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Alterations in Autarceologic Susceptibility to Experimental Poliomyelitis.*

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Dissemination of the virus of poliomyelitis is widespread, but the paralytic disease is limited; exposure tends to be uniform, while the occurrence of the frank disease is variable; infection tends to be general in the population, paralysis is selective.

The varying frequency with which paralytic poliomyelitis results from exposure to the virus indicates that autarceologic factors in the host largely determine the occurrence of paralysis. There are epidemiologic indications that autarcesis varies with climate and season, in different individuals in the same locality, and in the same individual. Other observations suggest that autarceologic susceptibility is dependent upon some inherent endocrine fault, which operates through its effect on a barrier to the virus (upper respiratory mucous membrane) or through some change in cellular affinities for the virus. That the former may be the case in the monkey and the latter in man is suggested by the fact that animals failing to develop the frank disease following repeated intranasal instillations of virus do not acquire subclinical immunity; whereas, there is epidemiologic evidence that this is the common occurrence in man.

Observations suggestive of the endocrine fault involved are the occurrence of the disease in certain types of individuals indicative of endocrine differences, as described by Draper; the familial occurrence of poliomyelitis¹; its occurrence following tonsillectomy²; and the frequency of the disease during pregnancy.³ Perhaps the single observation of a case in a castrate male is likewise suggestive in this connection.

In a discussion⁴ of the rôle of autarcesis in the epidemiology of poliomyelitis, it was suggested that some knowledge might be gained of the mechanism of this form of resistance by testing the influence

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¹ Aycock, W. L., *West Va. Med. J.*, 1934, **30**, 11.

² Aycock, W. L., and Luther, E. H., *New Eng. J. Med.*, 1929, **200**, 164.

³ Aycock, W. L., *Trans. Sec. Ped., Am. Med. Assn.*, 1932.

⁴ Aycock, W. L., *J. Prev. Med.*, 1926, **3**, 245.

of appropriate endocrine alterations upon the outcome of intranasal instillations of virus in the monkey.

More recent developments in the field of endocrinology—thinning of the vaginal mucosa by castration and recornification following administration of estrin to castrate animals,⁵ the finding that the changes in the vaginal mucosa during pregnancy are of similar endocrine origin,⁶ together with evidence that the nasal mucosa participates in such changes⁷—are consistent with a number of epidemiologic and clinical indications concerning the mechanism of susceptibility to poliomyelitis, and suggest that castration would increase the susceptibility of monkeys to intranasal instillation of the virus, while the administration of estrin would, on the other hand, increase resistance.

Ten immature female monkeys were castrated. After 24 days, 5 of them were given daily for a period of 12 days an intramuscular injection of 2000 International units of estrin† in oil. Thirty-two days after castration, all 10 animals received in each nostril, daily for 6 days, 3 drops of a 5% suspension of poliomyelitis virus. Changes in the nasal mucosa have not yet been verified. The procedures, although designed to give maximal effects, are not known to be optimal.

Four of the 5 castrated animals not treated with estrin developed poliomyelitis; while only one of the 5 which had received subsequent injections of estrin succumbed. Three of 5 normal control monkeys developed the disease, a figure which is in keeping with our average experience with animals similarly inoculated. While in either case the numerical difference in the experimental as compared with the normal animals could hardly be considered significant, the fact that the experiment was double in nature—designed on the one hand to increase resistance, and on the other to increase susceptibility—affords the difference in the outcome in the 2 instances more significance than would be indicated by the actual numerical variation from the normal in either case.

⁵ Allen, Edgar, *Contr. to Embryo*, Carnegie Inst., Washington, 1927, **19**, 1.

⁶ Davis, M. E., and Hartman, C. G., *J. Am. Med. Assn.*, 1935, **104**, 279; Hisaw, F. L., *Anat. Rec.*, 1935, **64**, 54.

⁷ Pighini, G., and Porta, C. F., *Valsalva*, 1934, **10**, 140.

† Amniotin kindly supplied by E. R. Squibb and Sons.