

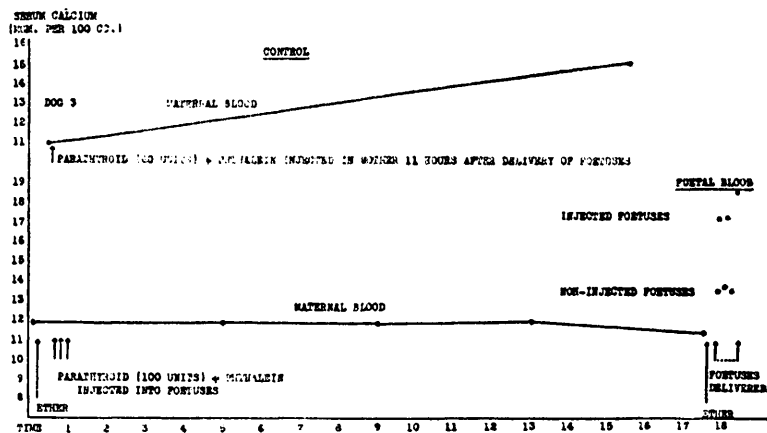


FIG. 1.

findings in the case of parathyroid extract are in agreement with previous observations which have been made upon the effect on the mother of the injection of insulin, adrenalin, and pituitrin in rabbit and guinea pig fetuses, *in utero*. No evidence has been obtained that these endocrine substances pass from foetus to mother across the placental boundary.⁵

The parathyroid extract (Collip) or "Para-thor-mone" used in the experiments was donated by Eli Lilly & Co.

¹ Williams, J. W., *Text-book of Obstetrics*, 1924, 196.

² Seitz, L., *Handbuch der normalen und pathologischen Physiologie*, 1926, xiv, 485.

³ Fischer-Wasels, B., and Berberich, J., *Hirsch's Handbuch der Inneren Sekretion*, 1927, i, 432.

⁴ Collip, J. B., *The Parathyroid Glands, Medicine*, 1926, v, 1.

⁵ Snyder, F. F., and Hoskins, F. M., *Anat. Rec.*, 1927, xxxv, 23.

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Cephalically Situated Respiratory Mechanism in the Brain Stem of the Cat.

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The origin and course of the reticulospinal tracts in the cat, recently described by Papez¹ suggested the possibility of their being respiratory tracts. It was thought that this problem could best be ap-

proached by localizing the respiratory mechanism in this region of the brain stem. The material reported in this paper summarizes the results obtained thus far. The animals were given 1 gm. of urethane per kilo of body weight. Both carotids were tied at the time of cannulation of the trachea. Ether was used during surgical procedure. As the animal came from under ether, hyperpnea usually resulted spontaneously.² Hyperpnea was hastened by heating the preparation, or as in most cases, merely by stimulating the central sciatic stump with moderately strong induction shocks. This "rapid respiration mechanism" could be "shot" in the following ways: 1—exposing the corpora quadrigemina; 2—sectioning the brain stem through, or caudal to, the superior colliculi. However, the superior colliculi can be exposed without interfering at all with the mechanism. It was also found that a complete section of the brain stem cephalic to the corpora quadrigemina did not alter the respiratory rate.³

A further study of this mechanism has been made possible by a method which exposes the floor of the fourth ventricle without interfering with respiration. By this method it has been found that a longitudinal lesion passing ventral to the corpora quadrigemina and lateral to the mid-line will *slow* respiration. But, heating the animal after this effect is produced, will *increase* the rate of respiration. It was noted, however, that this latter increase was not nearly as marked as the increase in respiration which was caused by heating the animal previous to placing the lesion.

The fact that the mechanism can be partially "shot" by lesions affecting one side of the stem only, is demonstrated very forcibly by placing a transverse lesion on one side involving the lateral region of the stem. Such a lesion can be placed at any level between the inferior colliculi and the facial nuclei. After such a lesion, the respiratory rate is slowed. Each respiration is deeper and both the expiratory and inspiratory phases are lengthened. However, between the latter respiratory acts, short rapid excursions are superimposed upon the inspiratory phase of the larger and slower excursions. A corresponding lesion on the opposite side eliminates the short rapid excursions entirely. The respiratory mechanism in an animal having two such lesions is so affected as to cause no increase in respiration when the animal is heated. Extensive lesions, transverse or longitudinal, through the medial reticular formation at any place within the above levels do not interfere with the respiratory rate.

The above results suggest, in general, a center which is con-

cerned with the production of rapid respiration located somewhere in the region of the inferior colliculi, possibly in the cephalic pons.⁴ The tracts concerned, probably afferent,⁵ pass through the medulla and pons in a lateral position.

¹ Papez, J. W., *J. Comp. Neur.*, 1926, xli, 365.

² Henderson, Yandell, *Am. J. Physiol.*, 1910, xxv, 310.

³ Trevan, J. W., *J. Physiol.*, 1916, l, 43.

⁴ Lumsden, T., *J. Physiol.*, 1923, lvii, 153.

⁵ Coombs and Pike, *Am. J. Physiol.*, 1918, xlv, 569.

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Experimental Production of Gastric Ulcer.

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My interest in the experimental production of gastric ulcer was aroused by the finding of two well defined ulcers in the stomach of a cat which had received injections of histamine for another investigation. The cat is a suitable animal to use for the experimental production of gastric ulcers. It exists on a mixed diet comparable to that of man and develops ulcers fairly readily. The above observation led me to examine carefully the stomachs of all cats treated by histamine. Of 4 cats examined 1 showed a spontaneous ulcer on the lesser curvature 1.5 cm. from the pylorus.

There are 2 problems in the pathology of gastric ulcer, first the origin of the initial lesion, and second, the cause of the chronic character. It is a well established fact that histamine when injected subcutaneously increases the flow of gastric secretion and especially the hydrochloric acid. The acid content of the gastric juice has been regarded by some for many years as a potent factor in the causation of chronic gastric ulcer. Ulcers of the peptic type occur in 4 situations: (1) stomach; (2) duodenum; (3) stoma of gastro-jejunostomy; (4) lower end of oesophagus. These areas are more or less continuously in contact with gastric secretion. Gastric ulcers have been known to follow injury caused by the passing of a stomach tube.

The following experiments have been carried out in order to ascertain whether or not trauma associated with hyperacidity might be causative factors in the production of gastric ulcers. I traumatized the gastric mucosa in the following manner: laparotomy was