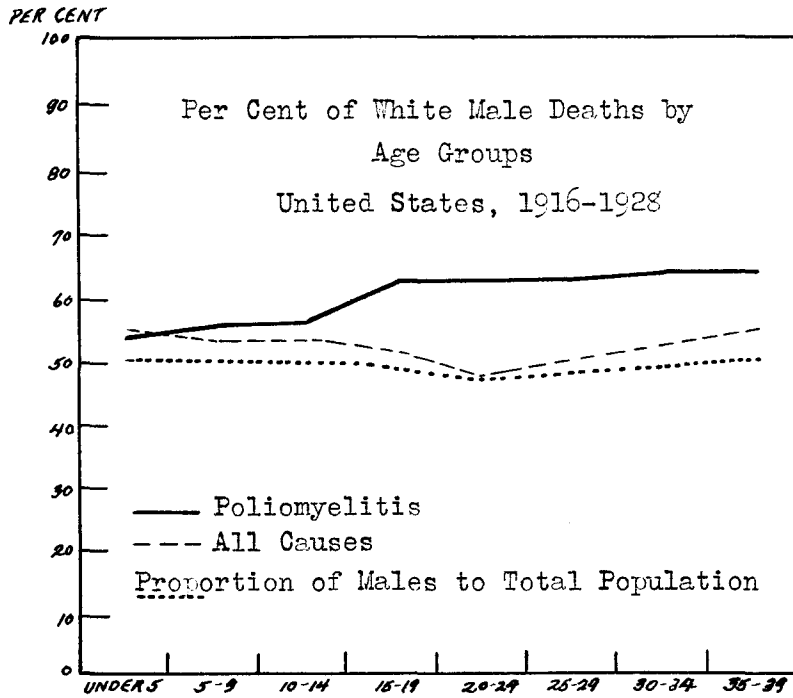
were in isolated localities without any facilities for investigation, or even diagnosis in most instances. The residual paralysees of the disease have, however, made it possible for a few observers to make diagnoses and study such epidemics in retrospect.

According to Grunwell, a United States Navy surgeon, there was an epidemic of poliomyelitis on the island of Guam which had apparently started in May, 1899. (Grunwell, 1900) Though he did not arrive on the island until several months later and had to rely on the accounts of others concerning the acute stage, he states very clearly that the extremes of age in the cases observed were from 15 to 50 years of age. Prior to this time poliomyelitis was unheard of in Guam.

An interesting epidemic occurred on the Pacific island of Nauru in 1910. This island lies almost on the equator and has such a climate as would be expected. The population at that time consisted of about 1250 natives, 80 Europeans, and about 1000 adult laborers from China and the Caroline Islands, recently imported. (The proportion of Chinese and Carolinians is not given.) In January of 1910 an epidemic started with a case in one of the Carolinian laborers. Inside of 14 days there were attacked: 470 of the natives, 220 laborers from the Caroline Islands, and only 2 Europeans. The Chinese were not affected. Muller's report says that very few children below 12 years of age were sick, and no aged persons. There is no reason given to explain why the native

CHART V

POLIOMYELITIS DEATHS and
DEATHS FROM ALL CAUSES



(After Jungeblut and Engle, U.S. Census Rep. 1916-28)

better diagnosis, probably accounts for this trend of incidence.

Another explanation for the apparent susceptibility of children to poliomyelitis may eventually be found in the recent work of Aycock upon castrated monkeys. Starting with the hypothesis that pregnant women are more susceptible to poliomyelitis than are non-pregnant (generally accepted), and assuming this to be due to changes in the estrogens, he attempted to prove that estrin activity influenced susceptibility. He has been successful insofar as he has shown that administration of estrin to castrated monkeys does increase their resistance to nasally instilled poliomyelitic virus, presumably through the effect of this substance on the nasal mucosa. (Aycock, 1937) This may be a factor in the development of a greater non-specific resistance in those past the age of puberty.

Sex Incidence. One of the most constant epidemiological findings in poliomyelitis is the preponderance of male cases over female. The official morbidity statistics of the United States are not recorded by sex so a complete report is not possible, but from a compilation of 36,000 cases the ratio of male to female cases is 1.3 to 1.0 . The case fatality is very close to the same ratio. The sex incidence ratio does not vary with the season, geographical distribution, density of population, or in rural as compared to urban groups. Age is the only factor which influences this ratio, the male incidence tending to rise with age.

