the anterior hypophyseal hormone known to us. We have employed bovine and rat implants and folliculin-free and growth hormone-free extracts of the ripe human placenta and of the urine of pregnant women. The implant method shows such an effect with only 2 daily treatments and sacrifice on the fourth day. In some cases hyperplasia was seen to have begun before lutein structures were present in the young ovaries. The secretion of corpora lutea therefore could not have been responsible for this stimulus to mammary growth, though the growth was always most marked in cases in which corpora lutea were coincident with a hypertrophied follicular apparatus.

4416

Hyperplasia of Mammary Apparatus of Adult Virginal Females Induced by Anterior Hypophyseal Hormones.

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The mammary tree of adult virginal rats invariably undergoes some hyperplasia after a month of daily treatment with alkaline extracts of the anterior hypophysis, solutions very rich in growth hormone and almost completely devoid of the hormone which stimulates the ovaries of immature rodents to precocious development. By more chronic treatment with such an extract this can be brought to true milk secretion. Mammary hyperplasia takes place with more speed after treatment with the anterior hypophyseal ovary-stimulating hormone (bovine or rat hypophysis implant, or extracts of placenta and of the urine of pregnant women). The hyperplasia is, in fact, remarkable after only 10 days dosage with the latter methods.

4417

Effects of Thyroparathyroidectomy on the Teeth of the Rat.

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The effects observed by Erdheim[1] and later by Toyofuku[2] in the teeth of parathyroidectomized white rats led Erdheim to emphasize

[1] Erdheim, J., *Frankf. Z. Path.*, 1911, vii, 175.

[2] Toyofuku, T., *Ibid.*, 1911, vii, 249.

the relation of the parathyroids to calcium metabolism. The uncertainty as to the mode of action of the parathyroids caused us to undertake a similar study of the teeth of some thyroparathyroidectomized rats which had been produced for another purpose.

The two rats whose teeth were studied were males and had been kept, like the others, on a standard diet of 60% cracked corn, 20% powdered milk, 16% powdered casein, 3% alfalfa meal, 0.5% sodium chloride, and 0.5% calcium carbonate. Both showed typical tetany after the operation, and both showed gross dental defects before death. Rat 1 had a brown line along the anterolateral edge of the right upper incisor when it died on the 29th day; rat 2 had suffered a spontaneous fracture of both lower incisors and marked elongation of the opposing upper incisors before it died on the 82nd day. Microphotographs, with detailed descriptions, of the transverse sections of the teeth of these rats will be published elsewhere.[3] The following is a brief summary of the findings:

RAT 1. Pulp and odontoblasts well preserved, dentine well calcified. The greatest disturbance occurred in the enamel epithelium and in the bone adjacent to one of the teeth. The ameloblasts were disorganized in places, and there was proliferation of the outer layer of the enamel epithelium. These layers showed some folding. At one level a striking exostosis of the alveolar bone had taken place. This abnormal bone growth obliterated the peridental membrane as it progressed inwards towards the tooth, and it finally lay in contact with the enamel itself. In so doing it either pushed aside or induced atrophy of the enamel epithelium in its path.

RAT 2. Conditions varied greatly from level to level, but were abnormal throughout. Pulp showed some congestion, vacuolation of the intercellular substance, various degrees of atrophy of the odontoblasts, and sporadic calcareous nodules. Capillary canals running from the pulp into dentine were abnormally numerous. Dentine was in the main under-calcified, and showed a zone of fracture; but there were zones of over-calcification also. In general the dentine was normal in contour and thickness. The enamel was most irregular. In places it was very poorly calcified, leaving a conspicuous organic matrix in the sections. In other places it disappeared, so that the space which it ought to have occupied was filled with peridental tissue and with disorganized masses of enamel epithelium in various stages of proliferation, disarray, and atrophy. Marked folding occurred at some levels. The alveolar bone showed exostoses, disorganizations, atrophied bone cells, and irregular calcifi-

[3] Skillen, W. G., and Jung, F. T., *Am. Dent. J.*, 1929.

cations (*i. e.*, both under and over-calcification, as judged from the staining).

These findings do not differ essentially from those reported by Erdheim and Toyofuku (who removed only parathyroids), but do contradict a statement by Gies.[4] An interpretation of the data will be attempted in our final paper.

4418

Ultrafiltration Studies on the Virus of Poliomyelitis.*

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Ultrafiltration studies on so-called filterable viruses, including fowl-plague, vaccinia, herpes, foot-and-mouth disease and mosaic diseases of plants, have been reported by Andriewsky,[1] Doerr and Pick,[2] Levaditi and Nicolau,[3] Duggar and Karrer-Armstrong,[4] Levaditi, Nicolau and Galloway,[5] Olitsky and Boëz,[6] Zinsser and Tang,[7] Berger,[8] but to our knowledge no such investigations have thus far been reported on the virus of poliomyelitis. While the filterability of this virus through Berkefeld candles has been reported (Landsteiner and Levaditi,[9] Flexner and Lewis[10]) it is still not known whether the etiological agent is a small bacterium or a true ultrascopic virus. With this in mind we have undertaken a program of investigation designed to elicit more accurate information as to the magnitude of the organism concerned. The present paper deals with some of our observations thus far.

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[4] Gies, W. J., and collaborators, *J. Natl. Dent. Assn.*, 1918, v, 527.

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[1] Andriewsky, P., *Centralbl. f. Bakteriol.*, Abt. I., Orig., 1915, lxxv, 90.

[2] Doerr, R., and Pick, R., *Ibid.*, 1915, lxxvi, 476.

[3] Levaditi, C., and Nicolau, S., *Compt. rend. Acad. des Sci.*, 1923, clxxvi, 717.

[4] Duggar, B. M., and Karrer-Armstrong, J., *Ann. Missouri Bot. Gard.*, 1923, x, 191.

[5] Levaditi, C., Nicolau, S., and Galloway, I. A., *Compt. rend. Acad. Sci.*, 1926, clxxxii, 247.

[6] Olitsky, P. K., and Boëz, L., *J. Exp. Med.*, 1927, xlv, 685.

[7] Zinsser, H., and Tang, E. F., *Ibid.*, 1927, xlvi, 357.

[8] Berger, E., *Z. Hyg.*, 1928, cviii, 315.

[9] Landsteiner and Levaditi, *Compt. rend. Soc. Biol.*, 1909, lxvii, 592.

[10] Flexner, S., and Lewis, P. A., *J. Am. Med. Assn.*, 1909, liii, 2095.