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

performed and the stomach exposed. The serosa was incised and dissected back exposing 5 mm. of the subserous tissue. A piece of brass wire 3 mm. in diameter heated to a dull red was plunged through the remaining stomach layers. The stomach wound was closed by 2 layers of purse-string sutures and the laparotomy wound closed in the usual way. Twenty-four animals have been so treated. Seventeen were used as controls while the remaining 7 were treated by injections of histamine (ergamine acid phosphate B & W) in amounts varying from 10 to 20 mg. per dose. Injections were begun 2 days after operation and continued every alternate day thereafter until the animal was chloroformed.

In order to determine the approximate length of time required for the mucosa in different areas of the stomach to heal, 8 received cardiac trauma, 5 pyloric and 3 both cardiac and pyloric. In all of the above the cardiac lesions had healed completely in 14 days while those in the pylorus required 17 days at least. In the animals which received both cardiac and pyloric injuries the latter were not healed in any case, proving conclusively that it requires a longer interval for the healing of pyloric lesions. The pylorus was therefore chosen as being the most suitable location for the further carrying out of this experiment.

Pyloric lesions were therefore produced in the remaining 7 animals and in addition each received subcutaneous injections of histamine with the following results:

Cat	*Length of time treated*	*Result*
1	8 days	Extension of original lesion. No attempt at healing
2	12 days	Extension of original healing. Perforation
3	15 days	Extension of original lesion
4	15 days	Extension of original lesion
5	15 days	Extension of original lesion
6	11 days	Extension of original lesion
7	11 days	No extension of original lesion. Some attempt at healing. Three small ulcers in mucosa and pylorus

Although in none of the above may it be claimed that a chronic peptic ulcer has been produced, I believe that histamine in some as yet undetermined manner led, undoubtedly, to the extension of the lesions produced. It appears probable, however, that the extension of the lesion was due to the increase of hydrochloric acid induced by the action of the histamine. Further experimentation may clarify this point.

I wish to thank Dr. H. U. Williams for invaluable suggestions relative to the carrying out of this work.