ELSEVIER

European Journal of Cardio-thoracic Surgery 16 (1999) 266–272

EUROPEAN JOURNAL OF CARDIO-THORACIC SURGERY

www.elsevier.com/locate/ejcts

Clinical features and oesophageal motility in patients with tight fundoplication☆

Marcello Migliore*, Giulio Deodato

Section of General Thoracic Surgery, Department of Surgery, University of Catania, Catania, Italy

Received 22 October 1998; received in revised form 12 July 1999; accepted 13 July 1999

Abstract

Objective: Tight fundoplication is a well known complication of surgery for gastroesophageal reflux. We have noted, in clinical experience, that some patients operated for gastro-oesophageal reflux develop pharyngo-oesophageal dysphagia. This study was undertaken to elucidate, by comparing motility data in patients with and without tight fundoplication, the pathophysiologic characteristics of the swallowing mechanism and to clarify the cause of dysphagia in these patients. **Methods**: Sixteen patients with postoperative persistent dysphagia following a fundoplication have been studied, of those 10 presented a lower oesophageal sphincter resting pressure above or equal to 30 mmHg (group A). Clinical work-up included a questionnaire, oendoscopy, manometry and barium meal or video-roentgenography. Oesophageal manometry was performed using a slow pull through technique while the stationary pull through technique was used for the pharyngo-oesophageal segment. The following parameters were evaluated: (a) amplitude of pharyngeal contraction; (b) upper oesophageal sphincter resting pressure; (c) amplitude of upper oesophageal sphincter contraction; (d) amplitude of oesophageal contraction; (e) lower oesophageal sphincter resting pressure. The results were compared to those of 21 patients who had a fundoplication with normal lower oesophageal sphincter pressure (group B). **Results**: in group A there were three males and seven females, with a mean age of 51 years (ranging from 28 to 60 years). Previous operations were Nissen in two and Nissen Rossetti in eight patients. Three out of 10 patients of group A presented pharyngo-oesophageal dysphagia. Mean lower oesophageal sphincter 36 versus 21 mmHg and upper oesophageal sphincter 86 versus 42 mmHg resting pressure, pharyngeal 147 versus 76 mmHg and oesophageal amplitude, upper oesophageal contraction 251 versus 103 mmHg were significantly higher in patients of group A versus group B. An increased number of repetitive contractions was also found in group A. The presence of a strong correlation was demonstrated between the pharyngeal amplitude and the closing tone of the upper oesophageal sphincter (R2 0.742 and R2 0.739) in both groups. **Conclusion**: Tight fundoplication is, in our experience, always associated with total fundoplication. The appearance of pharyngo-oesophageal dysphagia in the postoperative period in patients operated on to correct gastroesophageal reflux using a total fundoplication, should not be under-estimated because it suggests an obstruction of the distal oesophagus.

Keywords: Gastro-oesophageal reflux; Fundoplication; Laparoscopy; Complication

1. Introduction

Tight fundoplication (TF) is an early and persistent complication of surgery for gastroesophageal reflux (GOR); it consists in a high pressure of the lower oesophageal sphincter (LOS) due to the creation of an excessively tight wrap (Fig. 1) resulting in an immediate and persistent postoperative difficulty in swallowing and belching. The postoperative persistence of these refractory symptoms for 6–12 months give an extremely poor quality-of-life.

The most common causes of TF are wrong patient selection, absence of preoperative functional tests or an inappropriate surgical technique.

Despite several studies reporting on complications of surgery for correction of GOR [1–5] very little information is available in the medical literature on the pathophysiological characteristics of the oesophageal motility in patients with TF; nevertheless we have recently noted in clinical experience that some patients operated for correction of GOR with a fundoplication develop postoperatively, as a predominant and new symptom, the pharyngo-oesophageal dysphagia (POD). The aim of the study was therefore to elucidate in patients with a TF the pathophysiologic characteristic of the swallowing mechanisms and to determine the cause of POD by studying the motility patterns of the

☆ Presented at the 12th Annual Meeting of the European Association for Cardio-thoracic Surgery, Brussels, Belgium, 20–23 September, 1998.

* Corresponding author.

