EPIDEMIOLOGY OF POLIOMYELITIS AND RECENT ADVANCEMENTS IN LABORATORY DIAGNOSIS*

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Acute anterior poliomyelitis has possibly been more widely studied from the standpoint of epidemiology than any other disease.

There is evidence that it is an ancient disease. On an Egyptian plaque which dates from the thirteenth century before Christ is shown a man with a crutch and a withered limb (Stele of Ruma in the Ny Carlsberg Glypotek in Copenhagen).[1] However, the first epidemic was reported in 1891. It is thought that during the nineteenth century poliomyelitis gradually changed from an endemic to an epidemic disease.

At the first international poliomyelitis conference in 1949, Sabin reviewed the epidemiologic patterns of poliomyelitis in different parts of the world during the past 40 years.[2] This data indicated to him that:

(1) The risk of acquiring paralysis from poliomyelitis virus is not the same for all population groups in the same region or country, and it varies markedly for people living in different parts of the world.

(2) The epidemiologic picture as regards occurrence of epidemics, level of endemic attack rates, and age selection patterns is continually changing and is not the same in all countries with advanced sanitation and hygiene. As an example, the epidemic pattern noted in Swedish cities from 1911 to 1931 did not appear in New York City until 1931. The highest incidence of poliomyelitis occurs most frequently in the six to ten year age group now, rather than in the zero to five year age group as it did during the early epidemics.

(3) The urban and rural incidence of the disease may be almost identical in some localities and markedly different in others, suggesting that factors unrelated to density of population may be responsible for high rates encountered in sparsely populated regions.

(4) The incidence of the disease was found to be different in some racial groups but not in others, and economic status did not account for these differences.

(5) Although the total amount of poliomyelitis increases when epidemics begin to occur in an area with a previously low endemic rate, the available evidence indicates that the disappearance of the infantile age selection pattern, that is, the occurrence of a high percentage of cases in age group zero to five years, does not necessarily lead to a greater total incidence of the paralytic disease.

(6) The data reviewed points to the necessity of investigating factors unrelated to specific virus immunity but peculiar to certain regions, populations, and age groups, which may determine whether the incidence of paralysis associated with poliomyelitis infection shall be high or low.

During the three years since the first International Poliomyelitis Conference, numerous scientific advancements have been made which give promise to helping solve some of the problems. These include the classification of the strains of poliomyelitis virus into three immunologic types (Brunhilde, type I ; Lansing, type II; Leon, type III), the propagation of the viruses in tissue culture, and the development of complement fixation tests.

The Committee on Typing of the National Foundation for Infantile Paralysis in 1951 presented the results of a well organized, systematic study of a series of 100 selected strains of poliomyelitis virus.[3] Of these, 85 were type I (Brunhilde), 12 were type II (Lansing), and three were type III (Leon).

The propagation of poliomyelitis virus in tissue culture provides criteria by which the presence of the virus can be recognized in vitro and hence may afford a basis of technique of isolating virus from patients or animals, for the screening of chemotherapeutic and antibiotic substances. Within the past year complement fixation antigens for the three immunologic types of poliomyelitis virus have been developed.[4] However, more experimental work must be done before the tests can be used as a critical diagnostic or epidemiologic tool.

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Recently, viremia has been reported in the experimental animal when fed the virus by mouth, the viremia occurring three to seven days before onset of paralysis.[5] The virus had not previously been found in the blood stream.

Koprowski, Jervis and Norton fed 20 volunteers a living Lansing-like strain of virus adapted to cotton rats and characterized by low pathogenicity for monkeys.[6] None showed clinical evidence of the disease; some had virus in their stools; all non-immune volunteers showed a prompt rise in serum antibody. These results indicate that a satisfactory vaccine giving active immunity may soon be practical.

Turner and his colleagues showed the presence of neutralizing antibodies to the Lansing virus in 72 per cent of infants under three months of age.[7] By the age of one year the proportion had declined to ten per cent. It then gradually rose until after 15 years of age 89 per cent gave positive tests. The titers on most of the children became positive during the summer.

Further proof that neutralizing antibodies for poliomyelitis occur in the blood of a large percentage of the population was obtained when the Red Cross Gamma Globulin was evaluated as a prophylactic agent for poliomyelitis.[8] In this carefully conducted study, 54,772 children received injections ; half received Gamma Globulin, the other half received Gelatin. During the period from the beginning of the second week after the injection through the fifth week only six cases of poliomyelitis occurred among the children who had the immune Globulin, while 38 cases occurred among those getting Gelatin.

Several interesting observations have been made in areas where native populations have mixed with outsiders. A study of poliomyelitis occurring in the American troops during the years 1943 to 1948 showed that the rate at which the soldiers contracted poliomyelitis was highest among those in the Far East and the Philippine Islands.[9] The incidence of paralytic poliomyelitis among the natives in these areas is low and it usually occurs in the zero to five year age group. During the war the chance of an American soldier getting paralytic poliomyelitis was ten times greater in the Middle East than in the United States.[10]

A study of the poliomyelitis epidemics in South Africa showed paralysis to be ten times more frequent in the Europeans than in the native Bantu Tribe. Among the Bantu most cases occurred in infants under five years of age, while in the Europeans the incidence in the six to ten year age group was as great as in the zero to five age group. The natives were found to have a greater amount of gamma globulin than the Europeans.[11] This might account for their greater immunity.

Faelber in South Africa did virus studies at frequent intervals on the stools of 29 infants during their first year of life.[12] Of the 29 infants, he was able to follow only 16 for the full year. Of these 16, four were proven to have the virus in the stool and subsequently developed antibodies. None developed paralytic poliomyelitis.

