animals showed the presence of a few corpora lutea, lutein cysts, and developing graafian follicles. The injection of a known dosage of the extract to a group of 5 rats over a period of 5 days produced an average increase in ovarian weight of 63%, while the administration of *the same total dosage* over a period of 10 days to 5 littermates gave an average increase of 90%. It has been shown previously that these 2 findings are characteristic of the gonad-stimulating hormone found in pregnant women and not of anterior pituitary extracts (Fluhmann[2, 3]).

Administration of the untreated urine failed to induce any demonstrable changes in the atrophied ovaries of 2 hypophysectomized rats, but a concentrated extract induced an extensive development of the theca and interstitial tissue in 4 other instances. It has been shown by Noguchi,[4] Collip *et al.*,[5] and Smith and Leonard[6] that this is a characteristic of the gonad-stimulating hormone of pregnant women.

Conclusion. The gonad-stimulating hormone found in the urine of a patient with a chorioepithelioma uteri had certain biologic characteristics similar to those of the substance occurring in women during normal gestation.

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Accelerated Production of Poliomyelitis.*

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When I injected poliomyelitis virus in *Macacus rhesus* monkeys either intrasciatically, intracerebrally or directly into the cord in the region of the first lumbar segment, periods of 4, 7 and 2½ to 3 days, respectively, elapsed before paralysis came on. Such latent inter-

[2] Fluhmann, C. F., *Endocrinology*, 1933, **17**, 550.

[3] Fluhmann, C. F., PROC. SOC. EXP. BIOL. AND MED., 1933, **30**, 1014.

[4] Noguchi, K., *Jap. J. Med. Sc. Pharmacol.*, 1931, **5**, 104. Quoted by Collip *et al.*[5]

[5] Collip, J. B., Selye, H., and Thomson, D. L., *Nature*, 1933, **131**, 56.

[6] Smith, P. E., and Leonard, S. L., PROC. SOC. EXP. BIOL. AND MED., 1933, **30**, 1248.

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vals are considered the incubation periods of the virus. It is curious that the time elapsing before paralysis appeared was shorter the nearer the injection was made to the lumbar area. Unlike the virus of smallpox, for instance, which multiplies *in situ,* the virus of poliomyelitis spreads away from the cortex and down the cord,[1] practically disappearing from the site of the injection in the cerebrum. It does not cause a great amount of functional damage in the animal until a few days after it reaches the lower part of the cord. In its manner of progress, it acts like a catalyst, a catalyst with nearly an obligate affinity for grey fibers or for axis cylinders of medullated nerves.

In previous experiments, I tried to recover the virus from the stools of humans but failed. However, the stools obtained from monkeys, before and after paralysis, showed a difference in that the feces obtained at the time of paralysis contained a toxic factor that was not present before the onset of paralysis when the animals were in normal condition.[2] The appearance of increased toxicity in the stools of these monkeys coincided somewhat with a marked drop in the agglutinin titer value of their blood serums withdrawn during the stages of paralysis against various enteric organisms.[3]

Because of these findings, my attention was directed to the colon organisms. Could the destruction of nerve cells be due to the effect produced by a combination of poliomyelitis virus and enteric toxin? Such combinations are not new in medicine as Shope[4] has shown in swine influenza and as has been pointed out in Oroya fever.[5] Perhaps the virus needed only the addition of enteric toxins to produce the disease sooner in monkeys. The following experiments were done to test this latter point.

Twenty-three strains of enteric organisms were planted in 0.5% glucose broth, the toxin harvested 10 days later[3] and brought up to a pH of from 7.6 to 7.8.

Nine animals were divided into sets of 3. Each one was injected intracerebrally as noted in Table I. The injecting needle was held in place for a few minutes in order to prevent seepage. All operations were done under complete ether anesthesia. The injection and results are noted in Table I.

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1 Fairbrother, R. W., and Hurst, E. E., *J. Path. and Bact.,* 1933, **17**, 30.

2 Toomey, J. A., and vonOettingen, W. F., PROC. SOC. EXP. BIOL. AND MED., 1933, **30**, 1082; *Am. J. Dis. Child.,* in press.

3 Toomey, J. A., *J. Inf. Dis.,* 1934, **54**, 74.

4 Shope, R. E., *J. Exp. Med.,* 1931, **54**, 373.

5 Oroya Fever, *J. Am. Med. Assn.,* 1933, **100**, 191.

TABLE I.
Intracerebral Injections.

	Positive Control	Negative Control	Virus Toxin Injection
Set I	1.25 cc. 20% Virus plus 1.25 cc. Normal Saline	1.5 cc. Enteric Toxin plus 1.5 cc. Normal Saline	1.25 cc. Enteric Toxin plus 1.25 cc. 20% Virus
Monkey and Result 6 lb. Animals	Animal A—paresis on the 7th day and quadriplegia on the 9th day.	Animal B—no reaction.	Animal C—hemiparesis in 24 hours, paralysis the 3rd day and quadriplegia the 5th day.
Set II	1.5 cc. 10% Virus plus 1.5 cc. Normal Saline	1.5 cc. Enteric Toxin plus 1.5 cc. Normal Saline	1.5 cc. Enteric Toxin plus 1.5 cc. 10% Virus
Monkey and Result 6 lb. Animals	Animal D—paresis the 9th day and paralysis the 11th day. Animal lived.	Animal E had no reaction.	Animal F—hemiparesis in 24 hours, paralysis the 5th day and quadriplegia the 6th day. Animal died.
Set III	1 cc. 10% Virus plus 1 cc. Normal Saline	1 cc. Enteric Toxin plus 1 cc. Normal Saline	1 cc. Enteric Toxin plus 1 cc. 10% Virus
Monkey and Result 4½ lb. Animals	Animal G—paresis the 8th day, paralysis the 10th day, quadriplegia the 13th day.	Animal H had no reaction.	Animal I—hemiparesis in 24 hours, paresis of the right side and left foot the 5th day and quadriplegia the 7th day.

After abdominal section, combinations of enteric toxin and virus which were introduced into the small intestine in an area isolated between 2 intestinal clamps produced the disease quicker than when only virus suspensions were used. Enteric toxin injections alone had no effect.

After abdominal section, combinations of enteric toxin and virus which were injected into the subserosa of the small intestine (25 cc. of 2% virus and 25 cc. of enteric toxin) produced the disease more quickly than when only virus suspensions were used (50 cc. of 1% virus). Enteric toxin injections alone had no effect (25 cc. of enteric toxin and 25 cc. of saline).

The enteric toxin, obtained after growing the ordinary colon bacilli found in the gastrointestinal tract for 10 days in glucose broth, when combined with poliomyelitis virus, accelerated the production of poliomyelitis in the *Macacus rhesus* monkey.