E-mail address: mmiglior@mbox.unict.it (M. Migliore)

1010-7940/99/$ - see front matter
PII: S1010-7940(99)00254-7

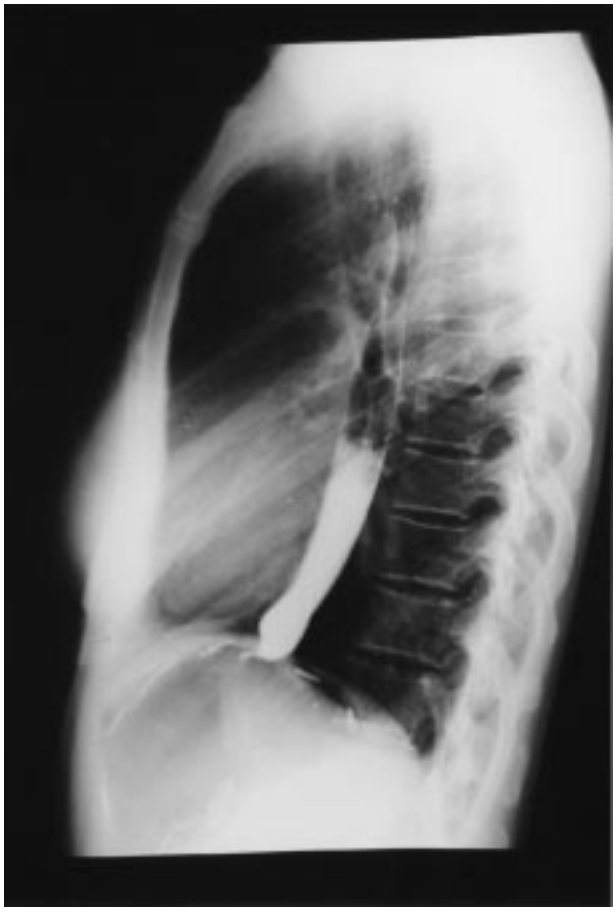

Fig. 1. Roentgenogram demonstrating a tight Nissen, causing a chronic obstruction at the gastro-esophageal junction resulting in oesophageal retention of barium and gastric distension.

pharynx and the entire oesophagus, and then to correlate the data with the features observed in patients without TF.

2. Material and methods

From January 1, 1990 to December 31, 1997, 16 consecutive patients with persistent postoperative dysphagia following fundoplication for treatment of GOR have been evaluated. All patients underwent a diagnostic work-up which included a detailed clinical questionnaire covering oesophageal dysphagia, POD, chest pain, epigastric pain, heartburn, gas bloat, weight loss, regurgitation and salivation. The diagnostic tests used to identify the cause of complication were oendoscopy, barium meal or video-roentgenography, and manometry.

Criteria for inclusion in the group A (TF) were: (a) the presence of postoperative persistent dysphagia and (b) the absence of oesophageal clearance failure.

An operation for GOR with a fundoplication but without postoperative dysphagia and a manometric study performed within 1 year following surgery were required to include the patients in the group B (control group).

3. Manometric studies

Manometric studies were performed after an overnight fast with the use of a standard motility catheter consisting of four water-filled polyvinyl tubes bonded together with lateral openings spaced 5 cm apart. The proximal ends of the polyvinyl tubes were connected to pressure transducer and in turn to a polygraph connected to a computer system. A constant infusion of distilled water was delivered by a pneumohydraulic system with an infusion rate of 0.6 ml/min. The stationary pull-through technique was used to study both oesophageal sphincters pressure and relaxations. In particular pharyngo-oesophageal manometry was performed as previously described [6]. The oesophageal body function was assessed by performance of 10 dry and 10 wet swallows with 5 ml of water every 20 s (recording site 5, 10 and 15 cm above LOS).

We measured several manometric variables including the following: (a) the amplitude of pharyngeal contraction; (b) the upper oesophageal sphincter (UOS) resting pressure; (c) the UOS contraction amplitude; (d) the coordination between pharynx and UOS relaxation; (e) the coordination between UOS contraction and upper oesophageal contraction; (f) the amplitude of oesophageal contraction; (g) the lower oesophageal sphincter (LOS) resting pressure and (h) the lower oesophageal sphincter relaxation.

4. Manometric interpretation and definitions

The LOS relaxation was considered complete (100%) when the resting tone dropped to the intragastric baseline pressure. The UOS relaxation was considered complete (100%) when the resting tone decreased to the oesophageal baseline. The definition of co-ordination between the pharynx and the UOS and between UOS and the upper oesophagus have been published recently [6]; pharyngo-sphincteric co-ordination was considered normal when the sphincter relaxation occurred before the onset of pharyngeal contraction and the pharyngeal contraction was completed before the onset of the UOS closure; Sphincter-oesophageal co-ordination was considered normal when the peak of the UOS closure preceded the peak of the upper oesophageal contraction measured 5 cm distally.

