

# Pathogenesis of Poliomyelitis

## Reappraisal in the Light of New Data

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The questions of special current interest in the pathogenesis of poliomyelitis are concerned with (i) the origin of virus in the alimentary tract, (ii) the source and significance of viremia, (iii) the mode of invasion of the central nervous system, and (iv) the factors which determine invasion and variations in the extent of involvement of the central nervous system. Concepts of the pathogenesis of poliomyelitis have varied and changed over the years with each new advance in our knowledge of the nature and behavior of the causative agent. Soon after the discovery of the virus, poliomyelitis was compared to meningococcic meningitis—the virus was pictured as propagating in the nasopharynx and occasionally invading the central nervous system across the hematoencephalic barrier. When experimental work indicated that polio as well as certain other neurotropic viruses had the capacity of spreading along insulated neural pathways, poliomyelitis was pictured as an infection in which the virus multiplied in the nose and spread to the central nervous system along the olfactory pathway. The experiments with the old MV strain of polio virus, which we now recognize as a strict neurotropic variant selected by repeated intracerebral passages in monkeys, and on which this concept was chiefly based, are as valid today as they ever were, but the inferences with respect to the natural disease became untenable when olfactory blockade in human beings failed to prevent poliomyelitis (1) and when the ol-

factory pathways were not found to be involved in the human disease (2, 3). In 1944, after a study of the localization of virus in human beings and in cynomolgus monkeys infected by the oral route with recently isolated virus, I proposed the following course of events as a working hypothesis of the pathogenesis of poliomyelitis: the virus entered by way of the mouth, localized, and multiplied in various levels of the alimentary tract with incidental invasion of the lymph nodes, blood, and viscera and with occasional invasion of the central nervous system along neural pathways connected with the peripheral sites of viral multiplication (4).

When it was subsequently found that virus was readily detected in the blood of monkeys, chimpanzees, and human beings in the early stages of infection (5) and that amounts of antibody, which were incapable of protecting monkeys from paralytic infection via the olfactory neural pathways, were protective in monkeys that received virus by the oral or intramuscular routes (6), Bodian interpreted the available data as indicating that the virus in the blood passed into the central nervous system across certain limited (presumably modified) vascular zones, but because of the character of the distribution of lesions in the central nervous system, he still found it necessary to assume that from the point of penetration further spread within the central nervous system occurred along specific neural pathways. When Bodian found virus in pieces of intestine containing Peyer's patches but not in adjacent pieces of ileum, he proposed that polio virus multiplies initially only in the tonsils and Peyer's patches. From these lymphatic collections, the virus was pictured as

moving outward onto the surface of the alimentary tract and inward into the blood, which distributed it to other susceptible "target organs" such as lymphatic structures, "brown fat," and the central nervous system (7).

This interpretation of the existing data has come to be regarded as the theoretical basis for explaining the ability of relatively small amounts of antibody, either passively introduced or actively induced by killed virus vaccine, to prevent invasion of the central nervous system and the resulting paralysis. If this view is correct, the effect of the presence of antibody prior to infection should be all or none with regard to involvement of the central nervous system—that is to say that, if invasion is not prevented, there should be no modifying effect on the extent of involvement leading to nonparalytic or milder paralytic forms of the disease. The observations of Francis and his associates (8), however, suggested that the incidence of nonparalytic infections of the central nervous system due to polio virus was not strikingly affected during the 1954 field trials of the Salk vaccine, and there were also indications that in general the paralytic disease was milder when it was not prevented in vaccinated individuals. Hammon and his associates (9) had previously also suggested a modifying effect of antibody when paralysis was not prevented during the field trial of gamma globulin.

Faber (10) brought forth evidence of early invasion of the regional nerve ganglia after feeding polio virus to cynomolgus monkeys, and he has maintained that polio viruses multiply only in nerve cells in monkeys and human beings. According to this view, the ingested virus is absorbed by the nerve endings of the buccopharyngeal and lower alimentary mucosa, multiplies in the neurons of the regional ganglia, and from there moves centrifugally back into the mouth and throat and sometimes also centripetally into the central nervous system. Viremia, according to Faber, is the result of the continued absorption of this excreted virus into the lymphatics of the alimentary tract with an ultimate overflow into the blood. Having demonstrated that direct inoculation of thousands of infective doses of virus into the vertebral artery produced specific lesions in the spinal cord, Faber assumes that virus which is absorbed into the blood from the alimen-

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