

Dysphagia and Gastroesophageal Junction Resistance to Flow Following Partial and Total Fundoplication

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Abstract

Background Esophageal peristalsis and basal gastroesophageal junction (GEJ) pressure correlate poorly with dysphagia. **Aim** To determine intraluminal pressures that reflect GEJ function and to determine manometric correlates for dysphagia before and after fundoplication.

Methods The relationships between maximal intrabolus pressure, residual GEJ relaxation pressure and peak peristaltic pressure for water swallows were determined in normal volunteers and patients with reflux disease before and after fundoplication. GEJ anatomy was assessed by radiological, endoscopic and surgical criteria, whilst dysphagia was measured with a validated composite dysphagia score.

Results Dysphagia was significantly associated with lower peak peristaltic pressure in the distal esophagus and the presence of a hiatus hernia preoperatively, as well as higher residual pressure on GEJ relaxation postoperatively. Peak distal peristaltic pressure and residual GEJ relaxation pressure were predictors of intrabolus pressure after total fundoplication ($p < 0.002$). Residual GEJ relaxation pressure was four times higher after 360° fundoplication ($N=19$) compared to 90° fundoplication ($N=14$, $p < 0.0001$). Similarly, intrabolus pressure was elevated 2.5 times after 360° fundoplication and nearly doubled after 90° fundoplication and both were significantly different from controls ($N=22$) and reflux disease patients ($N=53$, $p < 0.0001$).

Conclusions Gastroesophageal junction impedance to flow imposed by fundoplication is associated with dysphagia when there is suboptimal distal esophageal contraction strength and relatively high residual GEJ relaxation pressure.

Keywords Gastroesophageal junction · Manometry ·
Dysphagia · Reflux disease · Hiatus hernia ·
Laparoscopic fundoplication

Abbreviations

GEJ Gastroesophageal junction

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Introduction

Laparoscopic fundoplication has a well-proven antireflux effect, but outcomes are sometimes marred by postoperative dysphagia, gas bloat and an inability to belch.^{1,2} These side effects are either the result of restricted GEJ movement or altered gastrointestinal tract function secondary to fundoplication. Fundoplication certainly imposes a restriction to GEJ opening, resulting in an impedance to flow that is not normally present³ and this restriction remains during swallowing and transient sphincter relaxations.⁴

Objective measurements of GEJ function hold the key to better understanding of the mechanics of antireflux procedures and for minimising adverse outcomes. GEJ relaxation during swallowing and transient sphincter relaxations has two components: relaxation of the intrinsic or lower esophageal sphincter and focal inhibition of the diaphragmatic crura during inspiration. Normally, these relaxations significantly reduce intraluminal pressure within the GEJ and frequently abolish it completely. The pattern of GEJ relaxation is often altered by antireflux surgery, rendering it incomplete. This incomplete GEJ relaxation, which can be detected by manometry, reflects impedance to flow at the GEJ and has been linked with dysphagia after fundoplication.⁵ Another objective measure, intrabolus pressure, is generated when a swallowed bolus is compressed between the driving force of the oncoming peristaltic esophageal contraction against the pressure generated by the GEJ (Fig. 1).^{6,7} Thus, intrabolus pressure reflects both GEJ and esophageal body function. Intrabolus pressure is most pronounced in the distal esophagus and is greatly increased by esophageal outflow restriction in experimental animals and during elevation of intragastric pressure by abdominal compression in humans.^{6–8} In the clinical setting, total fundoplication and possibly hiatal hernia alter intrabolus pressure.^{5,9}

Dysphagia is an intriguing symptom that is experienced prior to surgery by some patients with reflux disease in the

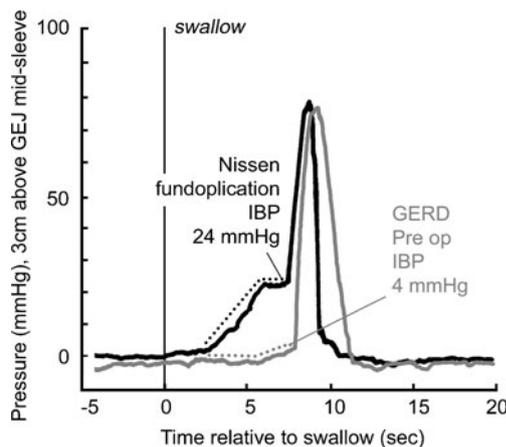


Fig. 1 Distal esophageal intrabolus pressure (IBP) during esophageal peristalsis in the same individual before and after 360° fundoplication. Intrabolus (or distal esophageal ramp) pressure appears as a steadily increasing pressure rise (ramp) with a plateau, which precedes the upstroke of pressure generated by the esophageal peristaltic contraction.^{34–36} The plateau itself occurs as the esophagus dilates to accommodate the compressed bolus as it is propelled distally by advancing peristalsis.³⁴ The plateau of the intrabolus pressure ends when the pressure in the bolus and the peristaltic contraction equals or exceeds the pressure within the GEJ (exceeds residual GEJ relaxation pressure).^{6,37,38} The dynamic change in intraluminal pressure just above and within the GEJ is accompanied by relaxation and opening of the GEJ, a drop in pressure in the direction of flow occurs and the bolus flows into the stomach.^{6,37,38}