Tight fundoplication was defined as a fundoplication with a postoperative lower oesophageal sphincter pressure of greater than or equal to 30 mmHg (the mean +3 SD of control).

Oesophageal ramp pressure represented, as defined by Mathew at al. [7], a rise in intraluminal oesophageal pressure just before swallow induced peristaltic contraction.

Dysphagia was considered persistent when the duration was longer than 12 months. Pharyngo-oesophageal dysphagia was defined as difficulty in initiating the act of swallowing within 1 s.

Table 1
Symptoms observed in patients of the group A

	No. of patients
Oesophageal dysphagia	7
Chest pain	7
POD	3
Weight loss	3
Epigastric pain	3
Gas bloat	2
Salivation	2
Regurgitation	1

4.1. Statistical methods

Statistical analysis was performed using the Mann–Whitney non-parametric test to evaluate the significance between groups. A probability value less than 0.05 was considered significant. The differences between groups with respect to abnormal relaxation, numbers of repetitive contractions and ramp pressure were analyzed using Fisher's exact test. The difference was significant when $P = 0.015$; where appropriate, correlation coefficient R^2 was used to determine the relationship between variables.

Data relating to patient demographics were expressed as the mean and range and data relating to manometric characteristics as the mean±, range and standard deviation (SD).

5. Results

5.1. Clinical data

Ten patients met our criteria and thus are the subject of the study forming group A: there were three males and seven females with a mean age of 51 years (range 28–60 years). The other six patients were excluded because dysphagia was the consequence of oesophageal clearance failure.

In the group B we included 21 patients, seven males and 14 females with a mean age of 53 years (range 28–79 year).

Previous operations of the patients in the group A were Nissen in two and Nissen-Rossetti in eight and in the group B Nissen in five, Nissen Rossetti in 12 and Toupet in four. Four out 10 patients of the group A had no manometric studies performed prior to surgery. Three out 10 patients of the group A have been operated in our Department.

In group A the symptoms are summarized in Table 1. All three patients with POD presented chest pain and weight loss as associated symptoms.

5.2. Manometric studies

The LOS resting pressure was 36.6 (SD 7.8, range 30–54) in the dysphagia group and 21.4 (SD 2.7, range 18–28) in the group B ($P < 0.001$) (Fig. 2). Relaxation was abnormal in six (60%) patients of the group A and in three (14 %) patients of the group B ($P = 0.015$).

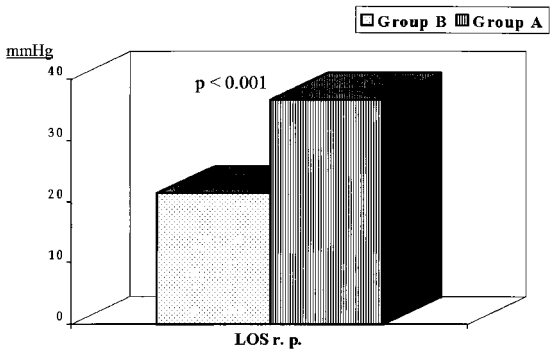

Fig. 2. The pressure difference and *P*-value of the postoperative lower oesophageal sphincter in group A and B.

The mean resting UOS pressures was 86.6 (SD 29, range 60–140) in the group of TF and 42.8 (SD 18.5, range 20–80). The amplitude of the pharyngeal contractions were 147.8 (SD 56.4, range 100–230) in group A and 76.7 (SD 31.3, range 30–160) in group B. The UOS contraction was 251.4 (SD 83.3, range 123–310) in the dysphagia group and 103.4 (SD 54.4, range 30–210) in the control group. Pressure changes and statistics are shown in Fig. 3.

Two out of three patients with POD presented a crico-oesophageal incoordination as shown in Fig. 4; nevertheless one of these presented an incomplete relaxation of the sphincter. The third patient with POD showed a very high pressure of the pharyngeal contraction with a long duration resulting in an inco-ordination between pharynx and UOS relaxation. A strong correlation was found between pharyngeal amplitude and UOS contraction (R^2 0.742 and R^2 0.739) in both groups.