PART II
EPIDEMIOLOGY OF THE HOST

To those attempting to work out some foundation for the mode of spread of poliomyelitis one fact is evident, that in this respect it is a very contradictory disease. The group working for the Milbank Foundation concluded that this was probably the resultant of two varying factors, the spread of the virus and the susceptibility of the individual. In the following pages these two epidemiologic variants will be considered separately in their relation to poliomyelitis.

There is an increasing belief that those who develop the disease rather than immunity when infected with the poliomyelitis virus vary in some manner from the normal. The factors which, from experience, have something to do with susceptibility to the disease are age, sex, race, and constitutional factors.

Age Incidence. As stated before, the first attack of a virus on a population, at least the first after a very long period, should bring to light some knowledge as to the susceptibility of the various age groups. This type of "virgin soil" epidemic should give us some idea as to whether there is such a thing as an acquired specific immunity to poliomyelitis.

Such epidemics have occurred but, as would be expected, all

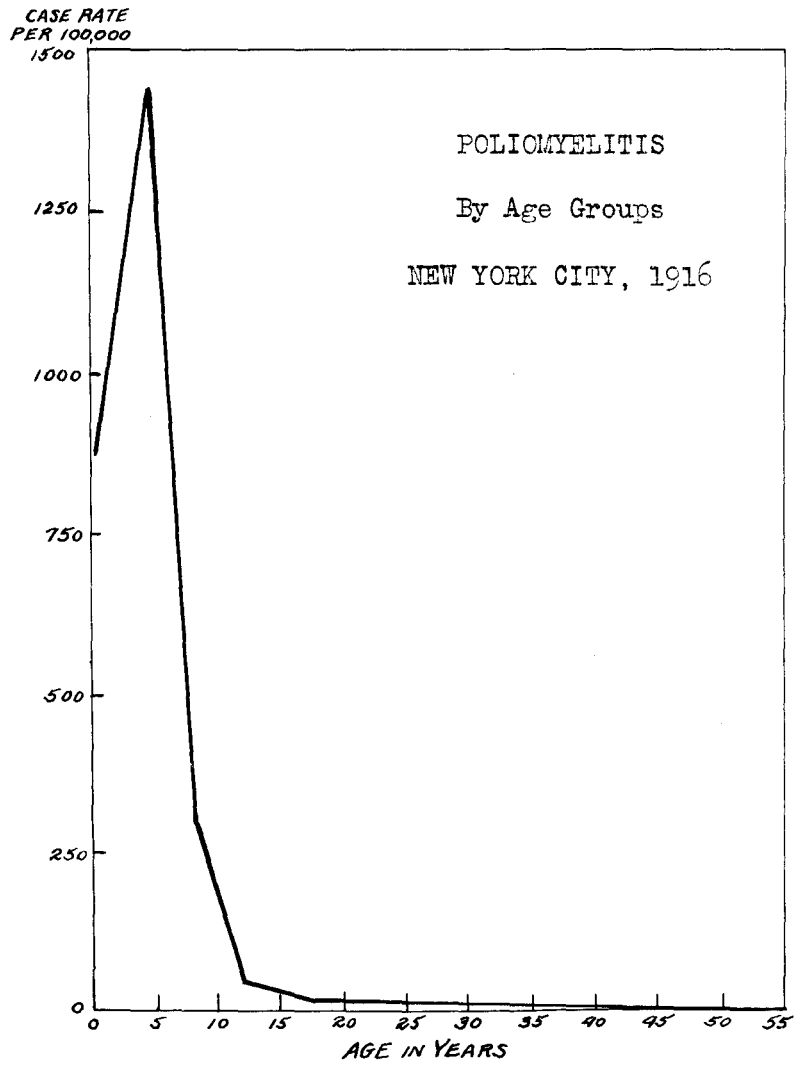
exact similarity of these two curves.

The case fatality for the various age groups varies considerably. Roughly, it is inversely proportional to the incidence for the age group. The high case fatality for the older age group is not compatible with our present knowledge of the disease. Why the older group should succumb more readily to an infection which, in general, they show more resistance toward cannot be explained. The only possible cause now offered is an acquired sensitivity to the virus.

