Commentary

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The duodenum: a conduit or a pump?

In the article by Nguyen *et al* in this issue (see page 624), a novel impedance method was used to characterise the transport of chyme in the duodenum in the postprandial period. The authors suggest that abnormal postprandial transport of chyme in the duodenum may be a factor that results in impairment of gastric emptying in patients with long-standing type I diabetes mellitus. The paper raises a number of interesting issues.

The first question is: does the human duodenum actually perform any hydrostatic or pumping function? In a previous *in vitro* model, Weems¹ suggested that the cat duodenum was incapable of hydrostatic function, in contrast to the ileum. This observation was supported by the very different nature of contractions observed in the mammalian upper small intestine and terminal ileum. Thus, Phillips and colleagues at the Mayo Clinic demonstrated that the prolonged propagated or giant migrating contractions,² which are associated with ileocolonic flow *in vivo*,³ are quite specific to the terminal ileum and do not normally occur in the human upper small intestine. The interdigestive migrating motor complex propels contents in an aboral direction during fasting. Inhibition of the interdigestive complex and the short length of propagation of individual contractions in the small intestine postprandially⁴ suggest that the duodenum might act mainly as a conduit incapable of hydrostatic function. If such a hypothesis is true, the pressure gradient generated by high amplitude contractions in the distal stomach would result in aboral movement of chyme through the duodenum postprandially.

Previous research using a different device that measures traction forces in the upper small intestine in healthy subjects suggested that the duodenum was capable of inducing bolus transport events similar to those described by Nguyen *et al*. Thus, Ahluwalia *et al* documented the traction forces in the upper small intestine⁵ and observed their modulation pharmacologically.⁶ Nguyen and colleagues used a novel impedance technique that is capable of measuring both the propagation of contractions in the segment of intestine assessed and the aboral transport of chyme in the intestine. The finding of abnormal duodenal transport in the postprandial period in a disease state is new, although other investigators have previously suggested that neuropathic disturbances associated with chronic intestinal pseudo-obstruction impair gastric emptying.^{7,8} Nguyen *et al*'s study, therefore, adds credence to the concept that impaired duodenal propulsion or increased resistance to flow in the small intestine caused by intestinal dysmotility may be co-factors impairing gastric emptying in people with long-standing type I diabetes mellitus.⁹

The second question raised by the observed phenomenon in the duodenal loop pertains to the mechanism that results in the abnormal motor function and duodenal stasis. For example, it is still not certain why there is a significantly lower number of propulsive bolus transport events. It is conceivable that this represents reduced motor function of the duodenum, but it is also possible that antral hypomotility or impaired gastroduodenal coordination may result in a reduced capacity for transport of chyme in the duodenum. Nguyen *et al* could not assess the relative hydrostatic contributions of gastric pumping transmitting the force through an open pylorus versus that of duodenal pumping. Techniques capable of simultaneously measuring contractions at multiple recording sites, their propagation, and their propulsive significance will provide opportunities in the future to understand better both the temporal and spatial relations of contractions and also their hydrostatic impact. However, these studies provide no insights into the underlying control mechanisms that alter the duodenum's propulsive function in diabetes mellitus.

Thus, applied physiologists and students of neuroenteric motor function welcome this technological advance; this study illustrates the potential for an enhanced descriptive understanding of processes in health and disease that were difficult to assess using previous technologies. Future studies should also tackle the underlying mechanisms by incorporating studies of pharmacological agents as well as intrinsic and extrinsic denervations in animal models.

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