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The Contribution of Intrabolus Pressure to Symptoms Induced by Gastric Banding

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ABSTRACT

Background & Aims: Mechanisms that ultimately lead to dysphagia are still not totally clear. Patients with laparoscopic gastric banding (LAGB) often complain about dysphagia, regurgitation and heartburn. Our aim was to evaluate the contribution of intrabolus pressure to symptoms of gastric banding.
Methods: This study investigated 30 patients with LAGB before and 3 months after conversion to Roux-en-Y gastric bypass (RYGB), evaluating symptoms with a 7-point-Likert-scale and esophageal peristalsis, esophageal bolus transit and intrabolus pressure changes using combined impedance-manometry.
Results: Conversion from LAGB to RYGB leads to a significant reduction in dysphagia (1.9 ± 0.4 vs. 0.0 ± 0.0; $p< 0.01$) and regurgitation (4.2 ± 0.4 vs. 0.1 ± 0.1; $p< 0.01$) symptom scores. For liquid swallows we found a modest but significant correlation between the intensity of dysphagia and intrabolus pressure ($r=0.11$; $p<0.05$) and the intensity of regurgitation and intrabolus pressure for viscous swallows ($r=0.12$, $p<0.05$) in patients with LAGB. There was a significant ($p< 0.05$) reduction in intrabolus pressure at 5 cm above LES before (liquid 10.6 ±1.0; viscous 13.5 ± 1.5) and after (liquid 6.4 ± 0.6; viscous 10.5 ± 0.9) conversion from LAGB to RYGB.
Conclusion: Current data suggest that intraesophageal pressure during bolus presence in the distal esophagus contributes to the development but not to the intensity of dysphagia and regurgitation.

Key words: dysphagia – intrabolus pressure – gastric banding (LAGB) – Roux-en-Y gastric bypass (RYGB) – esophageal impedance manometry.

INTRODUCTION

Esophageal dysphagia is a common reason for referral for specialized gastrointestinal investigations. Common causes of dysphagia include structural lesions (i.e. cancer, strictures, external compression), achalasia (i.e. primary esophageal motor disorder), esophageal motility abnormalities (i.e. esophageal spasms, ineffective esophageal motility), eosinophilic esophagitis and iatrogenic (post-surgery) conditions. The exact mechanisms and changes that ultimately lead to the sensation of swallowing difficulties have not been completely clarified: factors considered to play a role in development of dysphagia include contraction of the esophageal musculature, distension of the esophageal wall in a narrowed area or proximal of it, prolonged bolus presence in the esophagus and increased esophageal wall tension.

One common post-surgical problem is dysphagia induced by laparoscopic adjustable gastric banding (LAGB). Dysphagia induced by gastric banding is an interesting model for investigating the mechanisms leading to dysphagia, as the "lesion" causing esophageal stasis and distension is located distal to the esophagus. During LAGB the esophagus is not modified by the placement of the gastric band. Furthermore by conversion of the LAGB into a Roux-en-Y gastric by-pass (RYGB) the extrinsic compression is removed. Prior studies have documented changes in esophageal motility, mainly lower esophageal sphincter residual pressure (LESRP) and esophageal peristalsis and esophageal symptoms induced by LAGB [1]. On the other hand, bariatric surgery studies report improvement of esophageal symptoms (including dysphagia) following conversion from LAGB to RYGB. Thus, quantifying esophageal peristalsis and bolus transit in patients before and after conversion from LAGB to RYGB might provide additional insight in the pathogenesis of esophageal dysphagia.

Currently available combined impedance-manometry provides information on esophageal peristalsis and esophageal bolus transit during individual swallows. It also allows the identifying of the intraesophageal pressure changes during and prior to esophageal contractions and differentiate which changes occur while the esophagus is empty or filled with fluid content. Accordingly, we set up a study quantifying esophageal pressure changes observed before and after conversion from LAGB to RYGB and correlated these with changes in symptoms reported before and after conversion. Our hypothesis was that conversion from gastric banding to gastric bypass leads to improved peristalsis, bolus transit and a decrease in intrabolus pressure (IBP) and these changes parallel improvement of symptoms.

MATERIAL AND METHODS

Patients

The current study was a subset analysis of collected data of prospectively enrolled 30 patients with gastric banding who were planned to undergo gastric bypass surgery because of insufficient weight loss and disturbing symptoms. Patients underwent impedance-manometry (MII-EM) testing prior to and three months after conversion from LAGB to RYGB as part of a clinical trial investigating the reversibility of motility changes induced by gastric banding. Surgery was performed between 08/2006 and 11/2007 in the Surgery Department of the Canton's Hospital of St. Gallen, Switzerland. All patients gave their written informed consent.

