Comment

PERFUSION TECHNIQUES IN THE STUDY OF ABSORPTION IN MAN

Recently K. R. Soergel disagreed with G. E. Sladen and A. M. Dawson concerning their use of two-lumen and three-lumen tubes for intestinal perfusion (*Gut*, 1969, 10, 601-602).

We have recently made a study (to be published shortly) of the absorption of glucose, sodium, and water by human jejunum, using a two-lumen tube, with a proximal occlusive balloon, and have compared the results with the balloon inflated and deflated.

We have found that we could recover, above the inflated balloon, a volume of endogenous secretions ranging from 0 to 300 ml within 70 minutes. This volume varies with the subjects, and in the same subject with time, when the infusion lasts several hours.

With the balloon deflated the reflux of the infused solution could be demonstrated by recovering by a tube, the end of which was 12 to 15 cm above the infusion point, a glucose- and PEG-containing fluid.

The absorption of glucose with the balloon deflated, measured at different rates of perfusion, was significantly higher than with the balloon inflated. This effect was more important for higher rates of infusion. Although we cannot formally exclude an inhibitory effect of a balloon on the absorption phenomenon itself, we think that the reflux of the infused solution increases the length, and thus the surface, of the test segment.

The comparison of water and sodium absorption with inflated and deflated balloons gave less clear-cut results. In some experiments the difference was not significant. In others it was, and the results suggested that the reflux phenomenon was more important than the 'pollution' effect. We think, therefore, that the use of a balloon can combine the simplicity of the two-lumen tube with the precision of the three-lumen tube.

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ISOTOPE TECHNIQUE FOR MEASURING LACTOSE ABSORPTION

I refer to the interesting paper by Salmon, Read, and McCarthy on an isotope technique for measuring lactose absorption (*Gut*, 1969, 10).

The technique itself and the data handling and description seem impeccable, but I wonder if they are really serious in suggesting that this be used as a screening test for lactose malabsorption? The precise diagnosis will eventually require jejunal biopsy and enzyme assay and the question then arises how sensitive a screening test should be. I would suggest that the simplest procedure is to give patients an 80g dose of lactose and observe whether they complain of diarrhoea, cramping, bloating,

Notes and activities

CONFERENCE ON THE INTEGRATION OF GASTRIC FUNCTIONS¹

Twenty-eight scientists from the United States and four foreign countries met from 20 to 23 July 1969 in Hanover, New Hampshire, for a closed interdisciplinary conference on the 'Biology of the stomach and the pathogenesis of peptic ulcer'. In the recognition of the extensive evaluations that have already been made of the role of gastric acid secretory activity in ulcerogenesis, the participants specifically directed their attention to examining other factors having a bearing on this phenomenon.

J. S. Fruton (New Haven) reported that partial identification of the molecular structure of pepsin shows it to contain approximately 321 amino acid residues and 39 carboxyl groups. Preferentially, it attacks the peptide bonds between two aromatic acids of, which *L-Phe* is one. Secondary interactions related to adjacent molecular configurations, however, can significantly modify the rates of cleavages at different bonds; this suggests an explanation for the apparent non-specificity of this protease. P. S. Cammarata (Chicago) reported the synthesis of a sulphated mucopolysaccharide, which is a potent inhibitor of peptic proteolytic activity, both *in vivo* and *in vitro*. Its ability to compete with substrate for sites on the enzyme and as well to protect the substrate from proteolysis have been demonstrated. Poorly absorbed, this compound can be found coating the gastric lining for six to nine hours after ingestion. Taken in this manner, preliminary observations indicate that it affords prompt relief of ulcer pain. This finding is of particular interest, in the light of E. R. Perl's (Salt Lake City) discussion of the mechanisms for triggering pain receptor activity, and the recently recognized role of polypeptides in this regard. The possibility that gastric proteases, or byproducts of their action on protein substrates, might also serve to mediate information to gastric sensory pathways is intriguing.

In a session chaired by F. G. Moody (Birmingham, Alabama) circulatory factors and their relationships to ulcerogenesis were considered. O. Lundgren (Göteborg)

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Isotope technique for measuring lactose absorption—*concluded*
borborygmi, gas, or flatulence, the clinical criteria which the authors themselves use and at least two of which were present in their group of abnormal patients. In my experience, the clinical syndrome of lactose intolerance due to hypo- or alactasia has always been accompanied by abdominal symptomatology of the type mentioned in relation to an 80 g lactose tolerance test.

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