Precise records being available for the 1937 epidemic (but not, unfortunately, for previous ones) it has been possible to plot incidence against density for the 43 cases which occurred in the areas dealt with in Fig. III. In Fig. V the result is compared with the position in the present epidemic. It will be seen that in the four Auckland areas affected, incidence in 1937 was related directly to density, the opposite of our experience now. This diagram also distinguishes cases with paralysis or paresis from those without either, in case an altered standard of diagnosis should have obscured the issue. This does not help very much, however, the effect in the case of the present epidemic being complete confusion.

There are three possible explanations of this anomaly:—

(*a*) The apparent inverse relationship shown in the latest epidemic may be false. Such a relationship has been noted elsewhere,* however, and the results are too consistent to be lightly dismissed. If anything, the 1937 diagram is less likely to be reliable in view of the small number of cases involved.

(*b*) The very small proportion of non-paralytic cases in the 1937 record suggests that the picture may be incomplete. Present standards of hospitalization might have told a different story.

(*c*) It has been suggested above that the 1937 outbreak was not in reality an epidemic complete in itself, but merely a last flare-up in a process which began much earlier. The tail end of an epidemic might well be irregular. As I have already pointed out, when the spreading process is rapid the effect of variations of density must be slight or nil. If most of those who escape in the closing phases of an epidemic are persons who have gained immunity during the earlier stages, one would expect to find most signs of activity in the regions least affected when the process was at its height. Imagine a shower of sparks continually falling on heaps of materials, some of which are much less inflammable than others: at the end only the heaps which burn least readily will be alight.

The last seems the most likely explanation.

VII. SPREAD IN THE AUCKLAND URBAN AREA

In my previous paper spot maps were presented showing that in the early stages of the epidemic there was nothing to indicate spread from any particular focus. The next four diagrams (Figs. VI–IX) show the further course of the epidemic in the urban area.† Cases in residential institutions have not been included.

A certain ebb and flow over the area is apparent, but no tendency to centrifugal spread. The pattern of the last 50 cases is not unlike the first. The story is one of victims struck down at random almost simultaneously, and a battle fought to a finish without shifting ground. When poliomyelitis comes, it comes not single spies, but in battalions; not uniformed troops, but fifth-columnists, quietly infiltrating; then suddenly, here and there, the guise of a minor illness is thrown off, and paralysis is in our midst.

* In London in 1947. Sir Allen Daley and B. Benjamin (1948): *The Medical Officer, 80*, 171.

† Figure VI corresponds to Fig. I (I) in the previous paper. Some minor corrections have been necessary.