Evaluation of symptoms

Patients were asked to rate the severity of gastroesophageal symptoms (nausea, abdominal pain, postprandial fullness, non-cardiac chest pain, heartburn, dysphagia, regurgitation and others) on a 7-point-Likert-Scale (0-no symptoms, 1-very mild symptoms, 2-mild symptoms, 3-moderate symptoms, 4-moderate to severe symptoms, 5- severe symptoms, 6-very severe symptoms) before and after conversion from gastric banding to bypass.

Esophageal function testing

Esophageal function was tested using combined multichannel intraluminal impedance and manometry (MII-EM) measurements. Principles and technical details have been previously published [2]. We used the 9-channel esophageal function testing catheter (Sandhill Scientific Inc, Highlands Ranch, CO) with five pressure sensors 5 cm apart and four impedance measuring segments positioned across the four proximal pressure sensors. This allowed pressure measurements in the lower esophageal sphincter (LES) and at 5, 10, 15 and 20 cm above the LES. Impedance measuring segments were positioned at 5, 10, 15 and 20 cm above the LES.

After fasting for four to six hours, the catheter was inserted transnasally through the esophagus into the patients' stomach. The most distal sensor was placed in the high-pressure zone of the LES. After that, patients received 10 liquid (i.e., 0.9% normal saline) and 10 viscous (i.e., standardized viscous solution; Sandhill Scientific Inc, Highlands Ranch, CO) swallows, each 30 seconds apart. Double swallows and dry swallows were not included in the analysis.

Swallows were considered manometrically (1) normal if the contraction amplitude exceeded 30 mmHg at 5 and 10 cm above the LES and the onset velocity was less than 8 cm/sec, (2) simultaneous if the contraction amplitude exceeded 30 mmHg at 5 and 10 cm above the LES and the onset velocity was greater or equal to 8 cm/sec and (3) ineffective if contraction amplitude at 5 or 10 cm above the LES was less than 30 mmHg. Swallows were considered to have complete bolus transit if they had bolus entry at 20 cm and bolus exit at 15, 10 and 5 cm above the LES and to have incomplete bolus transit if bolus did not exit the measuring segment at 15, 10 or 5 cm above the LES.

Intrabolus pressure (IBP)

The information collected by the impedance-channel was used to determine the start and end of IBP segment in the corresponding manometry channel. The beginning of IBP was set at the time of bolus entry (i.e. 50% drop from baseline to nadir of the intraluminal impedance as detected by the semi-automated software). For swallows where impedance detected bolus exit (i.e. recovery to 50% difference between baseline and nadir impedance), the end of IBP was set at the time of bolus exit. For swallows where impedance found bolus retention (i.e. no recovery to values greater than 50%), the end of IBP was set at the inflection point of the contraction wave. Intrabolus pressure was defined as average pressure in the esophagus during impedance detected bolus presence prior to the onset of contraction (Fig. 1).

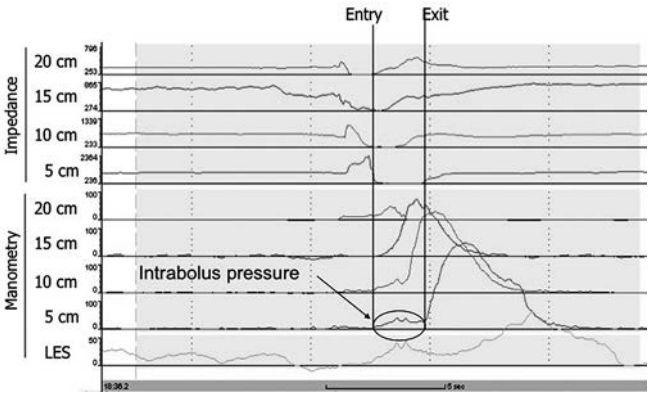

Fig. 1. Methodology of quantifying intrabolus pressure using combined impedance-manometry measurements. The design of the impedance-manometry probe allowed concomitant measurement of bolus presence (period elapsed between bolus entry and bolus exit) and pressure at 5, 10, 15 and 20cm above the lower esophageal sphincter (LES).

The average IBP for 10 saline and 10 viscous swallows was calculated in each patient, using the data collected 5 and 10 cm above LES.

