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Antiperistalsis in the upper third of the esophagus in man.

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The subject of this investigation was a child of thirteen years who, at the age of four, swallowed strong acids, and in consequence developed almost complete cicatricial stenosis of the esophagus, the upper end of the occlusion being about 5 cm. below the level of the sternum. During the nine years the child had taken nourishment almost exclusively by a gastrostomy tube. For months at a stretch not even a drop of water could pass through the esophagus into the stomach. Occasionally an opening of 2 to 3 mm. diameter appeared, allowing water and milk to pass from the mouth into the stomach. Evidently a chronic spasm of the injured portion of the esophagus had developed on the top of the mechanical stricture. During an investigation of possible means of controlling the esophageal spasm, the child frequently swallowed small quantities of barium milk while fluoroscopic observations were being made. There was little or no permanent dilation of the end of the esophagus above the occlusion. The presence of the barium milk in this region led at once to vigorous movements of the esophagus resembling the movements of the small intestine above an obstruction, as originally described by Cannon.[1] One could make out local rings of constriction (segmentation movements) at different levels, as well as regular peristalsis. There frequently appeared vigorous antiperistalsis, forcing some of the barium milk towards and even into the mouth. The esophagus was able to empty itself completely in this way. We could secure no evidence that the mechanism was under voluntary control. The antiperistalsis did not induce any special sensation, such as nausea. Material in the upper end of the esophagus was felt as "something stuck in the throat," whether or not antiperistalsis was present.

The musculature of the upper third of the esophagus in man is striated, with motor control via the vagi nerves. There is no

[1] Cannon, W. B., "Mechanical Factors of Digestion," 1911, p. 131.

evidence that local automatism ever develops in this part of the esophagus, at least as long as the vagi are intact. We have, therefore, in this case an illustration of antiperistalsis coordinated reflexly through the central nervous system.

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In vitro studies on ammonia and urea formation by tissues.

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This investigation was undertaken in the hope that light would be thrown on the subject of desaminase action by the various tissues of the body. The subject is in a more or less unsettled state, some workers[1] even disclaiming a true deaminizing action in tissues, believing that the ammonia production is due to deamidase action. Still others[2] take the view that the amino acids instead of yielding ammonia, are attacked in the carbon chain itself, thus being broken down and oxidized, the products formed producing cyanic acid which can be converted into urea by the addition of ammonia formed by deamidase action.

In this work an attempt was made to study the action of ammonium salts, or of mixtures of amino acids and peptides, or of amino acids alone upon ammonia and urea formation or utilization by tissues *in vitro*.

The tissues used were obtained from recently killed dogs or from the abattoir, in this case using hog tissue. These were minced as soon as possible, mixed with a buffer phosphate solution and after an hour the juice was filtered and pressed out. Of this well mixed fluid, 25 cc. portions were taken and incubated with the additions referred to. All the necessary control estimations were made and the methods used were critically studied before using them on the problem. The tissues studied were liver and kidney.

[1] Luck, J. M., *Biochem. J.*, 1924, viii, 814.

[2] Weiner, E. A., "Chemistry of Urea," 1923, London.