absence of stricture and also by some patients after surgery. The physiology of both preoperative and postoperative dysphagia is poorly understood. Objective physiological studies with formal assessment of dysphagia are rarely conducted both pre- and postoperatively, because these studies require a high level of commitment by the patient and investigator. So although substantial data support the relevance of intrabolus pressure and the extent of GEJ relaxation as useful indicators of GEJ mechanics, there remains a lack of understanding of how dysphagia relates to these manometric measures.

To address this knowledge gap, we undertook this prospective study using standardised methods to evaluate dysphagia, intrabolus and residual GEJ relaxation pressures in normal volunteers, patients with reflux disease with and without hiatal hernia, and in a subgroup of patients before and 5 months after partial and total fundoplication.

Methods

Study Overview

Patients referred for esophageal function tests as part of a preoperative assessment were invited to participate. Patients who underwent fundoplication were reevaluated 5 months after surgery. Subjects were excluded if they had a primary motility disorder such as scleroderma and achalasia, atypical reflux symptoms, a large hiatus hernia (>5 cm) or previous antireflux surgery. Healthy age-matched control subjects were recruited from responses to advertisement in community newspapers (those experiencing heartburn or regurgitation at least weekly were excluded). Written informed consent was obtained from all subjects and the Human Research Ethics Committee of the Royal Adelaide Hospital approved the protocol.

Age, gender and body mass index were systematically recorded. Subjects completed a self-administered questionnaire to evaluate heartburn, regurgitation and dysphagia using visual analogue scales (VAS, 0–10 scale, 10=severe). A validated dysphagia composite score recorded difficulty with swallowing a range of foods of increasing viscosity (scale 0–45).¹⁰ Abnormal 24 h distal esophageal acid exposure ($\geq 4\%$ time pH<4), endoscopically evident erosive or ulcerative esophagitis¹¹ or Barrett's esophagus with intestinal metaplasia were considered proof of reflux disease. The presence, type and size of a hiatus hernia were noted from endoscopy and barium swallow¹² reports obtained from referring specialists. In addition, the primary surgeon categorised the size of hiatus hernia seen at operation as small (<2 cm), medium (2–5 cm) or large (>5 cm). Postoperatively, patients recorded their satisfaction with surgical outcome using a visual analogue scale (0–10).

Manometric Technique

Esophageal manometry was performed using a water perfused manometric catheter with a 6 cm sleeve (3.5 mm diameter Dentsleeve International, Mississauga, Canada), which was introduced transnasally to the stomach. The catheter was taped into position so the sleeve was across the GEJ, with six proximal side holes spaced 5 cm apart and a distal side hole for intragastric pressure. The manometric recording system digitised pressures at 40 Hz (Gastromac v3.3.5.3, Neomedix Systems, Sydney Australia).

H₂ receptor antagonists were withheld for 48 h and proton pump inhibitors were ceased 5 days prior to testing. After a 6-hour fast, subjects underwent manometry using a standard protocol (supine, 5-min rest period then ten 5-mL water swallows, each 30 s apart).

Data Analysis

Esophageal primary peristaltic success was recorded as the percentage of complete peristaltic sequences (esophageal peak pressure ≥ 10 mmHg above esophageal end-expiration baseline for at least four of five esophageal channels). The median basal end-expiratory GEJ pressure referenced to end-expiratory intragastric pressure was recorded from the rest period. GEJ length was determined as the distance between the level (cm) at which pressure rose above gastric pressure (≥ 2 mmHg) to GEJ lumen pressure and the level when pressure fell (≥ 2 mmHg) to esophageal basal pressure.

The following end-expiratory pressures were measured for each *individual successful* water swallow: distal esophageal peristaltic pressure (8 cm and 3 cm above the GEJ, mmHg); maximal intrabolus pressure prior to the peristaltic pressure wave upstroke (3 cm above the midpoint of GEJ, mmHg); residual GEJ (nadir) pressure as a result of swallow induced GEJ relaxation (residual GEJ relaxation pressure, mmHg); and basal GEJ pressure within 5 s prior to swallow initiation (mmHg). Failed swallows (contraction peak pressure ≤ 10 mmHg for two or more adjacent esophageal channels); swallows with synchronous esophageal pressure waves and double swallows were excluded, because without successful peristalsis there is insufficient force to compress the bolus against GEJ pressure.

