**Alimentary tract**

**Is yield pressure at the cardia increased by effective fundoplication?**

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**SUMMARY** Yield pressure at the cardia was measured before and after fundoplication in 10 patients; five had a Nissen fundoplication and five a Belsey mark IV procedure. Surgery was considered successful if oesophagitis healed or if 24 hour pH monitoring returned to normal. There was a marked rise in yield pressure in all eight patients with a successful operation. Yield pressure remained low in two patients in whom oesophagitis persisted.

It is not obvious why fundoplication prevents reflux successfully in 90% of patients. The Nissen and Belsey mark IV procedures are the operations most commonly used and have many similarities, differing principally in the extent of fundoplication. In the Nissen this is complete whereas in the Belsey about two thirds of the oesophagus is encircled. When Nissen described his procedure it was generally believed that hiatus hernia and reflux were synonymous. His fundoplication was intended to increase the bulk of the upper stomach in the abdomen, making it difficult for the stomach to herniate into the chest. We know now that reflux can exist in the absence of hiatus hernia and that the Nissen operation works just as well when the whole stomach has herniated into the chest.

Many studies have shown that lower oesophageal sphincter (LOS) pressure is increased after a fundoplication and it is often suggested that the wrapped fundus contracts and squeezes the sphincter. These studies always contain patients whose reflux is cured while their LOS pressure is unaltered or even reduced. Bancewicz *et al* were recently unable to find a correlation between a rise in LOS pressure and effective surgery and suggested that the observed alterations in pressure were merely an artifact of the operation. In fact in the dog fundoplication prevents reflux even after resection of the lower oesophageal sphincter and it has been shown in isolated cadaver stomachs that the Nissen and Belsey fundoplications will reduce or very effectively prevent reflux of water into the lower oesophagus. Therefore, fundoplication is successful when there is no LOS pressure at all.

Since the demonstration of a high pressure zone in the lower oesophagus in 1956 lower oesophageal sphincter pressure has been used to assess the competence of the antireflux mechanism despite the poor correlation between it and oesophageal reflux. Older studies which identified other components of the antireflux mechanism have tended to be forgotten. These include the anatomical fundus of the stomach and the gastric sling fibres. It is possible that the barrier against reflux has several components and that the lower oesophageal sphincter may not be the most important.

We have shown that the yield pressure of the cardia – that is, pressure at which the cardia yields in response to rising intragastric pressure – can easily be measured at endoscopy and is significantly lower in patients with oesophagitis compared with controls. We believe that yield pressure represents the combined effect of all the antireflux mechanisms and is not simply another test of lower oesophageal sphincter competence. If this is the case then yield pressure should always be increased after successful antireflux surgery whereas previous studies have shown that LOS pressure is not.

In this study we have measured yield pressure...
before and after Nissen or Belsey mark IV fundoplication. Surgery was considered successful if oesophagitis healed or 24 hour pH monitoring returned to normal.

**Methods**

**Patients**

Ten patients were studied: five had a Nissen fundoplication and five a Belsey mark IV procedure. Patients were endoscoped at intervals up to one year before and after surgery when yield pressure was recorded and the presence of oesophagitis or Barrett’s mucosa was noted. (To avoid subjective bias oesophagitis was defined as the presence of linear streaking or frank ulceration.) Twenty four hour oesophageal pH monitoring was carried out preoperatively in one patient with Barrett’s mucosa without oesophagitis and one patient with alkaline reflux without visible mucosal damage. It was repeated in both these patients postoperatively.

Yield pressure was determined using the method previously described. In this technique resting gastric pressure (RGP) is measured at the start of the endoscopy before air is insufflated. The endoscope is then retroverted to view the cardia and air insufflated until the cardia is seen to open. The pressure at which this occurs (OP) is noted and yield pressure taken as OP – RGP. We now measure pressure using a transducer in the tip of a catheter small enough to pass along the biopsy channel of the endoscope (Gaeltec Ltd) connected to a recording box which gives an instant visual reading (Lower Oesophageal Sphincter Identifier, Synectics Medical).