In the distal oesophagus the amplitude of oesophageal contraction was 183.6 mmHg (SD 111, range 60–320) in patients of the group A and 64.7 (SD 27.8, range 30–123) in the group B ($P = 0.001$). The repetitive contractions were present in eight patients of group A and in three patients of group B ($P = 0.015$). The elevated ramp pressure (Fig. 5)

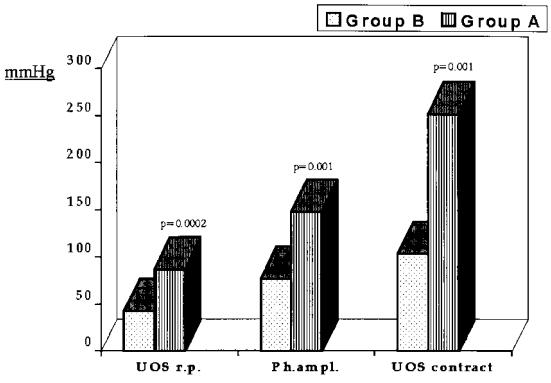

Fig. 3. The pressure differences and *P*-values at various levels of the pharyngo-oesophageal segment in the two groups. UOS r.p., upper oesophageal sphincter resting pressure; Ph.ampl., amplitude of pharyngeal contraction; UOS contract, amplitude of upper oesophageal sphincter contraction.

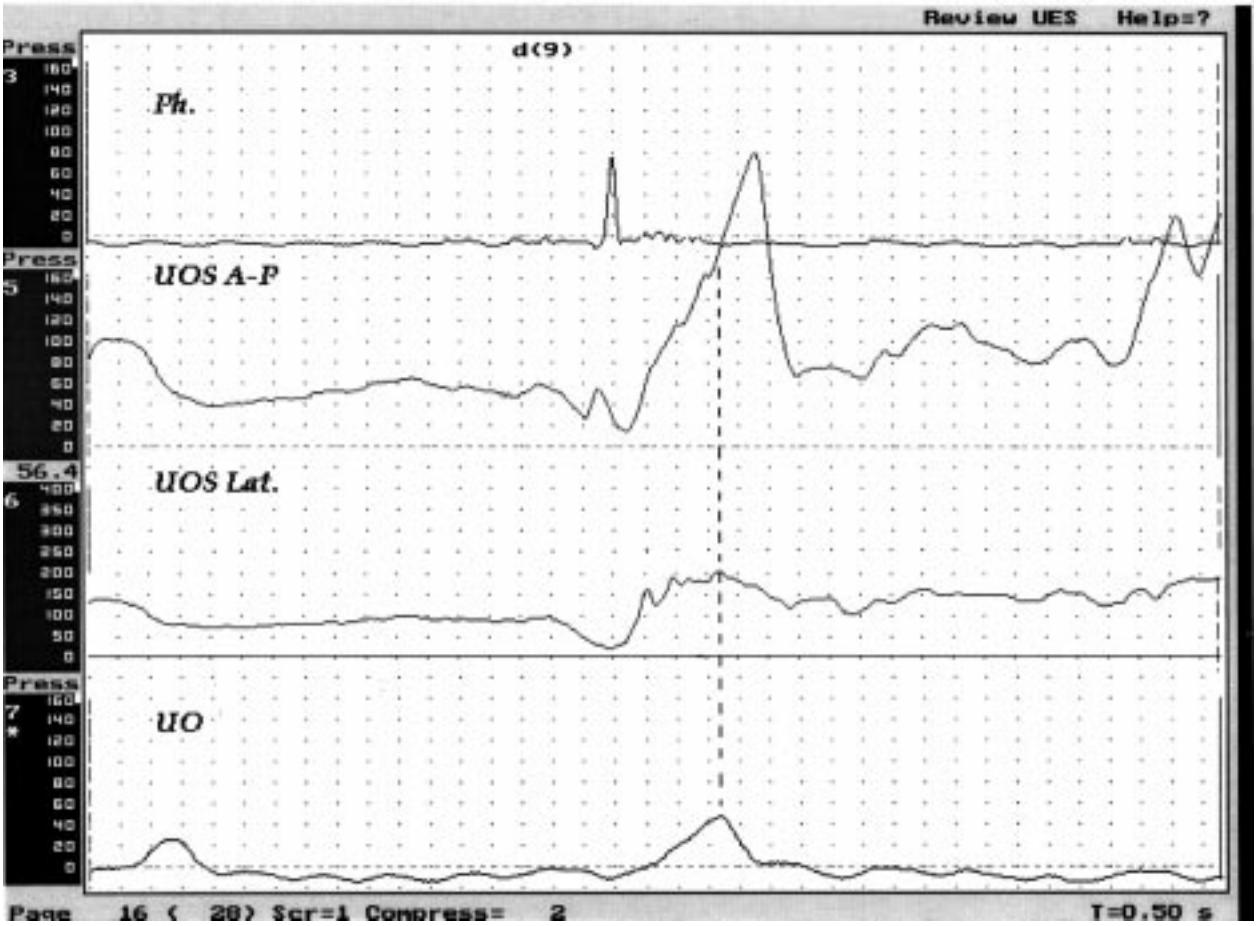

Fig. 4. Manometric tracing of the pharyngo-oesophageal segment in a patient with TF, showing a contraction wave in the UOS that is synchronous with the UO contraction. Ph., pharynx; UOS A-P, pressure of upper oesophageal sphincter antero-posteriorly; UOS lat., pressure of upper oesophageal sphincter laterally; UO, upper oesophagus.

was present in six patients of the group A and in 2 out 21 in group B ($P = 0.015$).

