VI. PREVIOUS EPIDEMICS AND "SILENT" INTERVALS

It is difficult from the records now available to arrive at strictly comparable figures, but the following is an estimate of the incidence in the Central Auckland District during the three previous major epidemics:—

1916	15·5 per 10,000
1925	7·8 per 10,000
1937	2·6 per 10,000

Between these years the disease was constantly active in New Zealand. In the Auckland Province during the eleven years intervening between epidemics of 1925 and 1937 there were 90 cases, an average of 8 per annum. Following on the 1937 outbreak, however, there was a significant lull. Details are lacking for the first three years, but here is the record for the Auckland urban area and Otahuhu from 1941 on:—

1941	Nil
1942	1 case
1943	Nil
1944	Nil
1945	1 case
1946	5 cases (plus 1 just outside)

The lull ended with a crash, the new epidemic commencing earlier in the season than ever before and producing more cases in a month than the last epidemic cast up in its full course. The incidence to date, 9·8 per 10,000, is sufficient to put it in a different class from the outbreaks of 1925 and 1937. If we consider the successively declining toll of the first three epidemics and the continuing activity between them, it becomes clear that the 1937 affair was merely the final movement of a composition which began in 1916, and that in 1947 we were in the presence of a new opus.

It would be interesting to know what was really happening during the lull, which can hardly have been a period of complete inactivity. Figure IV shows the location of the positive cases which came to light after 1940. They were pretty well dispersed. The two most densely, and the three most sparsely, populated of the districts shown in Fig. III produced no cases; the middle group had 1 each, except for Auckland City, which had 3. The 3 cases recorded in the early part of 1947, before the epidemic began, were also widely separated; similarly with the 6 occurring in 1946.

If our theory is correct, all this time a furtive but beneficent process was going on, in which an attenuated virus was passing from person to person and silently conferring immunity. This process, it would seem, was most effective in the more densely populated areas—in Newmarket, Mount Eden, and Mount Albert, for example. This could only be the case, however, so long as the number of spreaders, and perhaps the dispersive powers of the virus, remained low. Once they increased beyond a certain point, the differences between areas must be reduced to insignificance.

The results, if the virus increased in virulence as well as in dispersiveness, must now be very different. Those who had previously been visited might respond with a minor illness, or not at all. In areas where there had previously been little circulation, however, the path of the new invasion would be strewn with positive cases. These alone find their way into hospital, and appear in the statistical returns; of the minor reactions we normally hear nothing.