Statistics

The per-patient average IBP at 10 and 5 cm above LES for liquid and viscous was compared before and after conversion with a paired *t*-test. Correlations between IBP and symptom ratings were assessed using Pearson correlation. For statistical significance alpha was set at 0.05.

RESULTS

Patients

A total of 30 patients, of whom 24 (80%) women, mean age 44 ± 2 years (range 27-61 years) completed the pre- and post-conversion evaluation.

Symptoms

Before conversion to RYGB, 11 (37%) patients indicated nausea, 8 (27%) abdominal pain, 16 (53%) postprandial fullness, 11 (37%) heartburn, 26 (87%) regurgitation, 9 (30%) non-cardiac chest pain, 14 (46%) dysphagia and 8 (27%) patients reported other symptoms while the gastric band was inflated. Three months after conversion, only 2 (7%) patients reported nausea, 10 (33%) abdominal pain, 11 (37%) postprandial fullness, 2 (7%) regurgitation and 3 (10%) other symptoms (Fig. 2). Esophageal symptoms significantly improved following conversion from LAGB to RYGB whereby epigastric symptoms remained the same.

Conversion from gastric band to gastric bypass significantly lowered dysphagia (1.9 ± 0.4 vs. 0.0 ± 0.0; p<0.01) and regurgitation (4.2 ± 0.4 vs. 0.1 ± 0.1; p<0.01) symptom ratings on the 7-point Likert scale. Before conversion, 14 (46%) patients reported any degree of dysphagia, whereas none reported dysphagia after conversion (p<0.001). A similar change could be demonstrated in patients with regurgitation: before conversion, 26 (86%) patients reported regurgitation, after conversion only 2 patients reported regurgitation (p<0.001).

Changes in manometric and impedance parameters

Changes in manometric and impedance parameters (contraction amplitude, bolus presence time, IBP, LES resting and LES residual pressure) were measured in liquid and viscous swallows 5 and 10 cm above LES before and after conversion to RYGB (Tables I and II); after conversion to RYGB a significant (p<0.001) decrease of LES resting pressure could be shown (30.3 ± 3.2mmHg vs. 18.5 ± 2.7 mmHg, p=0.001).

Table I. Pressure measurements (mmHg) during liquid swallows in patients before and after conversion from gastric banding (LAGB) to gastric bypass (RYGB).

	LAGB mean ± SEM	RYGB mean ± SEM	p-value
Contraction amplitude 10 cm (mmHg)	69.7 ± 6.7	71.8 ± 6.7	0.699
Contraction amplitude 5 cm (mmHg)	71.0 ± 9.8	73.2 ± 6.7	0.766
Bolus presence time 10 cm (sec)	5.4 ± 0.3	5.5 ± 0.4	0.737
Bolus presence time 5 cm (sec)	6.2 ± 0.4	6.2 ± 0.4	0.935
Intrabolus pressure 10 cm (mmHg)	7.6 ± 0.7	6.4 ± 0.5	0.121
Intrabolus pressure 5 cm (mmHg)	10.7 ± 1.0	6.5 ± 0.6	<0.001
LES resting pressure (mmHg)	30.0 ± 3.2	18.5 ± 2.7	<0.001
LES residual pressure (mmHg)	3.3 ± 1.0	1.5 ± 1.0	0.179

Changes in intrabolus pressure

There was a significant (p<0.05) difference in IBP at 5 cm above LES before and after conversion from gastric band to gastric bypass (Fig. 3). In saline bolus transit, there was a change in pressure from 10.6 mmHg ± 1.0 vs. 6.4 mmHg ± 0.6 in banding vs. bypass, and 13.5 mmHg ± 1.5 vs. 10.5 mmHg ± 0.9 in viscous bolus transit.

There was also a difference (Fig. 3) in IBP 10 cm above LES to 5 cm above LES (lower pressure at 10 cm above LES); and lower pressure in saline than in viscous swallows (p=ns).

In saline bolus transit, 7.5 mmHg ± 0.7 vs. 6.4 mmHg ± 0.5 in banding vs. bypass; in viscous swallows, 9.1 mmHg ± 0.9 vs. 7.8 mmHg ± 0.8 in banding vs. bypass.

