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Mechanism of Zinc Sulphate Prophylaxis in Experimental Poliomyelitis.

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We have previously reported[1] that the application of 1% zinc sulphate to the olfactory mucosa of monkeys affords protection against poliomyelitis virus inoculated intranasally one or more months later. The basis of this protection is not yet understood.

[1] Schultz, E. S., and Gebhardt, L. P., *J. Am. Med. Assn.*, 1937, **108**, 2182.

Nasal mucous membranes removed a week or more after treatment generally present a normal appearance. Recently, however, we have studied membranes removed several hours to several days after treatment with varying concentrations of zinc sulphate solution.

With a little experience it is possible to remove olfactory mucosa, olfactory nerve filaments and olfactory bulbs, of both sides, all in one piece. These have been embedded and sectioned tangentially. Such sections afford the orientation necessary for a satisfactory study of the olfactory mucosa and related structures and within limits allow comparison of treated and untreated sides.

Our observations show that by the end of the first day, there may be a marked acute inflammation, associated with some desquamation and necrosis of epithelial cells. Following treatment with 1.5% zinc sulphate solution, this change may be so marked that much of the olfactory mucosa loses its epithelium. The inflammatory reaction tends to subside quickly and is followed promptly by regeneration. By the end of the 2nd day denuded areas may be again covered with an epithelium consisting of flat or cuboid cells with many cells showing mitosis. By the end of the 3rd, 4th or 5th day the epithelial surface again appears normal, except for some disorder in the arrangement of the cells and for a few scattered collections of PMN cells.

It is generally held that destroyed nerve cells are not restored. We are, however, not aware of any observations proving that this holds also for olfactory cells. Should the protection induced in monkeys by zinc sulphate be the result of a destruction of olfactory cells and not the result merely of an injury or chemical change in the dendrites of the olfactory neurons, it would then seem to follow that these structures are in reality regenerated in some way. This statement is justified in part on the ground that the majority of treated resistant monkeys become susceptible again in 2 to 4 months. Either the protection is not associated with a complete destruction of olfactory cells or certain cells in the olfactory area are differentiated into cells capable of forming axons and dendrites and of eventually restoring function. Such a provision does not seem entirely unlikely in a structure which is probably not altogether free of the risk of naturally induced injuries.

During the past summer, zinc sulphate solution was applied to the olfactory mucosa of several thousand persons. A thorough application is said to always produce an anosmia. In children, this disappears within 2 weeks. In adults, however, the sense of smell

may not return for several months. This, together with the fact that adults frequently suffer severe pain immediately following treatment, suggests that these differences may be explained on an anatomic basis. In adults, the superior meatus is narrower than in children,[2] it may indeed be narrow and deep enough to contribute materially to capillary attraction. This would result in the solution being held in contact with the mucosa for a longer period of time and thus add not only to the discomfort, but also to the duration of chemical action on the cells. The question of the possible risk of inducing permanent anosmia by such more prolonged and uncontrolled drug action will not be discussed here; nevertheless, it is of considerable theoretical interest that a few cases in adults are known to us in whom the sense of smell has been completely, or nearly completely regained after an anosmia lasting 2 to 4 months. This is in harmony with our observation that treated monkeys generally become susceptible again to intranasal inoculation of virus within 2 to 4 months after treatment. This raises the question, what is the mechanism underlying these late restorations of function in man and of susceptibility to infection in monkeys?

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Listerella Monocytogenes: A Cause of Meningo-Encephalitis in Man.

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In a preliminary note[1] we reported the isolation from a human case of meningo-encephalitis of a new organism which we identified with the genus Corynebacterium. We wish at this tme to report additional observations and to correct the classification of the organism.

The clinical history of the case is described in the preliminary note and has been more fully presented by Marcellus, Crouch and

[2] Shahinian, L., Bacher, J. A., McNaught, R. C., and Newell, R. R., *J. Am. Med. Assn.*, 1938, **110**, 1254.

[1] Schultz, E. W., Terry, M. C., Brice, A. T., and Gebhardt, L. P., PROC. SOC. EXP. BIOL. AND MED., 1934, **31**, 1021.