thology have normal gastric emptying after fundoplication and that these patients have minimal side effects from the operation. Those who remain outside the normal range are very likely to have postoperative symptoms. The presence of concomitant gastric pathology should not be ignored because it may be associated with delayed gastric emptying and persistent postoperative symptoms. On the other hand, surgical treatment of the concomitant gastric pathology should not be undertaken lightly because the additional procedure might alter gastric emptying and contribute to an unsatisfactory outcome.

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Discussion

DR. C. THOMAS BOMBECK III (Chicago, Illinois): The authors have very bravely attacked what was initially a very simple problem and what is now a very complicated one. In the late 1970s, there were several reports that patients with gastroesophageal reflux had delayed gastric emptying. Indeed I believe that when Dr. DeMeester was at the University of Chicago he reported one of the first of these. In 1985 it was first reported that the Nissen fundoplication corrected the delayed gastric emptying, normalizing it or at least accelerating it from the preoperative levels.

More recently, in our own laboratories, Drs. Carrallho and Richter have demonstrated in the experimental animal that a Wendell cardioplasty, a mechanical procedure that destroys the lower esophageal sphincter without at all endangering the vagi or impinging on the stomach, markedly increases or delays gastric emptying. We have questioned exactly why this occurs. Now we are trying to sort out whether it is due to acid contact with the esophageal mucosa producing, if you will excuse the pun, a reflux-reflex inhibition of gastric emptying, or if it is due to a simple mechanical effect, venting of gastric fundic pressure thereby destroying the efficiency of gastric peristalsis.

I would first like to ask the authors why they think some patients do have delayed gastric emptying. Second the authors demonstrated a group of patients who had no evidence of gastric pathology, some of whom had delayed gastric emptying and others who did not. There must be a difference between those two groups of patients. It is interesting to speculate that it might be in the degree of LES failure.

Two years ago before this association we presented a method of quantitating LES sphincter strength, which was considerably more detailed than the standard pull-back procedure, and I wonder if the authors have used that or any other in an attempt to distinguish these two groups of patients. Do patients with delayed emptying have a worse degree of LES failure than patients without?

At the same time that all of this has been going on, we have known very well that various types of gastric pathology have caused delayed gastric emptying, such as gastric outlet obstruction for one reason or another or gastric mucosal disease. This causes us to wonder, in terms of reflux and delayed emptying, which was coming first, the chicken or the egg? The results that you have heard here today seem to suggest that they arrive simultaneously. In fact it seems that these authors have defined two groups of patients. Those who have delayed gastric emptying because they have gastric pathology and those who have delayed gastric emptying because they have a failed lower esophageal sphincter. It would seem to be of great importance to be able to define those two groups of people before operation.

Would the authors recommend, therefore, that all patients should have studies of gastric emptying, perhaps gastric secretory studies, before they have a Nissen fundoplication? If not, why not?

DR. JAMES A. O'NEILL, JR. (Philadelphia, Pennsylvania): Dr. Hinder and his colleagues clearly describe that some patients with gastroesophageal reflux have delayed gastric emptying, and that the incidence of this problem in his patients may be as high as two thirds of those with gastroesophageal reflux. This is important because, at least in the past, some have suggested that some sort of gastric emptying procedure should be performed in such cases.

This very suggestion led us to look at this issue 5 years ago when we reported 18 patients with reflux strictures of the esophagus to this Association. At that time, we noted that 4 of our 18 patients had delayed emptying, and all of these patients were carefully studied. In all of these patients, the emptying normalized after operation following a Nissen fundoplication.

Recently one of our fellows reviewed more than 200 patients in whom a Nissen fundoplication was done. The incidence of delayed gastric emptying was 25%. It was 20%, as you saw in the reflux stricture group. Now what was noted, however, was that the majority of these patients had severe esophagitis. This leads me then to ask three questions. Why do you think that your incidence is 2.5 to 3 times what we have seen in our patients, although I must say the fact that our patients were children would place them in your no gastric pathology group? We have followed our patients with both milk technetium scans and scrambled egg scans as opposed to oatmeal.

Do you think that esophagitis is an issue in etiology that would perhaps suggest that the lower esophageal sphincter is to be indicted? And finally, as you did, I think all of us have rare patients who are not relieved after operation, and I wonder, does your data help you to identify these patients so that perhaps gastric emptying procedures could be applied selectively and appropriately?

DR. CARLOS PELLEGRINI (San Francisco, California): Indeed this paper illustrates some interesting physiologic and pathologic aspects that have direct clinical application.

In your paper you tell us that 64% of the patients had associated gastric pathology. Would you please define what you consider gastric pathology? Second why is there such a high incidence of gastric pathology in association with abnormal gastroesophageal reflux? Were these patients selected, or does this represent a consecutive series of patients?

You found that nine patients with gastric hypersecretion also had delayed gastric emptying. By contrast most other authors have found that patients with gastric hypersecretion have rapid gastric emptying. The typical case is that of patients with Zollinger-Ellison syndrome who empty very rapidly. Could you tell us what is your interpretation of this rapid emptying?