Laparoscopic Fundoplication

Patients with proven reflux disease who were suitably fit were offered laparoscopic fundoplication. The type of fundoplication undertaken was determined by informed patient preference. For total fundoplication, a loose 2-cm long 360° wrap was constructed over a 52Fr intraesophageal bougie, without division of the short gastric vessels.¹³

A partial fundoplication included a posterior esophagopexy to the right hiatal pillar, fixation of a length of esophagus within the abdomen, recreation of the angle of His, and construction of an anterior 90° fundoplication that covered the left anterolateral intraabdominal esophagus.^{13,14} In both procedures, the esophageal hiatus was routinely repaired with posterior sutures.

Statistical Methods

Data analysis was performed on both a per individual swallow and per subject basis.

Subject Analysis Normally distributed data (mean \pm SEM) were compared between groups using independent samples *t*-tests and one-way ANOVA models, whilst continuous data that were not normally distributed (median, interquartile range IQR {Q1–Q3}) were compared using Mann–Whitney and Kruskal–Wallis tests. Fisher exact tests were used to analyse simple contingency tables. Paired pre- and post-fundoplication data were analysed using a Wilcoxon signed rank tests for continuous data and McNemar tests for proportions. Predictors of the presence of dysphagia amongst patients were assessed using logistic regression models.

Individual Swallow Analysis Intraluminal pressures were analysed using linear mixed effects models. Patient identity number was entered as a random effect to adjust for dependence due to a subject being in more than one group (pre- and postsurgery) and for multiple swallows (ten swallows per subject). Where required, outcome data were log transformed prior to analysis, and then back-transformed to give estimates on the original scale (median value). Linear mixed effects were used to compare intraluminal pressures across groups (healthy controls, reflux patients \pm hiatus hernia and patients for two types of fundoplication) and to identify predictors of intrabolus pressure.

All statistical calculations were performed using Instat (version 3.0b, GraphPad Software Inc., San Diego, California) and SAS (version 9.2, SAS Institute Inc., Cary NC, USA). Significance was accepted for *p* values less than 0.05.

Results

Study Population

Twenty-five healthy control subjects met entry criteria; however, three were excluded because cardiac compression obscured intrabolus pressure. Of 65 patients with suspected reflux disease, 12 patients were excluded because of large

hiatus hernia ($n=2$), poor esophageal motility ($n=2$; $<50\%$ primary peristalsis) or lack of proof of reflux disease ($n=8$). Patients with reflux disease were divided into those with a hiatus hernia (reflux HH, $n=24$) and those without (reflux noHH, $n=29$). Erosive or ulcerative esophagitis was found in 30 patients and Barrett's esophagus in nine patients.

From the above group of 53 patients with reflux disease, antireflux surgery was the preferred treatment for 33 patients, whilst 20 patients chose continuation of medical therapy with an option for further review if required. Fundoplication was either a partial 90° anterior fundoplication (14 patients) or total 360° fundoplication (19 patients). Female patients tended to prefer a partial fundoplication, whilst many males chose a total fundoplication (Table 1). Prior to surgery, 17 of 33 patients had a sliding hiatus hernia <5 cm in size with operative confirmation in 13 patients (76% concordance).

Surgery significantly reduced reflux symptoms in all patients. After total fundoplication, more patients were free of reflux symptoms compared with partial fundoplication (89% *cf.* 50% heartburn free and 75% *cf.* 43% regurgitation free, respectively) and were slightly more satisfied with their surgery (median VAS 10.0{9–10} vs. 8.5{7–9}, respectively $p=0.05$).

Prevalence of Dysphagia

The prevalence of dysphagia in patients with reflux disease, as well as patients before and after fundoplication is shown in Table 2. Five months after fundoplication, no patient experienced severe dysphagia (VAS score $>7/10$). New onset dysphagia was reported after total and partial fundoplication (9/19 patients vs. 2/14 patients, respectively, $p=0.06$) (Fig. 2), with a small but significant increase in severity of dysphagia following total fundoplication (Table 2).