It has been observed many times that the age incidence during an outbreak of poliomyelitis will vary with the density of population. The greater the density, the younger will be the age group to suffer the most. This is, of course, compatible with the theory that there is an acquired immunity. In densely populated localities the people would necessarily be exposed to the virus more generally than in widely scattered groups. This, of course, would lead to a more complete group of immunes for each age group exposed, so that most of the susceptibles in the subsequent epidemics would be those experiencing their first exposure to the disease, Those born since the last epidemic.

Recently a tendency for an increasing incidence of poliomyelitis in the higher age groups has been commented upon by several observers. There is a feeling, however, that the major portion of this is due to the realization by present day physicians that poliomyelitis can and does occur in adults. This, added to

CHART IV



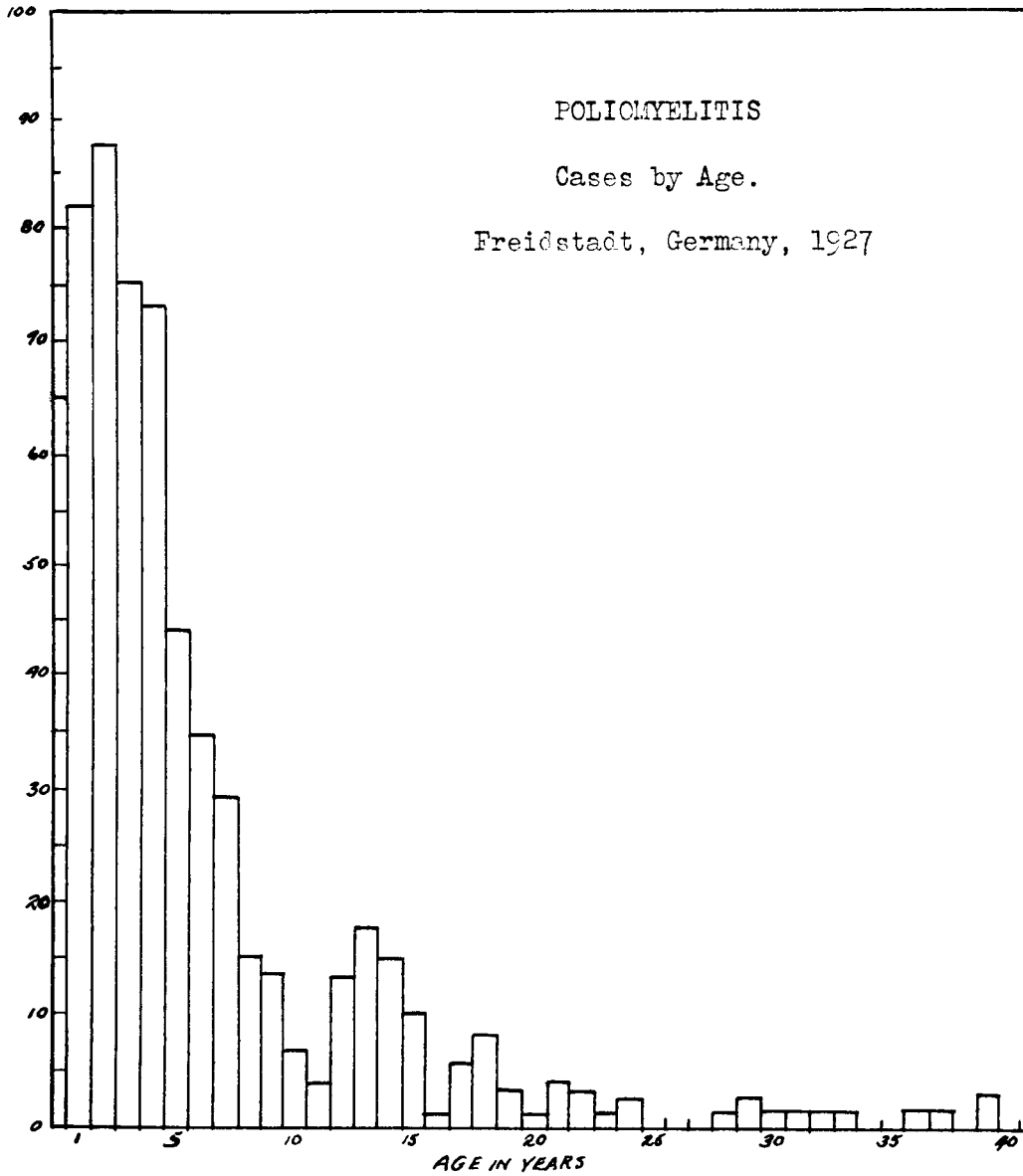
(From Lavinder, Freeman, and Frost, USPH Bull. 91, 1918)

children should be so little affected. (Muller, 1910)