6. Discussion

Tight fundoplication is the third most common complication [8] among the failures and complications of surgery for correction of gastro-oesophageal reflux and, probably, is the most unwanted because symptoms are generally severe, develop immediately following operation and persist for months and years. Diagnosis of a TF is a challenging problem for the surgeon because still today it remains difficult to differentiate a TF from a tight hiatus or an excessively long fundoplication and, finally, dysphagia can be also a consequence of an oesophageal clearance failure.

In recent years the laparoscopic technique increases dramatically the number of operations performed for correction of gastro-oesophageal reflux resulting in an increased number of reported failures and complications such as the creation of a tight wrap [4,9,10]. The pathophysiological mechanisms responsible for the development of postoperative dysphagia are still uncertain and the aetiology seems to be multifactorial [7]. Recently Peters et al. [11] showed that persistent dysphagia was more common in the laparoscopic group compared to open surgery (9.4 vs. 2.7%), while Watson et al. [12] and Orsoni et al. [13] reported fibrotic stenosis of the muscular oesophageal hiatus to be one specific complication of the laparoscopic technique. We believe that manometry is the most useful diagnostic test to study the unhappy patient with a TF; in fact manometric studies are of paramount importance to obtain information on oesophageal motility and can suggest the appropriate treatment.

We have noted that all patients with TF had a total fundoplication and that 80% of the operations were performed laparoscopically. The crura was always approximated and all fundoplications were performed via abdominal approach. We have also documented that four out of 10 patients with a TF fundoplication had no manometric studies performed prior to surgery demonstrating the importance of a full preoperative diagnostic work-up. A weakness of the study is due to the fact that four patients in group A did not have preoperative physiological studies, but this was out of our control because the patients were operated by