Table 1 Demographic data

	Healthy control $N=22$	Patients with reflux disease $N=53$	P value	Fundoplication anterior 90° $N=14$	Fundoplication Nissen 360° $N=19$	P value
Age ^a , years	43 (24–74)	47 (18–77)	0.26 ^c	54 (31–78)	47 (25–71)	0.06 ^c
Gender, M: F	10: 12	28: 25	0.06 ^d	2: 12	17: 2	<0.0001^d
BMI ^b , kg/m ²	24.9 \pm 0.8	28.8 \pm 0.7	0.001^c	25.4 \pm 1.3	28.9 \pm 0.8	0.02^c
BMI <25	12	9		8	1	
25–29	7	21		3	9	
30–34	3	16		2	9	
≥ 35	0	7		1	0	
Height ^b , cm	170.6 \pm 2.6	170.4 \pm 1.4	0.93 ^c	163.7 \pm 1.9	174.8 \pm 1.8	0.0003^c

^a Data are mean (range)

^b Data are mean (\pm SEM)

^c Independent t -test

^d Fishers's exact test

Significant differences shown in bold type

Measures of GEJ Compliance

Residual Pressure during Swallow Induced GEJ Relaxation Both types of fundoplication significantly raised residual GEJ relaxation pressure; however, the pressure elevation was four times higher after 360° fundoplication compared to 90° fundoplication (Table 3; Fig. 3).

Intrabolus Pressure Following 90° fundoplication, intrabolus pressure nearly doubled and more than doubled (about 2.5 times) in 360° fundoplication patients (Table 3). For the 360° fundoplication group, 95% of patients had an intrabolus pressure less than 15 mmHg preoperatively and greater than 15 mmHg postoperatively (Fig. 3).

Resting Gastroesophageal Junction Pressure Table 3 shows that, compared with controls, GEJ resting pressure was significantly lower in reflux disease patients and significantly elevated following 360° fundoplication but not following 90° fundoplication (Table 3).

Length of Gastroesophageal Junction Pressure The manometric length of the GEJ increased after 360° fundoplication (median{IQR} 2{2–3} cm, vs. 4 {3–4} cm, $p=0.03$), but not significantly after 90° fundoplication (3{2–3.8} cm vs. 3.5{3–4} cm, $p=0.19$, pre- op vs. postop respectively).

Relationships Amongst Intraluminal Pressures

In all groups there was a positive correlation amongst distal esophageal peak pressure, GEJ resting pressure, residual GEJ relaxation pressure and intrabolus pressure (Fig. 4, reflux disease patients not shown). Distal esophageal

Table 2 Prevalence of dysphagia

Group	N	Dysphagia = Yes	Dysphagia for solids only	Dysphagia for liquids score (0–10)	Dysphagia for solids score (0–10)	Composite dysphagia score (0–45)
Patients with reflux disease	53	22 (42%)	11 (21%)	0 {0–0}	0 {0–2}	0 {0–12}
Subset of reflux patients prior to fundoplication	33	12 (36%)	7 (21%)	0 {0–0}	0 {0–2}	0 {0–5}
90° fundoplication						
Preop	14	8 (57%)	4 (29%)	0 {0–1}	0 {0–4}	3 {0–15}
Postop	14	10 (71%)	4 (29%)	0 {0–1}	0 {0–3}	4 {0–13}
360° fundoplication						
Preop	19	4 (21%)*	3 (16%)	0 {0–0}	0 {0–0}	0 {0–0}†
Postop	19	13 (68%)*	7 (37%)	0 {0–1}	1 {0–2}	4 {0–12}†

Data are number (%) or median {IQR}

* $p=0.003$ McNemar test

† $p=0.02$ Wilcoxon matched-pairs signed-ranks test

peristaltic pressure was found to be a predictor of intrabolus pressure in all groups except the 90° fundoplication group. For example, in control subjects for every 1 mmHg increase in distal esophageal peak pressure, there was an estimated 0.03 mmHg increase in intrabolus pressure (Table 4).

Following 360° fundoplication, residual GEJ relaxation pressure was a predictor of intrabolus pressure in addition to distal esophageal peak pressure so that for every 1 mmHg increase in residual GEJ relaxation pressure, there was an estimated 0.3 mmHg increase in intrabolus pressure and for every 1 mmHg increase in distal esophageal peak pressure, there was an estimated 0.04 mmHg increase in intrabolus pressure.

Association of Gastroesophageal Junction Anatomy and Function with Dysphagia

Dysphagia in Patients with Reflux Disease In patients with reflux disease, greater distal esophageal peak pressure was

associated with a reduced likelihood of dysphagia (odds ratio=0.97, 95% CI 0.95–1.00, $p=0.02$). Reflux HH patients were far more likely to experience dysphagia than reflux noHH patients (odds ratio=0.27, 95% CI 0.09–0.86, $p=0.03$). These reflux HH patients were also significantly older than reflux noHH patients (52.9 {27–77} years vs. 42.8 {18–69} years, $p<0.01$) and experienced significantly greater regurgitation (median score 7.5 {5–10} vs. 5 {3–8}, respectively, $p=0.01$).

