Seasonal and Age Studies of Poliomyelitis and What They Suggest*

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A PREVIOUS study of the age distribution of poliomyelitis in comparison with that of measles and diphtheria,[1] in which diseases the phenomenons responsible for age distribution can be measured by direct observation or test, indicated that the virus of poliomyelitis is widespread and that it sets up a widespread immunity to the disease. It was concluded that the incidence of the disease is not in reality a measure of the extent of the distribution of its virus but is limited to a great extent by existing immunity which must have come from previous exposure to the virus.

This observation raised the question as to what determines the result of initial exposure to the virus—the frequency with which it causes an actual attack of the disease or produces (or at least begins) a subclinical immunization which may or may not be reinforced by subsequent exposures. While this study suggested that the frequency of the occurrence of the disease was not a function of facility of contact, it gave no indication as to whether immunization or disease is the result of some quality of the virus in respect to dose or virulence, or of some quality of resistance on the part of the host.

In a second study[2] evidence was presented which indicated that the extent of the distribution of the virus is the same in warmer climates, where the incidence of the clinical disease is less, as in cooler climates where the disease has its greatest incidence. An analysis of variation in incidence together with seasonal fluctuation in cooler and warmer climates indicated that the climatic variations could hardly be attributed to corresponding variations in the facility with which the virus is transmitted or in its virulence. It was shown, however, that the seasonal fluctuation, as well as the incidence, correlated with the extent of seasonal fluctuations in climate but not with climate itself. In other words, poliomyelitis is a disease of warmer seasons

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but of cooler climates, its incidence, generally speaking, varying directly with the magnitude of the change from the colder to the warmer season of the year, but not with the warmth of the climate itself.

It is known that numerous bodily functions undergo corresponding seasonal fluctuations doubtless in the nature of normal adjustments of the body to a varying environment. It was pointed out that some of these seasonal fluctuations in physiologic functions bear a direct relation to the occurrence of certain noninfectious diseases; to susceptibility to certain chemical compounds, the toxicity of which depends on the rate at which they are broken down in the body into their poisonous constituents, and which rate varies with the seasonal activity of certain bodily functions; and that susceptibility to certain infections is influenced by physiologic factors which undoubtedly undergo similar seasonal variations.

This study led to the opinion that resistance to poliomyelitis in the first instance may lie in a balance between some as yet undetermined physiologic processes which undergo seasonal fluctuations and vary in different climates by way of adjustment of the body to different or changing environment. Failure of the seasonal adjustment would result in a deficiency or discrepancy which would be greater in cooler climates where fluctuation in the seasons is greatest, and least in warmer climates where fluctuation is least. It was pointed out that certain observations regarding the type of individual in whom the disease seems to occur with greater frequency than in the average, a type suggesting some physiologic imbalance, tended to support this hypothesis.

In order to define more clearly this kind of resistance to disease as a matter of convenience; to distinguish it from immunity (i.e., resistance which arises as a reaction product between the infecting agent and the host) as well as to call attention more sharply to a form of resistance which since the advent of the germ theory of disease has, I believe, been neglected; I have suggested that it be designated by the term "autarcesis," which may be defined as the power to resist infection which resides within the individual's normal physiologic functions and which does not require the provocation of the infectious agent for its production.

From this conception of the epidemiology of poliomyelitis some idea of the nature of the virus reservoir of the disease can be formed which is in accord with other observations which need not be enumerated here. Furthermore, from an analysis of some of the general features of the disease some notion may be gained of the probable behavior of this virus reservoir which cannot be ascertained with the limited facilities available for the actual detection of the virus. This question has long been before students. Where does the virus maintain itself in areas where, and at times when, the disease is highly prevalent, in interepidemic periods, in areas where the incidence of the disease gives no hint of its presence other than the occurrence of sporadic cases, or more or less widely spaced outbreaks, and in the winter and spring when its presence is indicated only by occasional cases?

I have previously pointed out that we need not look for any condition peculiar to any one area which would serve as a virus reservoir. This is obvious from the simple fact that no part of the inhabited world is entirely free of the disease, and is further emphasized by the fact that regardless of the incidence its age distribution indicates an equally widespread immunization and, hence, an equally widespread distribution of the virus in all parts of the world from which figures are available.*

Since the widespread process of immunization which has been referred to apparently takes place with a rapidity corresponding to concentration of population, as is the case with such well known contact diseases as measles and diphtheria, it seems reasonably certain that the mode of dissemination of the virus of poliomyelitis is likewise by direct person to person contact. No evidence has been found that the virus reservoir may lie largely in chronic carriers. No instances are known where one individual seems to have been responsible for the transmission of the virus to any number of others over a considerable length of time. It would rather appear that the virus reservoir is in reality the upper respiratory passage of large numbers of individuals who, so far as the evidence indicates, harbor the virus for a relatively short time during which by far the largest proportion acquire an immunity without showing obvious signs of the disease. It is still a question as to what proportion of individuals may show mild symptoms following an infestation, known as abortive poliomyelitis, or what proportion of them become immunized without any noticeable reaction to the virus.

