HAEMATOGENOUS SPREAD OF POLIOMYELITIS VIRUS

by

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It is almost generally accepted that, when poliomyelitis virus spreads through a population, it causes subclinical and clinically unrecognizable abortive infections in the majority of the cases. Only a relatively small proportion of the infected individuals contract paralytic disease. The abortive cases which may show various signs of minor illness, i.e. sore throat, gastrointestinal disturbances, muscle pain ("summer grippe": SABIN and STEIGMAN (9)), and the non-paralytic meningitic forms, are essentially extraneurial forms of poliomyelitis because the virus has been recovered from them.

At the Third European Poliomyelitis Conference, held in 1950 at Amsterdam and Leiden, MOLLARET (8) insisted on the myotropic properties of poliomyelitis virus. In his view, many patients treated in the artificial respirator do not die from respiratory paralysis, but from myocarditis. As a matter of fact, myocardial lesions have not infrequently been found in poliomyelitis patients at autopsy. The occurrence of severe pain in certain muscle groups, e.g. those of the neck and the back, might also point to myositis (LARUELLE (7)).

Since JUNGBLUT and coworkers (5, 6) succeeded in isolating poliomyelitis virus from the skeletal as well as from the heart muscle of fatal cases, and JUNGBLUT (3) and VERLINDE and his associates (10, 12, 13) produced myocardial and skeletal muscle lesions in monkeys with members of the Columbia SK group of viruses as well as with classical poliomyelitis virus, a muscular

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involvement in poliomyelitis seems to be in the realms of possibility.

The extraneural distribution of poliomyelitis virus which occurs essentially during the initial phase of poliomyelitis infection, and which may result in minor illness, myositis, myocarditis, and meningitis, is difficult to explain by exclusive neural spread of the virus. One of the possible routes of non-nervous spread would be via circulation. If viraemia occurs, one would expect it to occur long before the paralytic stage, presumably in the initial phase, or, even during the early incubation period. Although viraemia during the first four days of the incubation period has been demonstrated in monkeys inoculated intramuscularly with members of the Columbia SK group of viruses (10, 13), we could not show a haematogenous spread in a small-scale experiment with the A.K. strain of poliomyelitis virus. The reason for this failure was perhaps the relatively low virulence of the strain, which might be diluted beyond the minimal PD₅₀ dose in the blood stream. Therefore, we have made similar attempts with the Leon strain of poliomyelitis virus ¹), which brings down monkeys regularly upon intramuscular inoculation. Practically 100% of monkeys inoculated by this route with 1 ml of a dilution of 10⁻¹ to 10⁻² of monkey cord come down with paralysis, showing typical anterior horn lesions.

EXPERIMENTAL.

A group of 8 rhesus monkeys was inoculated into the thigh or calf muscles with 1 ml of a 10% suspension of monkey cord having a PD₅₀ titre between 10⁻⁶ and 10⁻⁷. All of these animals were bled from the heart at daily intervals, and 6 animals were killed at different days following inoculation. The blood was collected in sodium citrate, centrifuged for 30 minutes at 3000 r.p.m., and the plasma was inoculated both intracerebrally (1 ml) and intramuscularly (10 to 40 ml) into rhesus monkeys. The sedimented blood cells were haemolyzed by adding sterile distilled water, and then centrifuged for 1½ hour at 40,000 r.p.m. in an ultracentrifuge (Spinco). The supernatant fluid was carefully removed, leaving 1 ml at the bottom of the tube, which was then inoculated intracerebrally into rhesus monkeys. Moreover, 10% suspensions, in distilled water, were prepared from the inoculated muscle, the corresponding sciatic nerve, and the inguinal lymphnodes. The latter

¹) The Leon strain was kindly supplied by Dr H. A. Howe, Baltimore Md. U.S.A.