Dysphagia after Fundoplication After partial fundoplication, greater distal esophageal peak pressure was associated with a reduced likelihood of dysphagia (odds ratio=0.94, 95% CI 0.89–1.00, $p=0.049$), but this finding was not significant for total fundoplication ($p=0.36$).

To further interpret manometric data in the light of pre- and postoperative dysphagia, data for both types of fundoplication were pooled and patients were grouped according to their pattern of dysphagia. There were four groups, patients with (1) no dysphagia pre- or 5 months postoperatively; (2) dysphagia before and after fundoplication; (3) dysphagia postop only and (4) dysphagia preop only. Analysis of intraluminal pressures by dysphagia status (Table 5) showed patients with ‘post operative dysphagia only’ had higher mean postoperative residual GEJ relaxation pressure. Patients with ‘no dysphagia pre or postoperatively’ had higher mean postoperative distal esophageal peak pressure than patients with dysphagia before and after surgery. Patients with new onset postop dysphagia had significantly greater increase in residual GEJ relaxation pressure than patients with dysphagia before and after surgery (7.4 ± 1.7 mmHg *cf.* 2.5 ± 0.5 mmHg, $p=0.046$). Postoperative residual GEJ relaxation pressure correlated with increased dysphagia for solids after fundoplication (linear regression $r^2=0.17$, $p=0.02$).

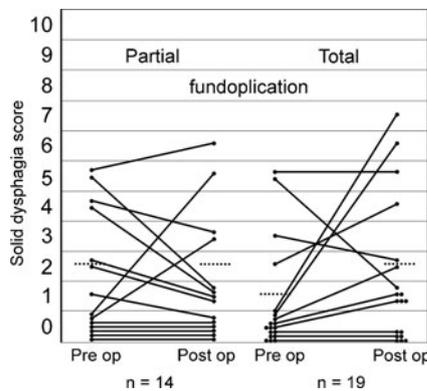


Fig. 2 Dysphagia for solids score before and after fundoplication (horizontal bar is mean value)

Table 3 Distal esophagus and gastroesophageal junction pressures

Pressures with water swallows ^a	Healthy controls <i>N</i> =22	Reflux patients, no hiatus hernia <i>N</i> =29	Reflux patients, with hiatus hernia <i>N</i> =24	Fundoplication anterior 90° <i>N</i> =14	Fundoplication Nissen 360° <i>N</i> =19	<i>P</i> value
Esophageal peak pressure 8 cm above GEJ, mmHg	90.1±8.1	72.1±6.2	71.1±6.9	68.6±9.4	77.5±8.3	0.33
Intrabolus pressure, mmHg	9.6±0.7	9.9±0.6	8.4±0.7	15.1±1.6 [†]	23.5±1.3 [†]	< 0.0001
GEJ basal pressure immediately prior to swallow, mmHg	17.4±1.4 [§]	10.1±1.3 [§]	7.3±1.3 [§]	16.8±2.5 [§]	26.0±2.1 [§]	< 0.0001
Residual GEJ relaxation pressure, mmHg ^b	0.7	0.4	0.3	1.5 [*]	6.0 [*]	< 0.0001
Pressures recorded during rest						
GEJ basal pressure during rest period, mmHg	18.3±1.7 ^o	7.9±1.4 ^o	5.5±1.6 ^o	13.2±2.0 ^o	25.7±1.7 ^o	< 0.0001

Data are mean (±SEM). *GEJ*, gastroesophageal junction

Analysed using linear mixed effects model, *P* values were adjusted for multiple comparisons using the step-down Sidak method:

[†] *p*<0.006 compared with all other groups

[§] *p*<0.01 compared with all other groups, except: Reflux, HH were not significantly different from Reflux, noHH; 90° fundoplication were not significantly different from healthy controls

^{*} *p*<0.004 compared with all other groups

^o *p*<0.02 compared with all other groups, except: Reflux, HH were not significantly different from Reflux, noHH; 90° fundoplication were not significantly different from Reflux, no HH and healthy controls

^a Parameters determined for individual water swallows

^b Data are right skewed, log transformed, analysed using linear mixed effects model (data are median)