In 1929 an epidemic of poliomyelitis occurred in New Guinea. So far as was known, no previous cases had ever occurred there, although it lies close to the British Solomon Islands where sporadic cases had occurred for a number of years. These islands were in close communication for many years and there is some doubt whether this might be regarded as a virgin soil epidemic, but the age incidence is notable. In one group of 138 cases 87 per cent were adults, while adults constituted 91 per cent of another series of 184 cases nearby. There was no known disproportion of adults in these localities or any other factor to explain the low rate of attack on children.

In spite of the obvious conclusions one may draw as to the importance of acquired immunity from these accounts, it is definitely established that clinical poliomyelitis throughout the world is a disease affecting predominantly the early age group. The chart facing this page, compiled from statistics on the epidemic in Friestadt, Germany, in 1927, is typical of most epidemics in this respect. Similarly plotted age incidence curves for almost all epidemics (and even endemic rates over a period of years) are so nearly like this one that their inclusion here is not necessary. The chart on the following page, prepared from data on the 1916 epidemic in New York, is presented here because it has been corrected to include the factor of percentage of the various age groups in the total population. It is striking to note the almost

CHART III



(From Krahn, Arch. f. Hyg. 101: 65, 1929)

No adequate reason is known for this difference. The possibility of greater exposure of males has been suggested but if this were true we should expect to find a greater immunity in males in the older age groups. It would seem that the most plausible explanation lies in a fundamental difference in the natural susceptibility. The work of Aycock upon the effects of estrogens in susceptible animals may lead to proof of this theory in the near future.

Racial Incidence. Since we are without any comprehensive survey of the racial incidence of poliomyelitis, little can be concluded in this respect. From available data it may only be said that no absolute racial susceptibility or resistance to poliomyelitis has ever been demonstrated. Studies have been made where the representation of races seemed ideal for such work, but the uncontrollable factors involved were such that the results are not of sufficient value to record.

While much time has been spent in studying the variations of susceptibility with respect to age, sex, etc.; and while this time might not have been spent in vain, most of those who have studied thus will admit that the problem of susceptibility has not been clarified to any appreciable extent thereby. In the light of the most recent work it is now felt that further study of the constitutional factors concerned may lead to a more definite answer.

Since it is quite well established that the only test by

which persons who are susceptible to poliomyelitis may be distinguished from those who are not is the failure of their serum to neutralize, widespread testing is indicated. However, this test is so expensive, involving the use of a monkey for each one, that its use is extremely limited. It is a regrettable fact also that of the number tested to date, in only a very small percentage have the constitutional differences of those who do or do not neutralize been studied. Therefore we must rely on clinical observations for the following considerations.

Constitutional Factors. Amoss pointed out in 1930 that natural resistance may be due to local defense mechanisms. He and Taylor demonstrated a substance possessing the power to neutralize or destroy the virus in the washings of the nasopharynx of healthy persons. This power diminishes or disappears as the mucous membrane becomes the seat of inflammation. In their opinion, this is the first line of defense.

Permeability or impermeability of the nasal mucosa constitutes the second barrier in their group. The prevalence of the disease in those exposed who had had recent tonsillectomies was considered strongly suggestive in this respect. In support of this theory, the recent work of German and Trask (1938) may be significant. In experiments with a large series of monkeys, it was found that various operative procedures involving the upper respiratory passages definitely increased their susceptibility to poliomyelitic virus.

A third line of defense consists in the integrity of the meningeal-choroidal plexus, the normal safeguard of the nervous system against infection via the blood stream. Flexner and Amoss, more than 25 years ago, showed that infection of monkeys with poliomyelitis virus by way of the blood stream, a route formerly closed, could be accomplished after setting up an aseptic meningitis by injecting a foreign protein. Nasal instillation infection was also rendered easier by such preparation.