Twenty four hour oesophageal pH monitoring was performed using a radiopill tethered 5 cm above the mucosal junction (Oxford Medical Systems Ltd). Results are expressed as a frequency duration index (FDI). This is calculated as the sum of the number of episodes per hour (frequency) and minutes of exposure per hour to pH of less than four (duration) for the whole 24 hour period. An episode is defined as a fall of at least 2 pH units from the current baseline to below pH 4 within 18 seconds and the duration is the time taken to return to pH 4.

**Results**

Yield pressure rose dramatically in eight patients (five Nissen, three Belsey Mark IV) (Figure). In all patients there was objective evidence that the operation had successfully prevented reflux. In six patients this was provided by healing of oesophagitis. One patient’s oesophagus was lined with Barrett’s mucosa without oesophagitis preoperatively and in this case pH monitoring, which was abnormal pre-operatively (FDI 21·8) returned to normal (FDI 0). One patient had alkaline reflux without mucosal damage and the total time her oesophagus was exposed to pH>7 fell from 69·5% of the 24 hour period preoperatively to 29·1% postoperatively.

Yield pressure in two patients remained at zero after surgery—that is, the cardia opened as soon as air insufflation began. Both had had a Belsey Mark IV procedure. One of these had not had a preoperative yield pressure recorded but failure of surgery was confirmed by the persistence of oesophagitis with later stricture formation. The other patient’s yield pressure was unaltered by the operation and his oesophagitis persisted.

**Discussion**

This study shows for the first time that yield pressure must be increased if fundoplication is to prevent reflux, supporting the concept that yield pressure represents the combined effect of all the antireflux mechanisms. Lower oesophageal sphincter pressure is clearly one of these but other studies have shown
that it can remain low even though the operation is successful. Reinforcement of another component of the antireflux mechanism must therefore be responsible for the rise in yield pressure after fundoplication.

A possible candidate is the gastric sling fibre mechanism. These fibres are a condensation of the oblique muscle coat of the stomach and run parallel to the lesser curve and around the cardia. Anatomists have been aware of them for centuries. Contraction of these fibres could interrupt the transmission of tension in the gastric wall which is produced by gastric distension. This would help prevent reflux because the transmission of gastric wall tension to the lower oesophagus tends to pull it open. There is experimental evidence to support this role for the sling fibres. If they are divided in the dog at laparotomy there is a striking fall in yield pressure. Conversely, if their action is mimicked by a circumferential suture placed around the isolated dog stomach a few centimetres below the cardia yield pressure rises as the circumference of the ligature is decreased.

It is not difficult to visualise a fundoplication affording the same protection to the lower oesophagus. This would explain how the operation reduces reflux in the cadaver stomach where there is no lower oesophageal sphincter tone. Although the author of this study felt that fundoplication provided a flap valve there is no evidence of such a valve in postoperative patients at endoscopy when the stomach is inflated with air and the cardia viewed with the retroverted endoscope.

Another surgical procedure that effectively prevents reflux is the insertion of an Angelchik prosthesis. Lower oesophageal sphincter pressure is often increased after this operation also, but once again reflux may be prevented without a rise in sphincter pressure. In fact the operation may be successful even though the prosthesis encircles the stomach well away from the cardia. Although we have not studied patients with these prostheses we would confidently predict that yield pressure would be increased by their insertion and believe that, like fundoplication, they act principally by interrupting the transmission of gastric wall tension to the lower oesophagus, protecting the sphincter in the same way that the sling fibres might do.

Although the numbers are small we have shown that successful fundoplication depends on increasing yield pressure. Most surgeons agree that successful surgery depends on achieving the correct tightness of the fundal wrap; if it is too tight dysphagia and gas bloat result, if too loose reflux is not prevented. With further study it should be possible to define a normal range for postoperative yield pressure. Measurement of yield pressure during surgery might then enable the surgeon to assess accurately the tightness of the fundoplication.

References


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