Significant differences shown in bold type

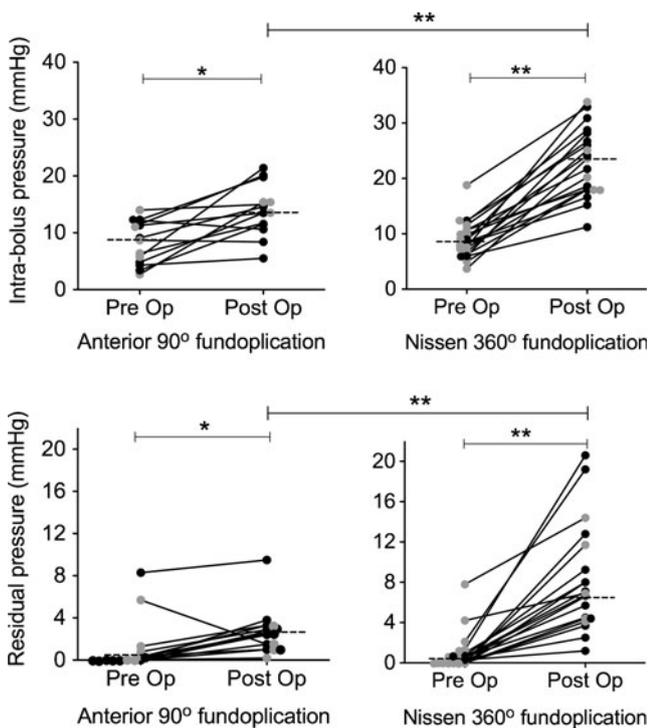


Fig. 3 Fundoplication significantly altered intrabolus pressure and residual GEJ relaxation pressure, with greater change after total fundoplication ($n=19$) than partial fundoplication ($n=14$) ($*p<0.01$, $**p<0.0001$). Median bar. Dysphagia status is shown as black dot dysphagia; gray dot, no dysphagia

Discussion

The major novel findings of this study are that dysphagia is linked with both suboptimal esophageal driving pressure pre- and postoperatively and the degree to which GEJ compliance is reduced by fundoplication. These findings are what we might expect intuitively and yet neither we,^{5,15} nor others^{16,17} have used a systematic approach to demonstrate these relationships.

Our study highlights that postfundoplication dysphagia is related to two things. First is the resistance to flow at the GEJ imposed by fundoplication. We found that a large increase in residual GEJ relaxation pressure was associated with *new onset* postop dysphagia. Further, we found that the circumferential extent of the fundal wrap significantly influenced intrabolus pressure and residual GEJ relaxation pressure. Many previous studies have concentrated on findings for one type of operation such as total fundoplication^{5,18,19} or inappropriately focused on GEJ resting pressure. GEJ relaxation can only be reliably recorded with a sleeve or with pressure sensors spaced at no more than 1 cm intervals.²⁰ Anderson et al.¹⁵ and Engstrom et al.¹⁶ used a catheter with a sleeve and also found these intraluminal pressures were elevated in proportion to the extent of fundoplication. However, these studies did not evaluate

patients preoperatively and postoperative findings were not interpreted in the light of preoperative dysphagia. In the present study, dysphagia was significantly associated with higher residual pressure on GEJ relaxation postoperatively.

Second, our study shows that dysphagia is related to suboptimal esophageal contractile strength. Preoperatively, patients with reflux disease and low distal esophageal contraction pressure were more likely to experience dysphagia. Patients who did not report dysphagia before or 5 months after surgery had significantly higher distal esophageal contraction pressure. Furthermore, distal esophageal peak pressure was a predictor of intrabolus pressure in all groups except following anterior 90° fundoplication, which is possibly a type II statistical error due to the small number of subjects in this group. Similarly, residual GEJ relaxation pressure was also a predictor of intrabolus pressure, although less consistently. This suggests that residual GEJ relaxation pressure during swallowing produces resistance to flow through the GEJ so that higher intrabolus pressure is required for flow to occur. A stronger distal esophageal contraction will generate greater bolus compression against the less compliant GEJ, evident as higher intrabolus pressure.

We propose that the esophagus adapts to increased GEJ resistance to flow by generating higher esophageal contraction pressures and that limits in this adaptive response may result in impaired bolus transit,^{6,19} and dysphagia.²¹ Scheffer et al.¹⁸ have proposed that greater esophageal contraction strength is necessary to overcome increased GEJ resistance after fundoplication. Our study takes this concept one step further, as our findings suggest that, independent of fundoplication, there is an inherent adaptive esophageal response mechanism to GEJ resistance that is a part of normal esophagogastric junction mechanics, since our study showed a positive correlation between intrabolus pressure, peristaltic amplitude and residual GEJ relaxation pressure in all the groups we studied.

The impact of fundoplication on the strength of esophageal peristalsis has often been debated with some studies showing fundoplication increases distal esophageal peak pressure,^{18,22–25} whilst others show a reduction or no change.^{17,21,26} These studies were confined to observations in fundoplication patients without any comparison with other patient groups or normal subjects. Our study shows that esophageal contractile strength varies according to the degree of GEJ resistance to flow in both unoperated and operated persons.