It is not unreasonable to assume that herein lies a large factor in the difference of susceptibility to the disease. It may also be the only factor in determining which of the cases will show paralysis and which will not.

Draper (1932) was the first to intimate that susceptibility to poliomyelitis might depend on an endocrine imbalance. Though much of the more recent work is not exactly corroborative of his findings, neither does it disprove them. His concluding remarks (after a study of the New York epidemics of 1916 and 1931) are; "Thus it appears that the constitutional structure of the infantile paralysis people points strongly to deficiencies of the three glands, namely, the pituitary, gonad, and adrenal cortex. What the significance of the lymphatism and mongoloid trend may be is still unexplained..... In conclusion, it may be said that this highly specialized type of child is a causal factor in the occurrence of infantile paralysis, of equal importance with the virus; but so far as the development of paralysis is concerned, the

constitution of the child is of greater significance than the virus."

Neal, who has an enormously wide clinical experience with the disease, notes nothing peculiar in the physical makeup of poliomyelitis patients. (Neal, 1940) In fact, the only part of Draper's statement with which she, with many others, is in accord is that endocrine imbalance may be a factor in the susceptibility to the disease. The experiments (noted above under sex incidence) with estrogenic substances are very convincing in this respect.

Jungeblut and Engle (1932) have claimed some success in the immunization of immature monkeys against intracerebral inoculation with poliomyelitis virus by the administration of anterior pituitary extract, but their results have not yet been uniform enough to be entirely convincing.

In the past fifteen years there has been considerable attention directed toward the relation of susceptibility to the various blood groups. The first reports on blood groups came from a study conducted during the epidemic of 1927 in Roumania. However, the results obtained by two separate groups of observers were so exactly opposed that no credence can be given to either. The most extensive study yet carried out in this field was conducted by Jungeblut and Smith (1932). Working with a total of 578 sera obtained from the 1931 epidemic in New York City, they found that the number of cases in each group compared very favorably with the percentage of each group in the total population. Their only

finding of significance was the low incidence in the B group in those over five years of age, suggesting some related factor which confers immunity upon this group. Later work by these men has shown that the normal sera of those in the B group shows a much higher titer in neutralization tests than the other three groups.

Apparently this work is not sufficiently convincing to arouse any great amount of interest as practically no new work has been reported in relation to this question.

The possibility of inheritance being a factor in susceptibility to poliomyelitis has often been considered. Dubois was convinced of the importance of this from his study of an epidemic in Zurich in 1930. He based his conclusions on a group of 310 cases in which the family history was available; among these he found 14 instances of recurrences in the second generation. It would seem that much more proof is necessary in this respect to be arrive at a conclusion. It may be significant to add that the most recent works (Neal; Trask and Paul, 1940) on the epidemiology of poliomyelitis do not even mention the inheritance factor.

In conclusion, it must be admitted that of the factors in susceptibility of the host considered above few are controllable quantities. However, insofar as our incomplete knowledge of this disease prevents us from making a direct attack upon it, further consideration of such factors must remain a part of our search for any means of prevention.

PART III
MODES OF TRANSMISSION

Though the infectiousness of poliomyelitis was generally accepted after Medin's report of his observations in Stockholm in 1890, it was not definitely established until 1903. In that year Landsteiner and Popper successfully transmitted the disease from a human to two monkeys. This work was corroborated by at least three other groups within the next year. Flexner and Lewis also carried the disease through a series of monkeys (1909). Since these experiments, many more have been conducted in an effort to establish the mode, or modes, of transmission of the infective agent. Although progress has resulted, there is, up to the present time, no unanimity of opinion. The various possibilities, each of which is the object of extensive research (especially in the last five years), are considered in the following pages.

Contagion. Direct contact infection from person to person, or contagion, is the most universally accepted theory of transmission of poliomyelitis. Proponents of this theory hold that the virus is carried and disseminated by man; that during the infective stage the virus is present in the nasopharyngeal secretions; and that it is transferred, probably by droplets, from infected

cases to individuals who come into sufficiently intimate contact with such cases, as in coughing, sneezing, or breathing their expired air in poorly ventilated rooms.

