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Discussion

DR. EDWARD W. HUMPHREY (Minneapolis, Minnesota): Dr. Stein has presented us with a method of mathematically modeling the lower esophageal sphincter area. I have long admired the efforts of Dr. DeMeester's group to raise the work on the lower esophageal sphincter from the realm of metaphysics to that of real science, and this paper is no exception.

I do have three questions on this work. The first is, what is the reproducibility of the sphincter pressure vector volume with time in the same individual? If it is good, it might permit the longitudinal studies to finally learn whether the motor abnormalities seen with esophagitis are the cause or effect of the abnormal reflux.

Secondly I made some extrapolations from the graphs in your manuscript. I found that although the absolute value of the abdominal portion of the pressure vector volume is less in the patients with esophagitis and the total volume is less, the proportion is the same. In patients with esophagitis, 67% of the total volume is below the diaphragm and thus in the abdomen; in your volunteers it was 63%. With the error in my extrapolation, those are essentially the same, and I wonder if you can explain that? Because 80% to 85% of patients with abnormal reflux have a hiatus hernia, I would have expected the abdominal portion to be considerably smaller in patients with esophagitis.

Third have you noted any differences that will predict which of the patients will be in the 10% to 15% that have a poor result from a fundoplication? If you could do that with this method, it would be a significant advance.

Thank you very much.

PROFESSOR MARTIN ALLGOWER (Basel, Switzerland): I have four questions (they are rather naive) and one comment. The Basel anatomist was telling us surgeons that we have a very astounding capacity to name and to cut structures he had never seen. I think it is the merit of Doris Lieberman to describe the anatomic reality of the lower esophageal sphincter.

My first question is, whether sphincter pressure vector volumes in a way do mirror the anatomic findings that Lieberman has been describing? Naturally the actual clinical application of the Lieberman procedure would be somewhat devastating to the patient! I wonder whether you agree that your quantification of LE-function constitute an interesting confirmation of Doris Lieberman's findings.

Second the merits of your three-dimensional imaging are to a large extent validated by their conformity with the increased esophageal acid exposure. Now my question is, what is the additional information with regard to therapeutic decisions taken from your values?

Thirdly the reflux disease without mucosal lesions seems to be the main real case for your method. Now could it be that your method picks out the known hypersecreters who would probably benefit from an early antirefluxplasty?

And fourthly one "philosophical" question: Does not the "amount of subjective suffering" constitute an important element of an indication for operation?

I enjoyed so very much to see Bombeck's finding substantiated and made more easily applicable.

Naturally I was particularly happy to see that the Nissen procedure really has stood the test of time. Thank you very much for this very good paper.

DR. PHILIP DONAHUE (Chicago, Illinois): My only slide—may I see it now, please?—illustrates a concept of the vector volume and introduces my three questions. This is actually one of Tom Bombeck's slides from his presentation here in 1987. A "full-bodied" sphincter is normal; "skinny, abnormal-looking" sphincters, are abnormal. After fundoplication, the contour of the sphincter is more normal.

My first question relates to vector volume: The computer program converts multiple virtual areas of segments of the sphincter into a volume, by multiplying an average area times the length. The radius of that cylinder is the critical factor, and I am concerned about the station pullback technique that you have used. You thought that it was better than rapid pullback technique, but we believe the rapid pullback eliminates subjective factors in estimating the average pressure along sphincters. When station pullback is employed, bias is introduced.

How do you avoid subjective bias in estimating pressures?

Secondly we have found that asymmetry of the sphincter is an important concept (Probably affecting only patients with marginal pressures). Can you tell us whether you have noted the presence of asymmetry in any of your patients with failed sphincter?

Finally the vector volume concept still does not identify 20% of patients who require surgery, because 20% of the patients you operated on had a normal study; we believe that abnormal (but as yet unmeasurable) aspects of gastric components of the reflux barrier can help explain reflux disease in some of these patients. I invite your comments about this possibility.

DR. HUBERT J. STEIN (Closing discussion): Thank you very much for those kind remarks. I would like to answer Dr. Humphrey's questions first. He addressed the reproducibility of the measurement over time. We performed reproducibility measurements within the same subject within 2 or 3 hours. Within this short period, all of the measurements, in other words, the rapid and stepwise pullback, were highly reproducible. We do not have long-term reproducibility studies yet, but it would certainly be interesting to see what they would show.

Why was the percentage of the intra-abdominal segment of the sphincter similar in volunteers and in the patients with esophagitis, even though many of the latter had a hiatal hernia? We have previously shown that the presence of a hiatal hernia does not necessarily mean that there is no intra-abdominal segment of the lower esophageal sphincter. The length of the intra-abdominal segment is determined by the insertion of the phreno-esophageal membrane, and even in patients with a large hiatal hernia, intra-abdominal pressure can be exerted on the sphincter through the hiatus. Patients with esophagitis frequently had an isolated defect in their intra-abdominal segment, however, but there were others in the same group of patients with a normal intra-abdominal segment but a short overall length of the sphincter and a defective total sphincter pressure vector volume.

Based on the three-dimensional sphincter image, can we predict in whom the antireflux procedure is going to fail? We have not gone that far yet, and I do not know whether this method will be able to give us these predictors. The four patients in whom the antireflux procedure failed to restore esophageal acid exposure to normal all had a breakdown of the repair. We assume that all of these patients had a sphincter pressure vector volume immediately after the operation. But the repair broke down over the course of time mostly because of technical factors, and the three-dimensional image was destroyed.

Dr. Allgower mentioned the anatomic studies by Dr. Lieberman, of which I am well aware because I am now working in the same department as Dr. Lieberman. She is pleased to see that our three-dimensional manometric images of the lower esophageal sphincter correspond very well with the anatomic equivalent of the lower esophageal sphincter that she has identified on the dissecting table.

What is the benefit of doing three-dimensional manometry if we can identify the patient with increased esophageal acid exposure with esophageal pH monitoring alone, and what are our indications for antireflux surgery? Manometry of the lower esophageal sphincter is necessary to identify those patients with increased esophageal acid exposure who will benefit from antireflux surgery.

Increased esophageal acid exposure can be caused by a defective lower esophageal sphincter, gastric causes or poor esophageal clearance. In our opinion an antireflux procedure should be performed only in these patients in whom increased esophageal acid exposure is due to a defective lower esophageal sphincter. Those are the only patients who really do benefit from antireflux surgery. It is particularly important to identify those patients with a mechanical defective sphincter who have not yet developed mucosal injury. This is because we know that, with progression of mucosal injury to esophagitis, stricture, and Barrett's esophagus, the motor function of the esophageal body deteriorates and the results of an antireflux procedure becomes less predictable. Three-dimensional manometry of the lower esophageal sphincter is superior to standard manometry, particularly in these patients.

The group of Dr. Donahue and the late Dr. Bombeck, who first presented three-dimensional sphincter images, are proponents of the rapid pullback technique to obtain the image. Because our validation study has shown superiority of the stepwise pullback in discriminating patients with and without reflux disease, we decided to use the stepwise pullback routinely. The stepwise pullback may be superior to the rapid pullback because it allows us to identify the respiratory invasion point and the intra-abdominal segment of the sphincter, which is a very important factor of the antireflux mechanism.

Could asymmetry of the sphincter contribute to its incompetence? I think yes, it does. At the recent Western Surgical Association meeting, we presented a paper on asymmetry of the lower esophageal sphincter. By evaluation of sphincter asymmetry, we can identify another 10% or 12% of patients who have a normal sphincter pressure vector volume but a markedly asymmetric sphincter. We believe that these patients have reflux because of the asymmetry of their lower esophageal sphincter.