There is no positive indication that the virus of poliomyelitis can actually be transmitted with greater facility at one season than another. In fact, we now see that increased opportunities for contact due to closer aggregation of the population in winter, long supposed to be responsible for the seasonal prevalence of the common contact diseases, can hardly be the true explanation. If this were true one

* There is only one probable instance of the introduction of the virus into a virgin population. On the island of Nauru, in 1910, the disease reached a very high incidence and apparently occurred with equal frequency at all ages.

seasonal curve should be common to all these diseases; and the seasonal curve of none of the contact diseases has been shown to coincide with variations in the aggregation of population. For example, the seasonal increase in diphtheria long supposed to follow the opening of schools, actually is on the upward trend before the opening of schools. Finally, it remains to be shown that the magnitude of seasonal variation in "opportunities for contact" even begins to approach the seasonal variations in the prevalence of these diseases.

In view of previous studies mentioned, which suggest an autarcetic factor which varies with season, one can at least raise the question whether an initial infestation with the virus of the disease produces the clinical disease or an immunity, largely according to the season of the year when infestation takes place. In this connection it may be stated that while mild cases are not infrequently detected, solely by reason of their proximity to frank cases, we have not found evidence that any considerable number of such mild febrile attacks, which might be regarded as abortive poliomyelitis, occur as an undercurrent of summer outbreaks of the frank disease. However, the evidence in regard to the age distribution indicates that the large majority of the population acquires an immunity in the course of a life time. This would imply that infestation with the virus is correspondingly widespread, from which it may be inferred that, on the average, approximately 1 out of 50 of the population receives an initial exposure each year, or 1 out of 600 of the population in any given month.

In the hypothetical case of a city of 100,000, shown in Figure I, approximately 166 initial exposures would occur each month. It might well be supposed that the virus of poliomyelitis might be spreading with equal rapidity throughout the year, but that during the winter and spring months by far the vast majority of initial exposures would produce no symptoms, or some mild reaction not recognized as poliomyelitis; while as the summer season arrived, on account of a general diminution in autarcesis, a larger and larger proportion of infestations would result in attacks of the recognizable disease rather than in a mild reaction which would pass unnoticed.

In the hypothetical case illustrated, it will be seen that an epidemic of an intensity which is seldom exceeded (2 cases per 1,000 population) could occur in the summer season without any actual increase in the rate of dissemination of the virus. It is not to be understood that this moving virus reservoir is necessarily present in every locality all the while. Local epidemics may follow its introduction or presence during the season of anautarcesis (summer and fall), or if a particular locality has been visited by the moving reservoir during the season of autarcesis (winter and spring) a wave of immunization would result which would render that community relatively free of the disease for a time. Of course many communities may pass through