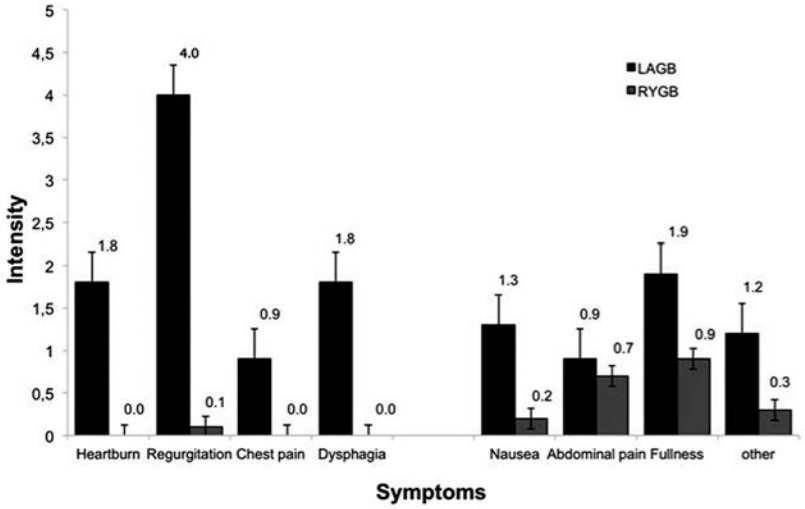

Fig. 2. Symptom intensity measured on a 7-point Likert scale* before and after conversion from gastric banding (LAGB) to gastric bypass (RYGB).
*Likert scale: 0-no symptoms, 1-very mild symptoms, 2-mild symptoms, 3-moderate symptoms, 4-moderate to severe symptoms, 5- severe symptoms, 6-very severe symptoms

Table II. Pressure measurements (mmHg) during viscous swallows in patients before and after conversion from gastric banding (LAGB) to gastric bypass (RYGB).

	LAGB mean ± SEM	RYGB mean ± SEM	p-value
Contraction amplitude 10 cm (mmHg)	73.8 ± 7.6	60.8 ± 5.6	0.02
Contraction amplitude 5 cm (mmHg)	67.7 ±7.7	68.3 ± 7.5	0.917
Bolus presence time 10 cm (sec)	5.3 ± 0.4	4.3 ± 0.2	0.187
Bolus presence time 5 cm (sec)	5.0 ± 0.5	3.9 ± 0.3	0.097
Intrabolus pressure 10 cm (mmHg)	9.0 ± 0.9	7.8 ± 0.8	0.285
Intrabolus pressure 5 cm (mmHg)	13.5 ± 1.5	10.5 ± 0.9	0.060
LES resting pressure (mmHg)	30.0 ± 3.2	18.5 ± 2.7	<0.001
LES residual pressure (mmHg)	4.4 ± 1.0	2.8 ± 0.9	0.306

Correlation between dysphagia/regurgitation and intrabolus pressure (IBP)

For liquid swallows we found a modest (r=0.11) but significant (p<0.05) correlation between intensity of dysphagia and IBP. Conversely there was no correlation (r=0.01; p=ns) between these parameters during viscous swallows.

Another significant but weak correlation (r=0.12, p<0.05) was found between the intensity of regurgitation and IBP in viscous swallows, whereas there was no correlation for liquid swallows. There was a significant difference in IBP in the distal esophagus (5 cm above the LES) before and after conversion to RYGB; this change was paralleled by an improved dysphagia after conversion.

DISCUSSION

Dysphagia is a frequent problem in patients with LAGB, caused by a mechanic obstruction (banding) below the lower esophageal sphincter (LES). To date, there are limited data about the contribution of IBP to the development of dysphagia. The current study investigated changes in IBP before and after conversion from LAGB to RYGB using combined impedance-manometry. We found a slight, but significant correlation between the intensity of dysphagia and IBP during liquid swallows and a modest but significant correlation between intensity of regurgitation and IBP during viscous swallows.

De Jong et al evaluated the changes in esophageal motility in 29 morbidly obese patients before and after LAGB [3]. Patients underwent a symptom assessment score (heartburn, regurgitation, nausea, belching, dysphagia) and a manometry at baseline, after 6 weeks and 6 months. There was a significant (p=0.003 and p=0.001, respectively) increase in LES pressure before (0.8 kPa (0.6-1.3), 6 weeks (1.3 kPa (0.9-1.9)) and six months (1.5 kPa (1.3-1.9)) after the operation. Dysphagia increased significantly (p=0.0001 and p=0.002, respectively) during follow-up leading the authors to conclude that LAGB causes an increase in LES pressure and dysphagia, but an overall decrease of reflux symptoms. Similar to us, Weiss et al [4] found in 43 patients a significant rise in LES resting pressure from preoperatively 10.9 mmHg (8-15.6) to 21 mmHg (11-26) postoperatively. Reflux symptoms decreased significantly, but no patient complained about dysphagia pre- or postoperatively.

