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Gastric Juice Acidity at the End of the Secretory Period.

FRANKLIN HOLLANDER. (Introduced by I. S. Kleiner.)

From the Department of Physiology, New York Homeopathic Medical College and Flower Hospital.

For many years, the theory of Heidenhain and of Pavlov—that the concentration of acid in the isolated parietal secretion is constant within narrow limits—was almost universally accepted. Most of the recent contributors, however, have taken the view that this fluid is a mixture of HCl and neutral chlorides, in varying proportions. The present paper is a refutation of one of the major arguments advanced by this latter group,[1] and deals with the decrease in acidity of the gastric juice during the latter part of the digestive period.

Variations in acidity of gastric juice are always observable, even in the relatively pure juice obtained from dogs with stomach pouches. Such variations were interpreted by Pavlov as resulting from partial neutralization by mucus. A major point in support of this explanation is the obvious parallelism of acidity and the rate of post-prandial secretion during any one experiment. In a previous communication, such correlation was confirmed[2] for food and small doses of histamine. When the gastric juice was collected by continuous drainage, samples being taken during quarter or half hour periods, it was observed that every increase or decrease in volume per period was accompanied by a like change in acidity. However, after increasing the rate of secretion sufficiently, as by the injection of a large dose of histamine, this parallelism vanished and the acidity became constant at a pH of 0.91. This is entirely in accord with Pavlov's conception.

In opposition to the theory, it was first suggested by Rosemann[3] and by Foster and Lambert[4] that the fluid secreted by the parietal cells possesses a fairly constant chloride content. With increased intensity of stimulation, and therefore increased rate of flow, the proportion of neutral chloride converted to acid is likewise increased, resulting in the commonly observed fluctuations in acidity. One of the chief arguments by Rosemann[5] is: If the low acidities observed

[1] Babkin, *Am. J. Surg.*, 1929, vii, 499.

[2] Hollander, *J. Biol. Chem.*, 1927, lxxiv, xxiii.

[3] Rosemann, *Pfluger's Arch.*, 1907, cxviii, 467.

[4] Foster and Lambert, *J. Exp. Med.*, 1908, x, 820.

[5] Rosemann, *Virchow's Arch.*, 1920, ccxxix, 67.

at the beginning of the secretory period are due to neutralization of mucus which is present in the pouch at this time, it might be expected that all such mucus would have been removed by the latter part of the experiment. Thus, there should be no corresponding decline in acidity at this time. However, such a reduction does occur, and the only other possible explanation—that of an increase in mucus flow—is not substantiated by a marked increase in mucin content of the last samples of juice.

In order to settle the argument centering about this point, numerous experiments have been performed with Pavlov and Heidenhain pouch dogs. Both foods and histamine injections have been employed as stimuli. The gastric juice was obtained by the continuous collection method; i. e., by the use of a soft rubber catheter kept in the pouch of a dog, supported by a sling in an upright position. Precautions were taken to obviate excessive mucus secretion by irritation; also admixture of exudate from the outer abdominal wall with the juice was prevented.

The first series of experiments employed a weak stimulus, so that the secretion at no time attained a pH value as low as 0.91. In such experiments a decrease in acidity is invariably found at the end of the secretory period. However, a comparison of the acidities at the beginning and towards the end of the period (*at times such that the secretory rates are about the same)* indicates that almost invariably the acidity is much lower at the beginning than at the end. Of course, when collection of juice was continued beyond this point, the acidity fell still lower and approached the value for unacidified mucus. Such values are obviously not comparable with the early ones, because of the greatly increased proportion of mucus which they contain.

In the second series, the histamine dose was raised to 0.4 mg. per kg. In all cases, by the second or third quarter-hour sample, the acidity had attained the maximum value where it remained throughout the experiment. Even the last sample collected showed the same acidity—or one below it by an insignificant amount—provided mucin clots were absent. Usually the presence of mucin in these samples betokens an almost complete cessation of acid secretion. Thus by increasing the rate of flow of HCl, the contact surface between mucosa and catheter is lubricated sufficiently to reduce mucus secretion to a minimum. This in turn, minimizes the reduction in acidity of samples collected towards the end of the secretory period.

These observations are wholly in agreement with the theory of Heidenhain and Pavlov. Furthermore, they satisfactorily account

for the evidence cited above in opposition to this theory. The agent which is effective in neutralizing the acid is not the mucus which is already present at the beginning of secretion. On the contrary throughout the experiment there is a continuous flow of such neutralizing and diluting fluid, due in great part to irritation of the mucosa by the catheter. With reduction in this mucus flow, the late fall in acidity becomes less and less observable.

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Does the Virus of Poliomyelitis Survive in the Monkey Testicle?*

CLAUS W. JUNGEBLUT AND RICHARD THOMPSON.

From the Department of Bacteriology, College of Physicians and Surgeons, Columbia University.

Among the points of major interest in experimental poliomyelitis is the question as to how long the virus is capable of surviving in different organs. Such data not only furnish information which is helpful in studying the factors responsible for selective tissue susceptibility, but may also add to our knowledge concerning the mode of invasion in the disease. From this viewpoint, the testicle has assumed particular prominence in practically all neurotropic virus diseases. It may either serve as portal of entry from which a generalized infection takes its beginning, or, more particularly, offer excellent ground for the development of a circumscript local lesion (herpes, vaccinia). It appears that infection by the testicular route so far has not been attempted in experimental poliomyelitis, save for the recent work of Thompson,[1] who reported his inability to infect rabbits by intratesticular injection with monkey virus. In these experiments it was also found that the virus does not survive for a period of even 24 hours in the rabbit testicle, as determined by subsequent transfer to the monkey.

We considered it important to extend these experiments to a study of the capacity of the testicle of a susceptible animal, i. e., the monkey, to serve either as a portal of entry for the disease or as a suitable living culture medium for the infectious agent. These experiments moreover afforded an opportunity to determine whether

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[1] Thompson, R., *J. Exp. Med.*, 1930, li, 777.