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Production of Experimental Poliomyelitis from Untreated Stools.*

HOWARD A. HOWE AND DAVID BODIAN. (Introduced by P. H. Long.)

From the Department of Anatomy, Johns Hopkins Medical School.

It is now a well established fact that poliomyelitis virus may be present in considerable quantities in the stools of patients suffering from either paralytic or abortive attacks of the disease. These findings have been summarized by Trask, Vignec and Paul¹ who recently described a method for treating human stools in order to render them suitable for intraperitoneal inoculation into monkeys. Such procedures unfortunately involve the use of bactericidal substances of which the effect upon poliomyelitis virus is unknown. It is thus desirable to obtain a method of utilizing the material from stools without the intervention of measures which may cause attenuation or other change in virus. Such a method has been found in the simple intranasal inoculation of monkeys with an untreated stool suspension. On September 15, 1938, a stool specimen was obtained from a 3-year-old quadriplegic child through the courtesy of Dr. Harold Hobart of the Children's Hospital, Washington, D. C. The stool was collected on the third day after the onset of paralysis. At the time the child was still febrile. The specimen was immediately made up into a thick suspension with distilled water and the supernatant fluid was placed in the ice box. Two Rhesus monkeys were inoculated intranasally with this fluid on 3 successive days, each animal receiving a total of 3 cc per nostril. Following the introduction of the material the nasal passages were rubbed gently with a pipe cleaner.

On the fourteenth and eighteenth days respectively the animals developed typical but not extensive paralyzes. They were killed and portions of the cord were removed for histological study and reinoculation. Typical poliomyelitic lesions were found in the grey matter of each spinal cord. Cultures of a suspension of each cord on blood agar and in blood broth were negative except for a *B. subtilis* contamination in one sample. Three mice were inoculated intracerebrally with 0.02 cc of 20% cord suspension from each

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¹ Trask, J. D., Vignec, A. J., and Paul, J. R., *J. Am. Med. Assn.*, 1938, **111**, 6.

case: all remained well although 2 monkeys inoculated intracerebrally and intraperitoneally with the same material developed typical poliomyelitis. In neither was the paralysis extensive although the cord lesions were characteristic.

It is thus possible to produce typical poliomyelitis in monkeys by the simple intranasal inoculation of untreated human stool. The method appears to be reasonably sensitive and should be applicable to the demonstration of small quantities of virus to which bactericidal measures might prove disastrous. It also makes it possible to work directly with poliomyelitis virus as it probably exists in transmission from one individual to another.

Although virus is present in human stools even in abortive cases,¹ various investigators² have been unable to demonstrate it in the feces of the macaque unless it was previously fed in large quantities. The direct intranasal inoculation of untreated monkey stool has likewise failed to reveal traces of virus in the intestinal contents at the height of paralysis. Three animals were inoculated intranasally on 4 successive days with 1 cc per nostril of an emulsion of stool taken from 2 animals which were prostrate after intracerebral introduction of MV virus. They showed no clinical signs of poliomyelitis although typical paralysis developed in controls receiving intracerebral inoculations of nerve tissue from the same cases.

The failure of virus to migrate from CNS to intestinal lumen in the macaque is consistent with the uniformly unsuccessful attempts² to infect this animal by the gastro-intestinal route. We have also been unable to produce poliomyelitis by means of MV virus enemas in 4 macaques which had previously been given a bloody diarrhoea by an 80% alcohol enema. These findings indicate quite conclusively that the Rhesus monkey is not readily susceptible to MV virus by way of the gastro-intestinal portal.

¹ Clark, P. F., Roberts, D. J., and Preston, W. S., Jr., *J. Prev. Med.*, 1932, **6**, 47; Leiner, C., and von Weisner, R., *Wiener klin. Wochensch.*, 1910, **23**, 817; Levaditi, C., and Landsteiner, K., *Compt. Rend. Soc. de Biol.*, 1910, **68**, 311.