We also assessed how a hiatus hernia might influence dysphagia and GEJ compliance. A small hiatus hernia was associated with low intrabolus, basal and residual GEJ relaxation pressures and these patients were more likely to experience dysphagia. A recent study found that hiatal hernia patients with reflux symptoms and no dysphagia had

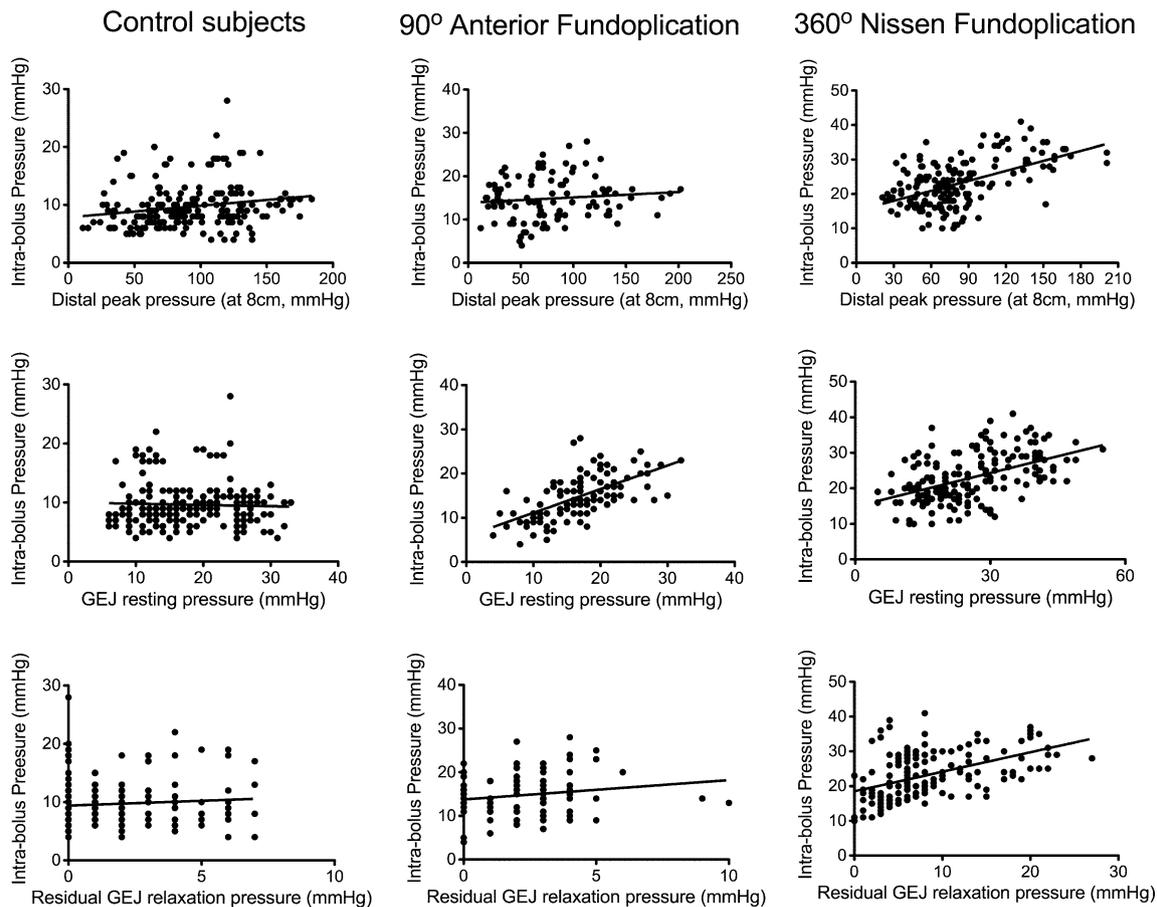


Fig. 4 Relationship of intraboli pressure with peristaltic esophageal body peak pressure (*above bolus*) and GEJ pressures (*below bolus*)

lower intraboli and residual GEJ relaxation pressure than hiatal hernia patients with dysphagia but no reflux.²⁷ Future studies need to assess whether the space the herniated stomach occupies in the hiatal canal may affect resistance to bolus transit through the GEJ and alter intraboli pressure.

