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Poliomyelitis Virus and Degeneration of Peripheral Nerves.*

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After intracerebral injection of poliomyelitic virus in *M. rhesus* monkeys, O'Leary, Heinbecker and Bishop[1] showed that motor paralysis preceded noticeable changes in the axones of somatic nerves coming from the segments, the nerves of which supplied the paralyzed muscles. In brief, destruction of nerve cells occurred first, and destruction of nerve fibers secondarily. Since paralysis is produced in from 5 to 8 days following the intracerebral injection of the Flexner strain of virus in *M. rhesus* monkeys, at least that same period of time and perhaps more would elapse before changes would become obvious in the fibers of the somatic nerves, after this method of injection.

Forty-two monkeys were injected subserosally with 25 cc. of a 1% suspension of virus, 10 animals with the same amount and percentage of virus contained in enteric toxin (paratypho-coli filtrate)[2] subserosally and 7 animals with 0.1 cc. of a 1% suspension of virus intracerebrally. Somatic nerves coming from the lumbar and sacral segments were removed and studied by polarized light between crossed Nicol's prisms.[3] Within 24 hours after the injection, the somatic nerves were found to have obvious changes in their fibers, *i. e.,* extensive vacuolization and destruction of fasciculi fibers. In 48 hours, the myelin might be granular, the nodes swollen with spireme-like isotropic threads above and below the cementing discs, and the neurokeratin framework might be prominent around the axis cylinder. The nerve fibers of animals that received virus in enteric toxin were always involved and almost completely destroyed in some sections within 48 hours. The animals that received virus intracerebrally did not show comparable changes,

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1 O'Leary, J. L., Heinbecker, P., and Bishop, G., *Arch. Neur. and Psych.*, 1932, **28**, 272.

2 Toomey, John A., PROC. SOC. EXP. BIOL. AND MED., 1934, **31**, 1015.

3 Sutton, T. S., Setterfield, H. E., and Krauss, W. E., Bulletin 545, Ohio Agr. Exp. Station, December, 1934.

even though 10 or more days had elapsed between the time of inoculation and the time the animals were sacrificed.

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Some Properties of Castle's Intrinsic Factor.

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It has long been known that the activity of pepsin is destroyed by exposure to alkali. Accordingly, fresh normal human gastric juice, collected after histamine injection, was at once brought to pH 10 with NaOH and let stand at room temperature for 30 minutes, after which it was brought to pH 6.0 with HCl. No peptic activity was detected by the hemoglobin method.[1] Using Reimann's technique,[2] 20 cc. of the juice was mixed with 20 cc. of raw liver hash, incubated at 37°C. and pH 2.5 for 2 hours, then adjusted to pH 5.0, and mixed with boiled tomato juice and fed daily for 7 days to a pernicious anemia patient with red cells at 1,300,000. The reticulocytes rose from 2.8% to a peak of 22.8% on the eighth day, the red cells reaching 2,100,000.

As pepsinogen is not destroyed under the conditions used, a preparation kindly supplied by Dr. John H. Northrop and Dr. R. M. Herriott with a specific activity of 2.3[3] and containing 6 mg. total nitrogen per cc. was tested, 4.5 cc. being injected intramuscularly in a case of pernicious anemia, the red cells being at 1,800,000. There was no rise in the reticulocytes nor red cells for the next 10 days, though the patient subsequently had a reticulocyte peak of 29.9% following the oral administration of one dose of 100 gm. boiled liver paste mixed with 100 cc. of normal gastric juice prepared as described for the first case. Boiled liver was used to rule out the effect of hepatic cellular enzymes.

Greenspon's paper[4] then appeared, suggesting that chilled alkalinized gastric juice was effective in pernicious anemia when administered orally 4 hours before meals in doses of 250 cc. daily.

[1] Anson, M. L., and Mirsky, A. E., *J. Gen. Physiol.*, 1932, **16**, 59.

[2] Reimann, F., and Weil, R., *Z. f. klin. Med.*, 1934, **126**, 568.

[3] Holter, H., and Northrop, J. H., PROC. SOC. EXP. BIOL. AND MED., 1935, **33**, 73.

[4] Greenspon, E. A., *J. A. M. A.*, 1936, **106**, 266.