Gamagaris et al investigated the longterm impact of LAGB on esophageal motility and pH-metry and identified a significant change in esophageal motility but without regarding dysphagia [1]. Manometry and 24 hour pH-evaluations were performed in 22 patients preoperatively and after 6 and 12 months. In patients with normal manometric parameters at baseline, there was a significant (p=0.014) increase in LES residual pressure after 6 months (3.9 ±2 vs. 8.9 ± 4mmHg). In patients with abnormal manometry at baseline, the LES pressure decreased significantly (p=0.011) from baseline to 12 months (98.7 ± 22 vs. 52.3 ± 24), without significant change in LES residual pressure. The results again concur with our findings showing a tendency of decrease in LESRP before and after conversion to RYGB (p=ns, Tables I and II).

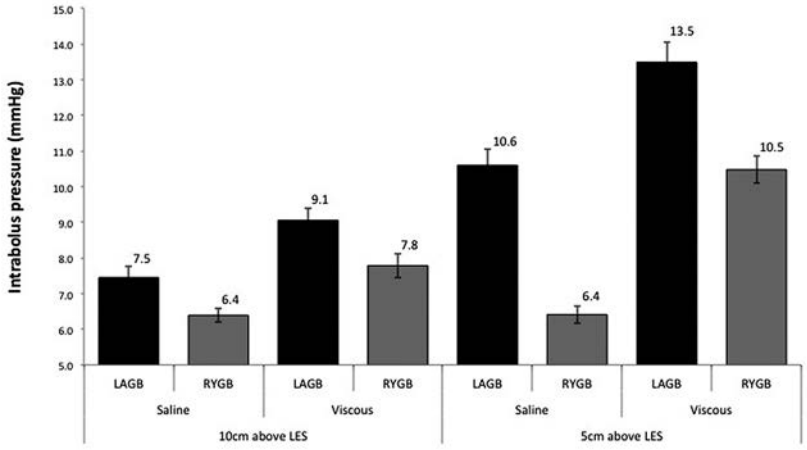

Fig. 3. Changes in intrabolus pressure at 10 and 5 cm above LES during liquid and viscous swallows before and after conversion from LAGB to RYGB.

Klaus et al concluded that in patients with preoperatively defective esophageal body motility, dysphagia (and regurgitation) worsened after LAGB [5]. They enrolled 587 patients who underwent LAGB. Preoperatively, 164 patients reported GERD symptoms (heartburn, dysphagia, regurgitation) which vanished after LAGB in 112 patients (68.3%). Still, 52 patients (31.7%) had ongoing or aggravated symptoms. In these patients, the dysphagia severity score increased after LAGB from 0.4 to 0.9 (p=0.03), without a significant difference of LES pressure pre- and postoperatively.

Burton et al compared 20 patients who underwent LAGB with obese controls, using combined video-fluoroscopy and high-resolution manometry [6]. Measurements were made with the optimal filling volume of the LAGB, 20% over and 20% under the optimal volume. With the optimal LAGB volume, there was no disturbance of esophageal peristalsis; increasing the volume affected the esophageal motility significantly. These data suggest that a stronger mechanical obstruction worsens esophageal motility leading to dysphagia.

In a prospective clinical trial over 12 years (median follow-up time of 79.9 ± 8.6 months), Naef et al evaluated the long term effect of LAGB on the esophagus [7]: 167 patients underwent manometry and barium swallows at baseline, every 3 months for the first year and then annually. At baseline, 56.9% had pathologic findings (GERD, hiatal hernia or gastritis). During follow-up, two thirds of all patients developed esophageal motility abnormalities and 25.5% showed a significant dilatation (35mm or more) of the esophagus. In 13% of them dilatation did not recover after band deflation, with persistent stage IV dilatation (achalasia-like dilatation). These results are similar to those reported by DeMaria et al, who found a marked dilatation of the esophagus in 17 of 36 patients after LAGB, 12 of these reporting dysphagia, vomiting, or severe reflux [8]. Interpreting these two studies, esophageal disorders with esophageal dilatation are common complications of LAGB, and in these patients, approximately two thirds experience dysphagia.