There are some limitations to our study. Patients undergoing surgery were not randomised for the type of fundoplication and gender bias was evident. This bias is probably due to the information we provide routinely to patients about the risks of fundoplication, notably that a total fundoplication carries a greater risk of increased flatulence than a partial fundoplication.²⁸ Women seem to be more concerned about this risk than men. From a technical perspective, we measured the distensibility of the relaxed GEJ indirectly and so are unable to assess the impact of GEJ opening diameter on intraboli and residual GEJ relaxation pressure and the incidence of dysphagia. Previous studies have shown GEJ opening diameter during swallow-induced relaxation negatively correlates with intraboli pressure and is related to the radial extent of fundoplication.¹⁵ Our study findings are limited by the use of a water bolus that is well tolerated and safe, but may not emulate the conditions for the dysphagia most commonly

reported after fundoplication, namely dysphagia for solids. Further, we specifically excluded synchronous and nonpropagating esophageal contractions from the analysis. However, 91% of patients with reflux disease displayed $\geq 70\%$ primary peristalsis whilst 42% had dysphagia.

The future is bright for addressing some of these limitations with recently established high resolution manometry (HRM).²⁹ HRM evaluation with a similar protocol and incorporating recent innovations for assessment of esophageal motor function^{29,30} holds promise for better understanding of postoperative dysphagia, including the identification of individual patients at risk of this side effect. HRM studies combined with intraluminal impedance for recording bolus flow with a viscous or solid bolus is also a promising option.³¹ Further, a relative ‘new comer’, the functional luminal imaging probe (FLIP), looks promising as a tool for measuring distension in the GEJ.³²

Currently, the mechanical components of antireflux surgery, namely, hiatal repair and fundal wrap, cannot be separately identified with either a 6-cm sleeve sensor or the 1-cm spacing of pressure sensors in currently available solid state HRM catheters.³³ HRM needs to evolve further to enable even closer spatial arrangement of pressure

Table 4 Predictors of intrabolus pressure

Predictor	Controls <i>N</i> =22	Reflux patients, no hiatus hernia <i>N</i> =29	Reflux patients with hiatus hernia <i>N</i> =24	Anterior 90° fundoplication <i>N</i> =14	Nissen 360° fundoplication <i>N</i> =19
Intrabolus pressure (mmHg)					
A 1 mmHg rise in each parameter, predicts a rise in intrabolus pressure of:					
Distal esophageal peak pressure	0.03* (0.01)	0.02† (0.01)	0.01* (0.01)	0.002 (0.01)	0.04* (0.01)
GEJ pressure	0.02 (0.03)	0.01† (0.04)	0.07 (0.04)	0.08 (0.08)	0.01 (0.04)
Residual GEJ relaxation pressure	0.02 (0.14)	0.25* (0.12)	0.25 (0.19)	0.32 (0.24)	0.30† (0.09)

Data are estimated increase in intrabolus pressure per 1 unit increase in predictor (mmHg)

GEJ, gastroesophageal junction. Linear mixed effects model, data are coefficient (std error); significance level:

* *p*<0.001

† *p*<0.01

* *p*<0.05

Table 5 Dysphagia and intraluminal pressures after fundoplication

Postoperative data	Dysphagia none <i>N</i> =9	Dysphagia before and after <i>N</i> =11	Dysphagia postop only <i>N</i> =12	<i>P</i> value†
Distal esophageal peak pressure, mmHg	104.9±10.3*	63.0±10.0*	72.2±9.6	0.02 †
Intrabolus pressure, mmHg	20.1±2.2	16.0±2.0	22.1±2.0	0.11†
GEJ basal pressure, mmHg	20.0 (17–21)	15.0 (10–22)	20.0 (16–34)	0.33*‡
Residual GEJ relaxation pressure, mmHg	4.4 (3.3–8.0)	2.5 (1.4–4.8)*	7.0 (3.6–10)*	0.04 *‡

GEJ, gastroesophageal junction. Data mean ± SEM or median (IQR). Per subject analysis; pooled data for both 90° fundoplication (14) and 360° fundoplication (19); the group with preop dysphagia only (*N*=1) was excluded from the analysis

† ANOVA with Bonferroni posttest

* Kruskal–Wallis with Dunn's posttest, *p*<0.05 between the * two groups

‡ Significant differences shown in bold type

sensors (≤ 5 mm spacing) in the distal third of the catheter in the region used to record GEJ topographic pressures.

Conclusion

Our study establishes that intrabolus pressure and residual GEJ relaxation pressure are influenced by the extent of the fundoplication and that these are key manometric measures of GEJ compliance. In reflux disease, preoperative dysphagia is associated with suboptimal esophageal function (low distal esophageal driving pressure) and altered anatomy (hiatus hernia). Postfundoplication dysphagia is associated with reduced compliance of the GEJ caused by the new fixed component of the antireflux barrier, as well as low distal esophageal driving pressure. We propose the esophagus has an adaptive response for resistance to flow across the gastroesophageal junction and limits in this adaptive response result in failure of bolus transit and dysphagia.

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