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DISCUSSION

DR. LLOYD M. NYHUS (Chicago): The vagus nerves have come a long way since Pavlov. Stimulation of the parietal cell to secrete hydrochloric acid, vagal release of the hormone, gastrin, interrelationship of the vagus and the hormones, secretin, and cholecystokinin and now, the beautiful dissection of the role of the vagus in control of gastric pressure and motility by Wilbur and Kelly.

These studies, confirm by elegant methodology, that preservation of the vagal nerves to the antrum allow normal or near normal function of the antrum and pylorus.

Following proximal gastric vagotomy, cinefluorography in our own patients demonstrates this continued antral function dramatically.

The authors have given us more information. They have differentiated between the regulation of gastric emptying of liquids by the vagally innervated proximal stomach by a transmural

pressure system and the emptying of solids by the vagally innervated antrum.

A major cause for concern with the operation of proximal gastric vagotomy is the unknown recurrent ulcer rate.

Is there a danger of vagal re-innervation of a proximal stomach by the nerves of Latarjet left intact to the antrum?

There is no answer to this question. However, Wilbur and Kelly have given to us new and important research tools which may help to answer this tantalizing question.

Finally, have the authors repeated the gastric transmural pressure recordings at longer intervals than 1 month after proximal gastric vagotomy? Longer observation may clarify certain of these important issues.

DR. EDWARD ROY WOODWARD (Gainesville): I had the pleasure of reading the complete manuscript and this is certainly a beautiful demonstration of the role of the vagus in the emptying function of the stomach.

We have been intrigued by the potentialities for the operation of proximal gastric vagotomy for duodenal ulcer and our early results in 15 cases are good. The basal acid output was sharply reduced in nearly all instances and we have been interested in that the stimulated secretion falls very little or none.

Our laboratory evaluation of this procedure has proceeded along somewhat different lines. In the dog we have used a meal of 500 Gm. of blenderized horsemeat introduced into the stomach. We observed that in the control animal the pH in the fasting stomach is 2.4 and the serum gastrin 23 picograms per ml.

There is a definite rise in the serum gastrin with this relatively large meal. We were surprised to find how long it took the normal dog's stomach to empty this semi-solid meal, 7 hours and 15 minutes with a standard error of 1 hour.

After proximal gastric vagotomy, the resting pH in the stomach was essentially the same. However, the serum gastrin level was nearly three times that in the control animal. Even more striking was the very sharp increase in the serum gastrin which followed the introduction of the semi-solid meat meal. The emptying time was essentially the same. Statistically, these figures are not different so that the emptying of the semi-solid meal was not affected by proximal gastric vagotomy.

DR. JAMES CHARLES THOMPSON (Galveston): There are several things brought out by this fine paper. One of them challenges an idea that I, at least, have held, that the duodenum functions as an osmoregulator. Many of our classic ideas about the dumping syndrome have stipulated that anything that either bypasses or destroys the function of the pylorus will lead to dumping because it will allow the rapid passage of hyperosmolar material into the upper small bowel. These studies are the first I know of to directly address that question and to come to the opposite finding.

Secondly, this study provides us with further information on some fascinating bits of knowledge that we are beginning to gather about the effects of various acid-reducing procedures on serum gastrin levels.

Clinically, all reports of operations of vagotomy and emptying procedures in which gastrin measurements have been done show that there is a great postoperative increase in fasting gastrin levels. This has been, I think, usually ascribed to a diminution in the acid inhibition, the acid feedback mechanism.

Drs. Wilbur and Kelly show us that in this operation at least there is another facet to be considered. There is a significant increase in antral pressure which probably would lead to some distention of the antrum and perhaps further release of gastrin. The coincidence of the two, that is the relatively high pH due to vagal denervation of the parietal cell mass and the increased pressure, would perhaps together be synergistically responsible for the rather high, sometimes doubled, gastrin levels and we and others have found postoperatively in patients with vagotomy and pyloroplasty.

Conversely, our results are somewhat different from those reported by Dr. Woodward in that we do not get stimulated levels of acid secretion.

Lastly, I think, as far as our remarks, this brings us to the question of when we do the operation of proximal gastric vagotomy, should we include an emptying procedure?

When Fritz Hölle in Munich first began his study of this operation, in a few patients he did no antrum procedure and then because of high acid levels and a few recurrences, he adopted pyloroplasty.

As you all know, Dr. Andrup in Copenhagen and Dr. Johnston and Dr. Golligher in England have reported now a rather large series in which their results seem to be quite good in patients in whom a proximal gastric vagotomy without emptying procedure had been performed.

I would agree with Lloyd Nyhus in that I think we have to wait awhile before we are sure this is going to be a safe operation. It would seem to me that now, control trials should be directed as to whether or not a gastric emptying procedure should be included with parietal cell or proximal gastric vagotomy, which, I suspect, will be the operation of the future (with or without an emptying procedure).

DR. KEITH A. KELLY (Closing): Dr. Nyhus, we have not studied the animals longer than the period indicated in our report, but it would be valuable to know if there is any return of vagal function in the proximal stomach after months or years.

Dr. Woodward, the semi-solid meals that you are using must be handled in a similar manner to the solid spheres that we have used, since you did not show any increase in their speed of emptying. Perhaps if you had given your dogs or your patients liquids, you would have found gastric emptying faster.

Dr. Thompson, I do not know whether the increases in transmural pressure that we have shown after proximal gastric vagotomy would result in an increased release of antral gastrin. The increases in pressure are not large, but perhaps they are great enough to augment the output of gastrin.

Our data would support the fact that a procedure designed to increase the speed of gastric emptying is not required with proximal gastric vagotomy. If anything, we have shown that proximal gastric vagotomy speeds rather than slows gastric emptying.

The concept emerging from these experiments is that the proximal and distal parts of the stomach each have separate functions. The proximal stomach accommodates ingesta, relaxes, and keeps transmural pressure low. The distal stomach grinds and mixes.

The vagus has an important regulatory role in both the proximal and distal stomach. When we operate for duodenal ulcer, it is important to divide as few vagal nerves as is consistent with the treatment of the disease.