Fundoplication has similar effects on the esophagus as LAGB. Both lead to an iatrogenic mechanic outlet obstruction with consecutive esophageal dysmotility and symptoms (heartburn, dysphagia, regurgitation). There are conflicting data regarding dysphagia after Nissen fundoplication (NF) and Toupet fundoplication (TF). Others found higher rates of dysphagia in short term outcome (3 months) following fundoplication, but 6 or 12 months after fundoplication the difference diminished [9-11]: this is probably caused by an adaption of esophageal motility. Booth et al, however, found a significant difference (p=0.018) in dysphagia one year postoperatively with more prevalent dysphagia after Nissen fundoplication [12], caused by a major mechanic obstruction with a total fundal wrap.

One of the limitations of this study is the small number of patients. Data were retrospectively evaluated with already an associative approach for a correlation between symptoms before and after conversion. Although there was a correlation, there was still no proof for causality. Last but not least, current evaluations on dysphagia were based on a special patient cohort with LAGB and after conversion to RYGB; it is not clear if these data are also valid for patients with other causes of dysphagia.

CONCLUSION

To our knowledge no other study has evaluated, up to 2013, the contribution of intrabolus pressure to esophageal symptoms. The model of conversion from LAGB to RYGB offers the possibility to explore within the same patient changes in esophageal symptoms and intrabolus pressure. Many studies have focused on evaluating esophageal dysmotility and changes in LES residual or resting pressure after LAGB, but without connecting measurable changes with dysphagia.

Current data document a significant higher intrabolus pressure in the distal esophagus in patients with LAGB with a modest correlation to dysphagia and regurgitation. We interpret these data indicative for the contribution of intraesophageal pressure during bolus presence to the development, but not to the intensity of dysphagia and regurgitation in patients with gastric banding.

Conflicts of interest. None to declare.

REFERENCES

1. Gamagaris Z, Patterson C, Schaye V et al. Lap-band impact on the function of the esophagus. Obes Surg 2008; 18: 1268-1272
2. Tutuian R, Vela MF, Balaji NS et al. Esophageal function testing with combined multichannel intraluminal impedance and manometry: multicenter study in healthy volunteers. Clin Gastroenterol Hepatol 2003; 1:174-182.
3. de Jong JR, van Ramshorst B, Timmer R, Gooszen HG, Smout AJ. Effect of laparoscopic gastric banding on esophageal motility. Obes Surg 2006; 16: 52-58.
4. Weiss HG, Nehoda H, Labeck B et al. Treatment of morbid obesity with laparoscopic adjustable gastric banding affects esophageal motility. Am J Surg 2000; 180: 479-482.
5. Klaus A, Gruber I, Wetscher G et al. Prevalent esophageal body motility disorders underlie aggravation of GERD symptoms in morbidly obese patiens following adjustable gastric banding. Arch Surg 2006; 141: 247-251.
6. Burton PR, Brown W, Laurie C et al. The effect of laparoscopic adjustable gastric bands on esophageal motility and the gastroesophageal junction: analysis using high-resolution video manometry. Obes Surg 2009; 19: 905-914.
7. Naef M, Mouton WG, Naef U, van der Weg B, Maddern GJ, Wagner HE. Esophageal dysmotility disorders after laparoscopic gastric banding – an underestimated complication. Ann Surg 2011; 253: 285-290.
8. DeMaria EJ, Sugerman HJ, Meador JG et al. High failure rate after laparoscopic adjustable silicone gastric banding for treatment of morbid obesity. Ann Surg 2001; 233: 809-818.
9. Chrysos E, Tsiaoussis J, Zoras OJ et al. Laparoscopic surgery for gastroesophageal reflux disease patients with impaired esophageal peristalsis: total or partial fundoplication? J Am Coll Surg 2003; 197: 8-15.
10. Broeders JA, Sportel IG, Jamieson GG et al. Impact of ineffective oesophageal motility and wrap type on dysphagia after laparoscopic fundoplication. Br J Surg 2011; 98: 1414-1421.
11. Radajewski R, Hazebroek EJ, Berry H, Leibman S, Smith GS. Short-term symptom and quality-of-life comparison between laparoscopic Nissen and Toupet fundoplications. Dis Esophagus 2009; 22: 84-88.
12. Booth MI, Stratford J, Jones L, Dehn TC. Randomized clinical trial of laparoscopic total (Nissen) versus posterior partial (Toupet) fundoplication for gastro-oesophageal reflux disease based on preoperative oesophageal manometry. Br J Surg 2008; 95: 57-63.