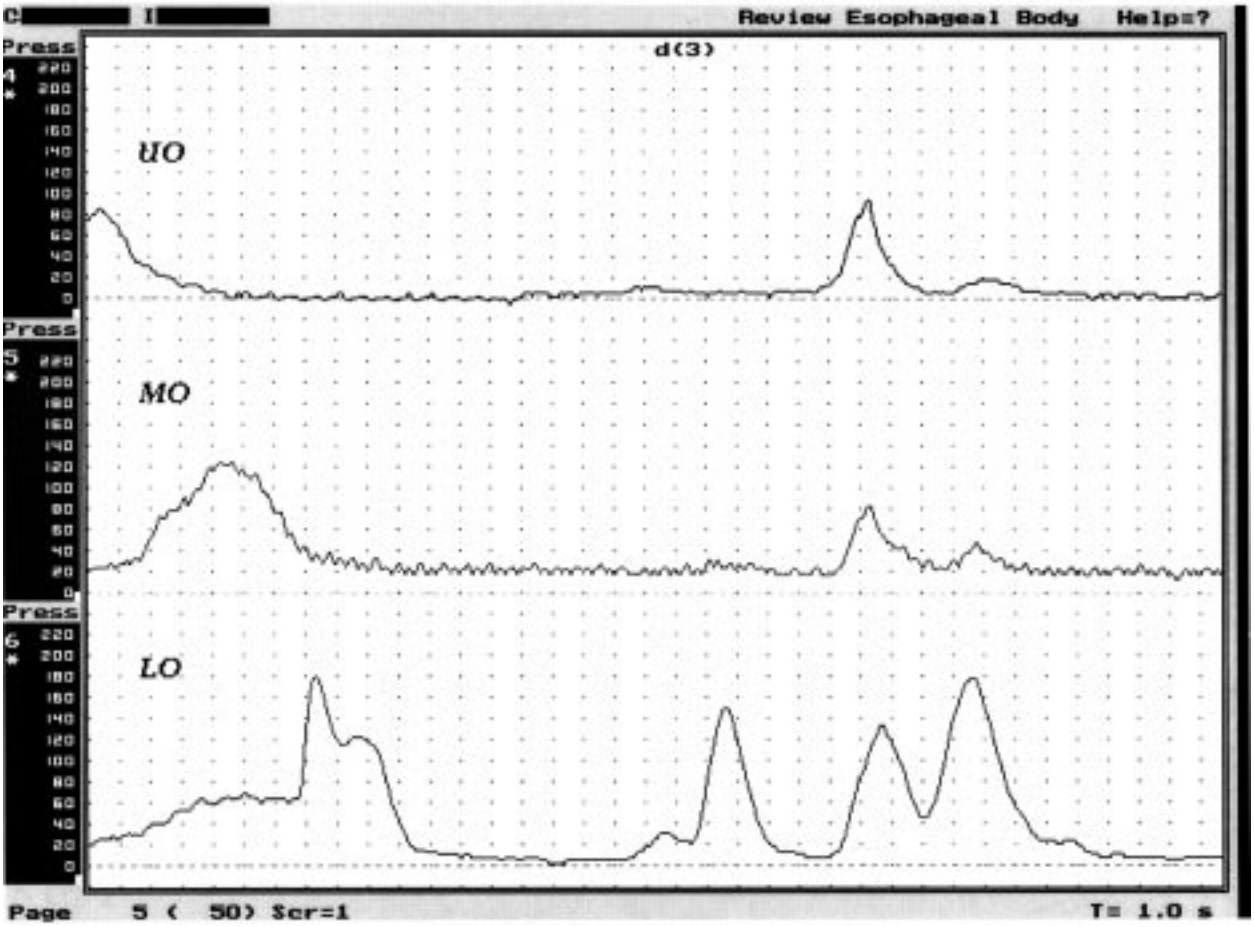

Fig. 5. Manometric tracing of the oesophageal body in a patient with TF, showing an elevated ramp pressure and the presence of repetitive contraction. UO, upper oesophagus; MO middle third of the oesophageal body; LO, lower oesophagus.

other surgeons. Although the possibility of preoperative dysmotility in the three patients with postoperative POD cannot be demonstrated, clinically none of these patients presented preoperative POD.

Regarding the LOS characteristics of patients with TF the mean resting pressure was 36 mmHg (ranges from 30 to 54 mmHg) and the relaxation was abnormal in 60% of them; this alteration could be secondary to surgical manipulation in the early postoperative period but is always abnormal at a later date, it should be attributed to the presence of a tight wrap.

Manometric changes of the oesophagus in patients with a TF have not been previously described although Skinner [14] and Low et al. [15] reported that manometry frequently showed a motor disorder which may be secondary to an oesophageal obstruction following the antireflux repair. We excluded six patients with persistent postoperative dysphagia because, on manometry, it was demonstrated a failure of oesophageal clearance and the LOS resting pressure was below 30 mmHg.

The current study clearly demonstrates that the entire oesophagus and also the pharynx increase the amplitude of contraction in response to the creation of a distal oesophageal wrap above 30 mmHg, twice that of the control group without oesophageal dysphagia: these responses seem to be physiological like a ‘tension’ in all the oesophageal tube and pharynx.

Mathew et al. [7] demonstrated recently that ramp pressure is elevated in patients with a laparoscopic Nissen fundoplication and that triple lumen perfused catheter, equipped with side hole sensors fails to show this manometric finding. On the contrary we were able to demonstrate, using a standard perfused motility catheter, that ramp pressure is more elevated in patients with TF while it is not always present in patients with a mean LOS of 21 mmHg (group B).

Only a few months ago, our group [16] and the Leuven group of Lerut et al. [17] reported the presence of POD in patients with fundoplication. From our knowledge and from a computerized research on ‘medline’, it seems that POD, as a predominant and new symptom following surgery for correction of GOR has never been reported, therefore the neuromuscular mechanisms involved in the development of this symptom is unknown. The strong correlation existing between the pharyngeal amplitude and the UOS contraction suggests a close relationship between the pharynx and the

oesophagus; this is the main reason because we believe that the UOS contraction should be considered a 'trans-sphincteric contraction'. This hypothesis can explain, even if the number of the patient is small, the development of POD in patients with TF.

It should be emphasized that, in our experience, all three patients with POD presented associated a TF and the motility abnormalities detected at manometry were that two out of the three patients with POD presented a sphinctero-oesophageal inco-ordination (Fig. 4) while in one patient a very high amplitude of pharyngeal contraction was demonstrated. All three patients with POD presented chest pain and weight loss as associated symptoms.