There are a number of other recent observations made in the laboratory and by the epidemiologist that deserve mentioning.

Bauer, Chaney and co-workers noted that a lowered serum albumin and an increased excretion of nitrogen and potassium in the urine during an attack of poliomyelitis was indicative of a poor prognosis.[13]

The Coxsackie group of viruses which have frequently been found in association with poliomyelitis virus and have a similar distribution in nature have been classified into 15 immunologic types.[14] They are immunologically distinct and their tissue affinities are different from poliomyelitis virus. They have been found in a wide variety of clinical syndromes, such as aseptic meningitis, encephalitis, epidemic pleurodynia, influenza, summer grippe, herpangina, appendicitis, sinusitis, and fever of unexplained origin.

Finally, I would like to call attention to certain factors that undoubtedly have some influence in determining whether or not the poliomyelitis patient develops paralysis. These are : exercise of a severe or prolonged nature during the period of invasion, operations on the mucous membranes of the mouth and throat which precede the infection by four weeks or less, inoculations during the same period, and pregnancy.

In summary, (1) poliomyelitis is undergoing a constantly changing epidemic pattern ; (2) there are three immunologic types (Brunhilde, type I ; Lansing, type II ; Leon, type III ; (3) a vaccine giving active immunity may soon be practical ; (4) passive immunity can be obtained satisfactorily with Gamma Globulin ; and (5) with the rapid advancement of laboratory techniques, clarification of the epidemiology of the disease would be expected within a few years.

ADDENDUM

The publication by Salk and others[15] indicates that a safe vaccine giving active immunity against all three types of poliomyelitis virus has been developed for experimental work. It appears that it is now only a matter of time before a practical vaccine for general use will be available.

Dr. Richard E. Dukes served as a Pediatrician at Carle Clinic from 1946-1980.

REFERENCES

1. Gear, J. G. S. : Immunity to poliomyelitis. Ann. Int. Med., 37 : 1-22, (July) 1952.
2. Sabin, A. B. : Epidemological patterns of poliomyelitis in different parts of the world (papers and discussion, First International Poliomyelitis Conference). Ed. I, Philadelphia, J. B. Lippincott Company, 1949, Vol. I, p. 1-33.
3. The Committee on Typing of the National Foundation for Infantile Paralysis : Immuniologic classification of poliomyelitis viruses. Am. J. Hyg., 54 : 191-274, (Sept.) 1951.
4. Svedmyr, A. ; Enders, J. F. ; and Holloway, A. : Poliomyelitis viruses propagated in tissue cultures as antigens in the complement fixation test. Federation Proc., 11 : 483, (Mar.) 1952 ; ibid, Proc. Soc. Exper. Biol. & Med., 79 : 296-300, (Feb.) 1952.
5. Horstmann, D. M. : Poliomyelitis virus in blood of orally infected monkeys and chimpanzees. Proc. Soc. Exper. Biol. & Med., 79 : 417-419, (Mar.) 1952 ; ibid, Federation Proc., 11 : 471, (Mar.) 1952.
6. Koprowski, H. ; Jervis, G. A. ; and Norton, T. W. : Immune responses in human volunteers upon oral administration of a rodent-adapted strain of poliomyelitis virus. Am. J. Hyg., 55 : 108-125, (Jan.) 1952.
7. Turner, T. B. ; Hollander, D. H. ; Buchley, S. ; Pentti Kokko, W. ' and Winsor, C. P. : Age incidence and seasonal development of neutralizing antibodies to Lansing poliomyelitis virus. Am. J. Hyg., 52 : 323-347, (Nov.) 1950.
8. Hammon, W. McD. ; Coriell, L. L. ; Wevile, P. F. ; Klint, C. R. ; and Stokes. Joseph, Jr. : Evaluation of Red Cross gamma globulin as a prophylactic agent for poliomyelitis. J.A.M.A., 150 : 757-760, (Oct. 25), 1952.
9. Paul, J. R. : Poliomyelitis attack rates in American troops, 1940-1948. Am. J. Hyg., 50 : 57-62, (July), 1949.
10. Paul, J. R. ; Havens, W. P., Jr. ; and Van Rooyen, C. El : Poliomyelitis in British and American troops in the Middle East ; isolation of virus from human feces. Brit. Med. J., 1 : 841-843, (June24) 1944.
11. Anderson, G., and Bersohn, I. : Cited by Bear, J. H. S.[1]
12. Grea, J. H. S. ; Mearsroch, V. ; Bradley, J. ; and Faerber, G. I. : Poliomyelitis in South Africa. Studies in an urban native township during a non-epidemic year. South African Med. J., 25 : 297-301, (May 5) 1951.
13. Bower, A. G. ; Morgan, F. M. ; and Chaney, G. L. : Nitrogen and potassium metabolism : the reaction pattern in poliomyelitis. Am. J. Med. Sc., 223 : 532, (May) 1952.
14. Contreras, G. ; Barnett, V. H. ; and Melnick, J. L. : Cited by Ward, R. A. : Some recent advances in poliomyelitis research. Am. J. Phys. Med., 31 : 206-214, (Aug.) 1952.
15. Salk, J. E. ; Bennett, B. L. ; Lewis, L. J. ; Ward, E. N. ; and Younger, J. S. : Studies in human subjects on active immunization against poliomyelitis. J.A.M.A., 151 : 1081-1098, (Mar. 28) 1953.

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