In support of this theory it may be said that the virus has never been found anywhere except in human beings or in animals or substances experimentally inoculated. That the virus is resistant to drying and cold has been proven by Flexner and Lewis in 1910, but the fact still remains that there is no definite proof of its existing as other than an obligatory parasite upon man.

This raises the question as to where the virus is harbored by man, where it grows, how it is discharged, and by what route it is conveyed to others.

The nasopharynx was thought for many years to be the only portal of entry for the virus of poliomyelitis, and also its means of exit. Only one other route, the gastrointestinal, had even been considered in the past. Recently, infection via the skin has been reported in the literature (Leake, 1935).

A great deal of evidence has been forthcoming to prove the contention that this virus does inhabit the nasopharynx of the host. Chief points in this evidence are; " (1) The detection of the virus in the nasopharyngeal mucosa of human cases at autopsy, and in the nasopharyngeal washings of cases and contacts. (2) The detection of the virus in the nasopharyngeal washings of monkeys with the experimental disease. Regardless of the route by which monkeys have been inoculated, the virus can be recovered from the

washings of the upper respiratory passages. (3) The production of the disease in monkeys by the nasal route, the virus having been demonstrated to pass the uninjured mucous membrane." (Wells, 1932)

The most significant work in regard to the transmission of poliomyelitis in recent years was reported by Trask and Paul. They have found that poliomyelitic virus can be obtained from patients in either acute or convalescent stages. Of this they say, in part; "Not only is the virus present in the stools but it is about twice as easy to isolate it from human stools or intestinal contents as from the human nasopharynx. This fact can be obtained from the literature. Of some 300 tests which have been made on human material from the nasopharynx, about 10 per cent have been positive for the virus; whereas, of some 90 tests on stools, 23 per cent have been positive." (Paul and Trask, 1939) That the virus could be isolated from stools was reported by Wernstadt more than 25 years ago but until now the fact had not gained much attention. Paul and Trask consider this development "so striking, that in some respects at least, it becomes necessary to consider poliomyelitis almost as an intestinal disease."

Supplementary to this is the fact that lesions in the intestinal tract have been noted in fatal cases, and also that the virus has been detected in the human mesenteric lymph glands.

In the last few years there has been an extensive investigation of the olfactory nerve as a possible route of infection of the central nervous system. Sabin and Olitsky conclude that this

nerve is the only apparent route of infection since mechanical interruption of the nerve prevents development of the disease in monkeys subjected to nasal insufflation with the virus (a method which usually shows a high percentage of "takes"). (1938) In support of this Flexner found, in similar experiments, that the olfactory lobes were the only part to contain the virus 48 hours after exposure.

It is interesting to note that only a year previous to the above work Sabin and Olitsky found that fatal cases have generally failed to reveal lesions at autopsy indicative of olfactory infection, intimating that there must be another possible route of infection. (Sabin and Olitsky, 1937) This suggestion has now been substantiated by German and Trask. In their laboratory, bilateral olfactory neurectomy did not prevent experimental poliomyelitis from developing in monkeys infected by intravenous or by intracutaneous routes. (German and Trask, 1938)

An interesting experiment was performed accidentally in 1935, when vaccines were being tried in this disease. It was found that human poliomyelitis can be established in a child by injecting the virus under the skin. (Leake, 1935) The most significant lesson to be learned from this mistake is that the virus may possibly be infectious regardless of the area of contact.

Disregarding the portal of entry of poliomyelitic virus, it was early established (Wickman, 1913) that person to person contact, or contagion, is at least one of the methods of spread of

this virus. Therefore, from a preventive medical viewpoint, it is not necessary to establish a definite portal of entry; isolation of cases is indicated no matter if the virus infects through the skin, respiratory, or gastrointestinal tracts. The question is whether any other preventive measure might have some effect to lower the incidence of this disease. This, of course, leads to the question of non-human spread, by objects either animate or inanimate. Certainly the preponderance of cases in which contagion cannot be proven would warrant serious consideration of this possibility.

Non-human Carriage. The possibility that epidemics of poliomyelitis may be milk-borne was investigated early, possibly because it attacks especially the age group which consumes the most milk. It was found that although a few epidemics may have been due to infection through the milk supply, the majority of attacks cannot be thus explained. It was also found that those small epidemics which were quite conclusively proven to be milk-borne were of a more explosive type than the usual epidemic. The age incidence did not follow the usual curve in these groups either, more older people were affected, indicating that this is not is not the usual means of dissemination of the virus. It may be significant that no epidemics have been reported as milk-borne in the years since pasteurization became so universal, yet we have epidemics as often as before.

