trophy at the end of this longer period, both being increased in weight approximately 100%. On injecting these animals with androtin, the secondary sex glands commence to regenerate. No hypertrophy of the adrenals was observed in 4 of the animals which were injected with androtin for the last 21 days of an 85-day period, and in the other 6, the hypertrophy was so slight as to be questionable. In this group of castrated animals which had an adequate amount of hormone to maintain the prostate and to reduce the hypertrophy of the adrenals, the pituitary hypertrophy was not altered during a period of 3 weeks.

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Spread of Poliomyelitis Virus from the Gastrointestinal Tract.*

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Flexner and Lewis[1] first showed that poliomyelitis could be transmitted by intranasal instillation, the spread of the virus being along the olfactory nerve to the brain. The olfactory nerve, however, is not the only unmedullated one situated in the nasal area, for it is precisely here under the mucosa that the 1500 cells of nervus terminalis are placed,[2] composing a network of cells and fibers as might be found in the myenteric or submucous intestinal sympathetic.[2, 3] The position of the plexus of the nervus terminal near the embryological juncture of the stomodeum and the possible upper tip of the mesenteron, *i. e.*, the upper end of the foregut of the endodermal tube is significant, since the other portion of the alimentary tract, the hind gut, also possesses the same kind of unmedullated nerve fiber plexuses. If the virus has a facultative or almost an obligate affinity for grey fibers, this area should also provide an easy portal of entry. Perhaps failure to produce the disease after the virus of poliomyelitis had been introduced into the gastrointestinal tract was because the virus never approximated the grey fibers.

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[1] Flexner, S., and Lewis, P. A., *J. Am. Med. Assn.*, 1910, **54**, 1140.

[2] Brookover, C., *J. Comp. Neur.*, 1917, **28**, 2.

[3] Huber, C. G., and Guild, S. R., *Anat. Rec.*, 1913, **7**, 253.

In other experiments where the abdomen of a monkey or a rabbit had been opened and the intestine watched after the introduction of the virus through a tube or needle, it was observed that the material was usually swept out of the small intestine into the colon. This was followed by a period of spastic hyperactivity in the small intestines. No approximation could have occurred. The following technic was then devised. The abdominal cavity of a completely anesthetized *Macacus rhesus* monkey was exposed through a midline incision. The small intestine was clamped by a pair of intestinal clamps a few inches above the ileocecal valve. Another pair clamped the intestines about ½ to over one foot above the lower instrument; 50 to 100 cc. of a 1% emulsion of potent poliomyelitis virus were introduced into the isolated portion of the intestinal canal through a 24 gauge needle until the gut was ballooned out. It was very important to introduce enough virus so that the intestines would be tensely dilated. The clamps were kept on until the pinch reflex had disappeared. The intestines were returned to the abdominal cavity and the incision sewed tightly and bandaged. Two animals who were tested in this manner developed typical poliomyelitis.

One might contend that the animal might have regurgitated some virus and thus have infected the olfactory area even though the injection had been made into the small intestine. Since the postganglionic fibers of the thoracolumbar sympathetic system are unmyelinated, at least to the large abdominal plexuses, and those of the intestinal blood vessel unmyelinated as far as the vertebral ganglia themselves, there was no reason why the disease should not have been produced by a subserosal injection of the virus. Such a procedure would absolutely rule out the possible contamination of the olfactory area. Accordingly, the abdomen was opened in the manner previously described and 10 to 20 cc. of a 1% suspension of potent poliomyelitis virus was injected subserosally at multiple points. The needle could be seen distinctly as it was inserted beneath the outer intestinal coat, and when the injection was started, a whitish, crescentic, dimpled lesion resulted which crackled as it extended. After a few minutes, the lesion became deep red or wine colored. What little leaking occurred, went outside and not into the gastrointestinal tract. The abdomen was then sewed tightly and bandaged. Two monkeys were injected in this manner and both developed poliomyelitis.