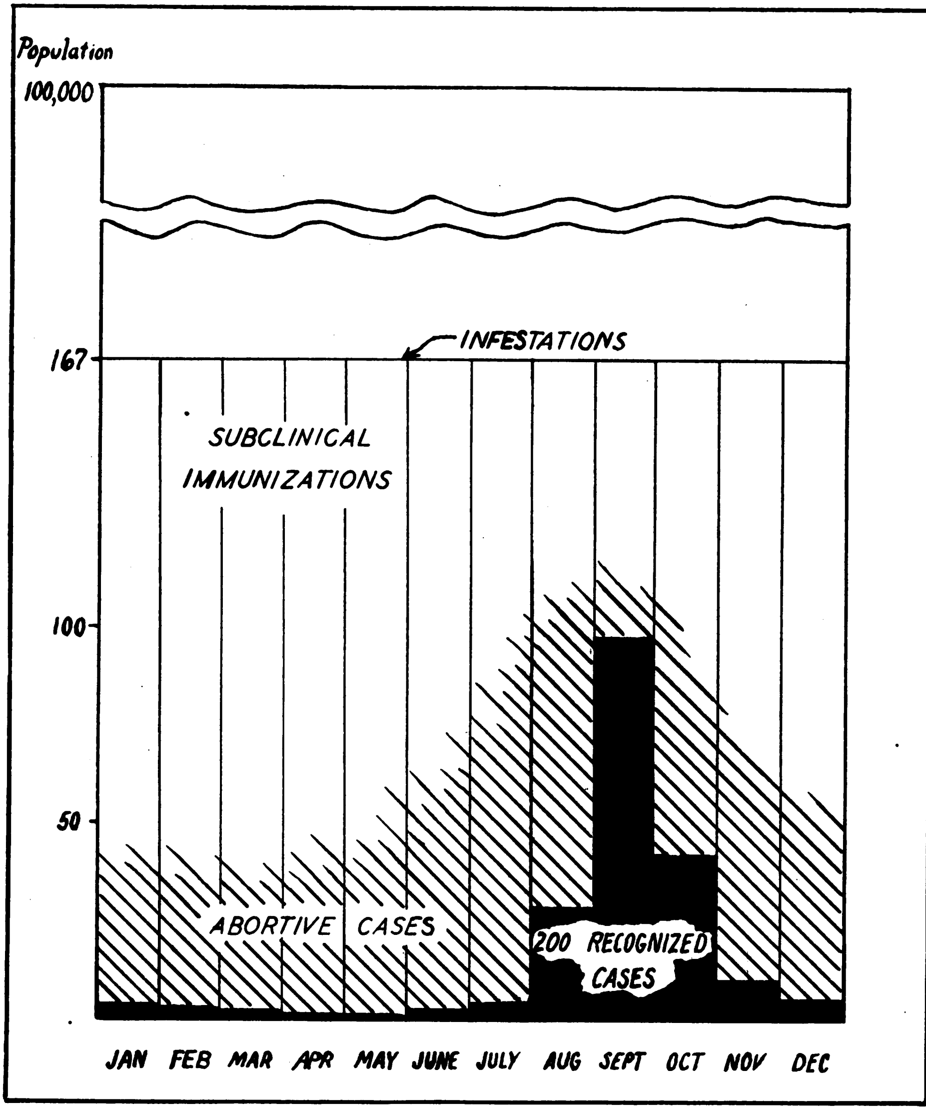

FIGURE I—This represents a hypothetical community of 100,000, in which initial infestations with the virus of poliomyelitis take place with an even frequency throughout the year, and in sufficient numbers to "saturate" the population in the course of an average lifetime of 50 years. ($1/50 \times 100{,}000 \times 1/12 = 167$ infestations per month.) This implied rate of dissemination is sufficiently rapid to bring about a practically universal immunization.

The solid area on the chart shows the occurrence of a severe epidemic (2 cases per 1,000 population) of recognizable cases which could occur without any increase in the rate of dissemination of the virus. The remainder of the supposedly uniform number of infestations represents, in unknown proportions, subclinical immunizations or abortive attacks.

the summer season when the virus reservoir happens not to be present and remain free of an outbreak, but at the same time there would be an accumulation in the number of non-immunes.

I wish it understood that the conception of the virus reservoir of poliomyelitis which has been outlined is only a probability, but a probability which is in accord with numerous observations at the bedside, on the statistical chart, and in the experimental laboratory, none of which alone would be conclusive, but all together begin to take on a real significance.

This conception of the epidemiology of the disease is presented with the idea that if its validity be supported by further observations, future studies looking to its control could proceed along more clear-cut avenues of approach rather than by chance and laborious exploration of innumerable possibilities. Thus, if this idea of the virus reservoir is correct, there does not appear to be any great promise in attempts to curtail the spread of the virus. From the experimental point of view our present knowledge indicates that artificial immunization, if accomplished, is likely to be by methods too tedious and uncertain for practical application. These studies, therefore, suggest that we should turn from the pursuit of the bacteriology (if this term may be applied to a virus disease) and immunology of the disease to an exploration of its autarcesiology in our search for means of prevention.

REFERENCES

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2. Aycock, W. Lloyd. A Study of the Significance of Geographic and Seasonal Variations in the Incidence of Poliomyelitis, *J. Prev. Med.*, 3, May, 1929.

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Periodic Medical Examination for Bus Drivers

THE sudden illness of a motor omnibus driver led to an accident which only fortunate chance prevented from having fatal results. The possible consequences of such accidents are so serious that the London *Daily Mail* advocates precautions against them. The example of the railways, where drivers are subjected to a searching medical test on appointment, and annually thereafter, is an admirable one. Something of the kind is already done by the London General Omnibus Company, which at least examines its new drivers and insists on a fresh test before a man who has been seriously ill returns to work. But not all the smaller proprietors are quite so careful. The *Daily Mail* advocates a systematic medical test, repeated at regular intervals, for every driver of a public conveyance. Our contemporary might have gone further and advocated the regular medical examination of all drivers of automobiles, public or private.—*Diplomate,* Sept., 1929.