In many early epidemics of poliomyelitis the water supply was considered as a possible means of transmission of the virus. No evidence to support the idea was found and the subject was apparently dropped. In 1928, however, Kling reopened the matter and presented his "hydric theory" which is given much credence in Europe today. He bases this theory on the belief that the virus enters the body through the intestine and that the feces are infective. Among the points presented in support of poliomyelitis being a water-borne disease are, "(1) That the disease has a seasonal distribution similar to typhoid." (2) That the disease follows the waterways. (3) That the relation of foci is determined by the course of a stream or by drainage contours. Kling also states that the Swedish villages which filter the surface water for drinking were more often attacked (36.8 per cent) than those that use deep well water (6.6 per cent).

Add to this the work of Paul and Trask in 1938 upon stool specimens and the observations of Kling seem to bear more weight. These men have found that, "Stool specimen suspensions offer a stable medium for the preservation of poliomyelitic virus. It remains viable in this medium for months. This suggests that, during epidemic times, heavy pollution of the sewage with the poliomyelitis virus is certainly possible."

Murphy, in his study of poliomyelitis in Omaha in 1937, also noted that the disease followed waterways or drainage contours as the epidemic progressed.

The full significance of water in the spread of poliomyelitis has probably not been uncovered and until further work is done in this field it must remain in the status of only a possible means of transmission. Whereas the European workers are now well convinced that we will find water to be the chief mode of spread of the virus, observers in this country are more inclined to leave it in a questionable position.

Fomites, as an intermediary means of transmitting poliomyelitis, have been given more attention than is warranted, in the opinion of modern epidemiologists. Theoretically, infection by this means is possible in any contagious disease. This possibility, of course, depends on the resistance of the virus to conditions outside the body. Poliomyelitic virus is resistant to these conditions but low temperatures are necessary. This, of course, is not compatible with the fact that poliomyelitis is a summer disease. The general trend has been to discount the importance of fomites in the dissemination of any contagious disease in favor of direct contact through infective droplets. It has been pointed out that the idea that diseases were transmitted by fomites was seriously deflated by the later discovery of "human carriers" in these diseases.

Second cases in the same house occurring at varying intervals after the first case are the usual cases cited in support of fomite infection. Such cases are now thought to be the result of infection from chronic carriers.

Probably no other means of transmission of poliomyelitis has been held under suspicion as long, in spite of much negative evidence, as infection through insects. Though the consensus of opinion at present is that insects play no part biologically in the spread of poliomyelitis, and at most a very small part as mechanical agents of transmission, a number of research groups are investigating the question further. Wells gives a set of epidemiological requisites to be satisfied before biological or vector transmission can be recognized, "(1) proof of the presence of the virus in the blood of human beings, (2) the ready production of the disease experimentally by the intravenous, or less possibly by the subcutaneous route, (3) finding the virus in a suspected insect, (4) the actual production of the experimental disease by the feeding of insects upon infected animals and subsequently upon normal animals." (Wells, 1932)

These requisites have not been fulfilled, but the fact that monkeys are not nearly as susceptible to poliomyelitis virus as is man may prove to be the explanation for the negative results thus far. This factor plus the recent accidental infection of children by the subcutaneous route suggests that herein may lie the solution of the transmission of poliomyelitic virus.

The epidemiologic evidence tends to favor insect dissemination. Chief points in the evidence are, (1) Poliomyelitis is similar in seasonal incidence to other insect-borne diseases. (2) It is characteristic of the disease that only a small percent

of cases can be traced to previous cases, and that the incidence in those known to have been exposed is low. (3) Certain facts in the spatial distribution of poliomyelitis suggest some insect vector, the rural incidence equals or exceeds the urban rate. Sporadic cases are explained by the possibility of an animal reservoir of virus, as yet undetected.

After his study of the Omaha epidemic, Murphy concluded, "-- The circumstances surrounding the epidemiology of poliomyelitis indicate the probability of a wind impelled or wind influenced vector as a mode of transmission of the disease, such as, for example, a mosquito."

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