The finding of rapid gastric emptying after Nissen fundoplication is, in my opinion, not surprising. Dr. Kelly showed rapid gastric emptying in dogs after fundectomy of the stomach, and I believe that the wrap in the Nissen fundoplication uses most of the gastric fundus. This deprives the stomach of its reservoir capacity and therefore rapid gastric emptying is to be expected. On the other hand we have shown that, in most cases, rapid gastric emptying is well tolerated and that few patients with rapid gastric emptying after gastric surgery complain of symptoms.

My final question has to do with the message of this study. Because of this common association with gastric pathology, are you suggesting that all patients with abnormal gastroesophageal reflux ought to be evaluated in terms of gastric secretion and motility? And if your answer is yes, how should we apply the results of these tests in planning the operation? For example, would you advise the addition of a parietal cell vagotomy in an asymptomatic patient with gastric hypersecretion who is operated on for abnormal reflux?

DR. ERIC W. FONKALSRUD (Los Angeles, California): I would also like to congratulate Drs. Hinder and DeMeester and their colleagues on an excellent presentation and for providing us with helpful and new information regarding the relationship of gastroesophageal reflux and delayed gastric emptying. Although there may be some differences in what you have observed in adults and what we see in young children, particularly those with central nervous system disorders, there may be more similarities than previously realized. More than 50% of both adults, as indicated in today's presentation, and young children with symptomatic reflux have delayed gastric emptying before operation. Although reduction of gastric volume by fundoplication may enhance gastric emptying transiently, approximately one fourth of the patients in the present study had symptomatic gastric dysmotility after operation, which is similar to our experience with children.

How long does the accelerated emptying after the fundoplication persist? Is this a matter of months or years? Have you performed a subsequent pyloroplasty on any of the five patients who had postoperative symptomatic delay in gastric emptying?

Fourteen children in our hospital required reoperation with pyloroplasty when gastric emptying subsequently showed more than 70% isotope retention at 90 minutes. None of these patients had apparent vagal injury from the fundoplication. A competent fundoplication combined with postoperative delay in gastric emptying may produce a form of closed loop obstruction, particularly in children who are often quite aerophagic. We now routinely study gastric emptying in all children with symptomatic reflux and have found a severe delay in more than 15% of patients. We have, therefore, combined a loose fundoplication with a pyloroplasty in most of these children. Repeat gastric emptying studies 6 weeks to 1 year after operation have shown less than 50% isotope retention at 90 minutes in each of the 68 children who underwent pyloroplasty combined with fundoplication.

The incidence of gas bloat and slipped or malfunctioning fundoplication has been reduced to less than 5% because pyloroplasty has been used more frequently in our experience. Before this time we had a much higher incidence of slipped Nissen fundoplications and other complications.

Have you been able to identify before operation which of your refluxing patients will have symptomatic delay in gastric emptying after fundoplication?

DR. JONATHAN E. RHOADS (Philadelphia, Pennsylvania): We have been impressed with the relationship between the time of gastric emptying and the level of serum proteins. I thought particularly in relationship to the first chart comparing the patients with gastric pathology and those without that I would like to know what the serum proteins were in the two groups and whether this has been controlled?

DR. RONALD HINDER (Closing discussion): I would like to thank all the discussants for their interesting questions. First Dr. Bombeck. I was interested to hear that you found delayed gastric emptying to occur after the Wendell gastroplasty. This lends support to the concept of venting of intragastric pressure into the esophagus through an incompetent sphincter; however this is not supported by our previous studies (Schwizer W, et al. Am J Surg 1989; 157:74–81) in which we showed that there was no relationship between sphincter competence and delayed gastric emptying in patients with foregut symptoms. The hiatal hernia lying in the negative intrathoracic pressure probably plays a role in delaying gastric emptying. It is also possible that lower esophageal pathology may also involve the proximal stomach, on the one hand producing reflux and on the other hand producing delayed gastric emptying. You also asked about sphincter vector volume. We are aware of the work that you have done and are also now measuring sphincter volume, but not on the patients in this study who all had conventional sphincter manometry. Concerning preoperative evaluation of the patients we found that there was no way that we could predict by our preoperative evaluation what the postoperative outcome would be.

As far as Dr. O'Neill is concerned, he conceded that in children the disease may be different than that seen in adults. There must, however, be some similarities, as Dr. Fonkalsrud suggested. The difference in adults is that we see more associated gastric pathology than in children, and this probably caused the delayed gastric emptying that we saw in our patients as opposed to that reported in children.

Dr. Pelligrini asked about our high incidence of patients with gastric pathology. These were not consecutive patients. They were problem patients and so obviously had a high incidence of gastric pathology. He asked whether we should do all of these tests routinely in our patients. In most patients we don't need to have all of this information and reserve this detailed testing for patients for whom our clinical judgment would suggest that it is required.

Dr. Fonkalsrud wished to know how long after operation we had carried out our gastric emptying tests and whether the results would change with time. We carried out our tests from between 1 and 35 months after surgery with a median of 12 months. We found that the results were similar in those tested early after surgery compared to those long after surgery. He raised the question whether pyloroplasty should be used in patients with delayed gastric emptying. This procedure should not be used routinely in patients with delayed gastric emptying because pathologic duodenogastric reflux may be the cause of the problem in some patients and could be enhanced by pyloroplasty.

Finally Dr. Rhoads asked if we knew the serum protein values in our patients. The patients were generally well nourished and I would assume that the serum proteins were normal.