Lerut treated one of these patients with a crico-oesophageal myotomy to solve the problem; despite crico-oesophageal myotomy is the most common surgical procedure to restore normal deglutition in patients with POD [18], in the present experience, because all three patients presented a TF and associated chest pain and weight loss, we preferred to treat the underlying problem which was, in our cases, the presence of a tight wrap: two patients required a surgical intervention for symptom relief and were operated through a left thoracotomy to enable full mobilization of the oesophagus and complete visualization of the cardia, upper stomach and fundoplication [14]; in both cases we took down the prior repair (one Nissen and one Nissen–Rossetti) and a Belsey Mark IV fundoplication was performed. The third patient is on follow-up after two oesophageal dilatations.

We believe that it is possible that in the follow-up clinic the surgeon does not ask about POD and, when the patient refers to it, the surgeon includes the patient in the large category of 'bolus pharyngeous or globus hystericus'; therefore we strongly believe that the symptom of POD is underrecognized.

To summarize a TF is, in our experience, always associated with a total fundoplication, therefore a TF could be synonimus of total fundoplication. It generates a significant augmentation of the amplitude contractions in the oesophageal body and, surprisingly, in the pharyngo-oesophageal segment. Where there is no failure of oesophageal clearance in the upper oesophagus the POD is explained with the higher pressures in the pharynx and with crico-oesophageal inco-ordination. Redo surgery, which consists of taking down the prior repair with the association of a partial fundoplication such as Belsey Mark IV, seems necessary to solve the symptom.

In conclusion, the appearance of pharyngo-oesophageal dysphagia in the postoperative period in patients operated for correction of GOR using a total fundoplication, should not be under-estimated because it suggests an obstruction of the distal oesophagus.

Acknowledgements

Marcello Migliore wishes to express personal thanks to David B. Skinner, MD (President and Professor of Surgery of The New York Presbyterian Hospital-Weill Medical College of Cornell University, New York, USA) for his continuous support and critical review of the manuscript.

References

[1] Little AG, Ferguson MK, Skinner DB. Reoperation for failed antireflux operation. J Thorac Cardiovasc Surg 1986;91:511–519.
[2] Leonardi HK, Cruzier RE, Ellis Jr FH. Reoperation for complication of the Nissen fundoplication. J Thorac Cardiovasc Surg 1981;81(1):50–56.
[3] Negre JB. Post fundoplication symptoms: do they restrict the success of Nissen Fundoplication? Ann Surg 1983;198(6):698–700.
[4] Collet D, Cadière GB, et al. Conversions and complications of laparoscopic treatment of gastroesophageal reflux disease. Am J Surg 1995;(Jun)169:622–626.
[5] Stirling MC, Orringer MB. Surgical treatment after the failed antireflux operation. Thorac Cardiovasc Surg 1986;92:667–672.
[6] Migliore M, Payne HR, Jeyasingham K. Pathophysiological basis for surgery of Zenker's diverticulum. Ann Thorac Surg 1994;57:1616–1621.
[7] Mathew G, Watson DI, Myers JC, Holloway RH, Jamieson GG. Oesophageal motility before and after laparoscopic Nissen fundoplication. Br J Surg 1997;84:1465–1469.
[8] Stein HJ, Feussner H, Siewert JR. Failure of antireflux surgery: causes and management strategies. Am J Surg 1996;171:36–40.
[9] Frantzides CT, Carlson MA. Laparoscopic redo Nissen fundoplication. J Laparoendosc Adv Surg Tech A 1997;7(4):235–239.
[10] Kozarek RA, Low DE, Raltz SL. Complication associated with laparoscopic antireflux surgery: one multispecialty clinic's experience. Gastrointest Endosc 1997;46(6):527–531.
[11] Peters JH, Heimbucher J, Kauer WKH, Incarbone R, Bremner CG, DeMeester TR. Clinical and physiologic comparison of laparoscopic and open Nissen fundoplication. J Am Coll Surg 1995;180(4):385–393.
[12] Watson DI, Jamieson GG, Mitchell PC, Devitt PG. Bitten Jones. R., Stenosis of the esophageal hiatus following laparoscopic fundoplication. Arch Surg 1995;130:1014–1016.
[13] Orsoni P, Berdath S, Sebag F, Picaul R. An unusual cause of dysphagia after laparoscopic fundoplication: a report on two case. Surgery 1998;:241–242.
[14] Skinner DB. Surgical management after failed antireflux operations. World J Surg 1992;16:359–363.
[15] Low DE, Mercer CD, James EC, Hill LD. Post Nissen syndrome. Surg Gynecol Obst 1988;167:1–5.
[16] Migliore M, Basile F, Iuppa A, Sanfilippo G, Catania G, Deodato G. Pathophysiology of the swallowing mechanisms in patients with tight Nissen fundoplication. VII World Congress of ISDE (Abstract). Can J Gastroenterol 1998;12(8):319.
[17] Lerut T, Lassen P, Knod J, Coosemans W, De Leyn P, Deneffe G, VanRaemndonck D. Laparoscopic antireflux surgery: a critical analysis. VII World Congress of ISDE (Abstract). Can J Gastroenterol 1998;12(8):197.
[18] Duranceau A, Lafontaine ER, Taillefer R, Jamieson GG. Oropharyngeal dysphagia and operations on the upper esophageal sphincter. Surg Ann 1987;19:317–364.

