

The populations concerned, and the numbers of positive cases, are so small that very wide variations would have to be recorded before the results from an analysis such as the above could be accepted as significant. A statistician would not, in fact, be impressed by any of the differences in incidence shown in these tables, except possibly by some of those in Table V. It is nevertheless interesting to note that the incidences recorded are rather higher, in every case except one, in the group of schools with the best accommodation than in those which were worst. The only valid conclusion from this analysis is, however, that it provides no evidence that differences in standards of sanitary accommodation (and, by inference, overcrowding) have any influence on the incidence of poliomyelitis in schools.

XII. DISCUSSION

This report has traced the course of the epidemic to a crucial point. The incidence as a whole has touched, or almost touched, the 1-per-1,000 mark. If my calculations are correct, one age group—that of boys aged 10 to 15—has just reached saturation.

What happens now?

The answer will be known by the time the report is published, but early in May the writer was rash enough to state publicly that "he would be surprised if, a month from now, the epidemic in the Auckland district was not virtually at an end."

My previous study focused attention on the older schoolboy. It was he who was most liable to introduce infection into the home. He tended to react to it himself at an early stage, getting the disease over quickly in its milder form. By the time the first positive case came to light in the household, half the boys of this age had already had a "suspect illness" and were immune from further trouble. To a lesser extent the schoolgirl aged 5 to 10 years old was similarly implicated.

Some may think that the conclusion that practically every boy of this age in Auckland has either been in hospital with poliomyelitis or has had a "suspect illness" during the present epidemic is absurd. Most of them must have been in Auckland during the last epidemic. Surely many must have gained immunity then?

The answer is: Very probably; if not then, certainly at some time during the intervening years. Many of these "suspect illnesses" must in reality be minor reactions in persons who have had a previous attack—which, of course, will usually have been a minor one too. These suspect illnesses are usually very mild, although recognizable when the observer is on the lookout for them and knows there is poliomyelitis about. Details will be found in my previous paper.

Consider the case of another virus disease, vaccinia. Nowadays for purposes of international certification no such thing as a negative reaction is recognized. A person who has never been vaccinated before normally reacts violently: the result is called a "typical primary vaccinia." Revaccination of one who has been successfully vaccinated before usually results in no more than redness and swelling along the scratch, the so-called "reaction of immunity." Revaccination after a very long interval sometimes produces an accelerated reaction, similar to primary vaccinia, but seldom so upsetting.

It seems to me that in poliomyelitis we meet with equivalents of all these types of reaction, but with a different emphasis. In vaccinia the virus is one whose virulence, though attenuated, is not allowed to fall below a certain level. In poliomyelitis it would appear that the normal condition of the virus, except during an epidemic, is a state of low virulence. Most people when attacked react in a manner akin to the "redness and swelling" of the reaction of