Report on Work Done under Grant from the National Foundation for Infantile Paralysis. (June 30, 1940 - Dec. 31, 1940)

The projected work on the distribution and elimination of virus in human poliomyelitis is now practically completed and the enclosed paper which was read at the 42nd General Meeting of The Society of American Bacteriologists on December 27, 1940, at St. Louis, Mo. contains a summary of the essential results. The work will be written up in detail and submitted for publication within a few months.

The results of the first experiments on Problem III, i.e. - Centrifugal Spread and Elimination of Virus in Experimental Poliomyelitis (Rhesus Monkeys) - were included in the June, 30 report. These experiments have now been repeated on additional monkeys, and the results thus far are the same although at least two more weeks are required for the final completion of the work.

Incidental Findings during the Course of the Above Investigations. -

A. The June, 30 report mentioned certain tests which indicated that Vitamin D deficiency was not a necessary prerequisite for successful infection of rhesus monkeys with poliomyelitis virus via a peripheral nerve such as the sciatic. Repetition of these experiments on additional animals has confirmed this conclusion.

B. Early during the course of these investigations it was found that rhesus monkeys inoculated with human virus may have a nonparalytic or even inapparent form of poliomyelitis with typical neuronal and infiltrative lesions in the spinal cord. In at least one instance of this inapparent form of the disease typical paralytic poliomyelitis was produced on passage. Histologically, the difference between the nonparalytic, transitory paralytic, and persistent paralytic types of the disease appeared to depend upon the number of nerve cells which were
visibly affected by the virus, the largest number of completely necrotic cells being found in the persistent paralytic type of poliomyelitis. These observations are of value not only in the proper evaluation of attempted transmission of human virus to monkeys, but also in the consideration of the basis of inapparent, nonparalytic, and transitory paralytic types of human poliomyelitis. Hitherto the concept of "abortive" poliomyelitis in monkeys rested on such inconclusive findings as fever and pleocytosis without any histological or biological evidence that these changes were brought about by poliomyelitis virus. With regard to the nonparalytic and inapparent types of the human, disease, it had been suggested that they occur because the virus fails to reach the medulla or spinal cord. The present observations, however, show that the virus may reach the lower motor areas without paralysis necessarily ensuing, provided the proper equilibrium between the host and the virus exists or is achieved before too many nerve cells are affected.

These observations have also led to the consideration of assigning a new role to heavy exercise in the development of paralysis. The history of heavy exercise (playing ball, swimming, hiking, etc.) is very frequently given by patients with paralytic poliomyelitis, and a limited personal inquiry has revealed that the interval between this exercise and the onset of paralysis is usually less than 24 hours. This short interval suggests not only that those individuals were already harboring the virus in their nervous system, but also that it might already have involved their medulla and spinal cord and that the exercise could be the factor which converts what might have remained an inapparent or nonparalytic type of poliomyelitis into the frankly paralytic type of the disease.
C. Inoculation of Human Virus into Mice with Subsequent Tests
for Immunity to Armstrong’s Virus. - The spinal cord and medulla
of each case of poliomyelitis which was inoculated into monkeys was simultaneously
injected intracerebrally and intraabdominally into 6 to 10 mice. This was done
not only to rule out the various types of encephalitis viruses but also to de-
termine whether or not poliomyelitis virus while not producing any apparent
disease in mice might perhaps produce immunity to Armstrong’s poliomyelitis
virus which is pathogenic for mice. In no instance did these mice develop any
illness nor was there any demonstrable immunity when they were tested with
Armstrong’s virus approximately two months after the original inoculation with
human virus which was pathogenic for rhesus monkeys.