7. Appendix A: Editorial comment

This study by Migliore and Deodata is a provocative one for in it they

describe for the first time a post (360°) fundoplication complication characterized by pharyngo-oesophageal dysphagia (POD). This unexpected finding, first published nearly a year ago in abstract form [1], was not observed by my associate and myself in an experience with 101 re-operations for failed antireflux surgery in 21 of whom too tight a wrap was considered the reason for re-operation [2]. Stein and associates, when reporting on a similar number of patients requiring re-operation for a failed antireflux procedure, likewise identified no patient with abnormalities of upper esophageal sphincter function [3]. In both of these studies, all patients had esophageal motility studies prior to undergoing re-operation. Migliore and Deodata however identified higher mean resting upper oesophageal sphincteric pressures and higher amplitude of sphincteric and pharyngeal contractions in the study group as compared to controls without dysphagia. They also identified pharyngo-oesophageal incoordination in some of these patients They explain these phenomena on the basis of increased 'tension' involving all of the esophagus and pharynx after a total wrap resulting in pressures at the lower sphincter of 30 mm of mercury or higher, a theory which requires confirmation by others.

There are a number of aspects of this paper that require comment. First let me say that the statistical methodology employed in the study, namely use of non-parametric hypothesis testing, is appropriate when comparing continuous data that are not normally distributed for which contingency tables with small individual cell counts are constructed. What is missing, however, is an appreciation of the total number of fundoplication operations, particularly those exhibiting postoperative dysphagia, from which the patients under comparison in this study were selected. Only in this way can we conclude whether or not the observations reported are based on a highly selected number of patients and therefore may not be representative of the large numbers of fundoplication procedures being currently performed by others.

Eighty percent of the procedures were performed laparoscopically. Is this approach playing a role in these reported findings? It has been shown that the risk of postoperative dysphagia following a laparoscopic antireflux procedure is higher than that following the open operation [4]. The majority of patients in both groups analyzed by Migliore and Deodata underwent a Nissen-Rosetti procedure in which the short gastric vessels are not divided, thus making it more difficult to achieve a loose 'floppy' wrap than is possible with division of these vessels as well as the posterior gastric artery by an open approach. While the authors emphasize the level of pressure at the lower sphincter following a total wrap, they say nothing about the length of the wrap which may be equally important in the production of postoperative dysphagia. Most surgeons now prefer a short wrap (1–2 cm). However, it is unlikely that any of these technical variations contribute to post fundoplication POD.

In summary, this is a provocative paper which should influence all surgeons performing antireflux surgery to study carefully patients who develop persistent dysphagia following operation to exclude the possibility of pharyngo-oesophageal dysfunction. Such studies will not only clarify the significance of the observations of the authors but may provide a physiologic explanation for the development of this complication, which in my experience is extraordinarily rare.

References

[1] Migliore M, Basile F, Juppa A, et al. Pathophysiology of the swallowing mechanisms in patients with tight Nissen fundoplication. Can J Gastroenterol 1998;12(Suppl 13):104-319.

[2] Ellis Jr FH, Gibbs SP, Heatley GJ. Reoperation after failed antireflux surgery: Review of 101 cases. Eur J Cardio-thorac Surg 1996;61:1106-1111.

[3] Stein HJ, Feussner H, Siewert JR. Failure of antireflux surgery: causes and management strategies. Am J Surg 1996;171:36-40.

[4] Hunter JG, Swanstrom L, Waring JP. Dysphagia after laparoscopic anti-reflux surgery. The impact of operative tecnique. Ann Surg 1996;224:51-57.

F. Henry Ellis, Jr.

Appendix B: Conference discussion

Dr T. Lerut (*Leuven, Belgium*): I can confirm that we have had, also, in our experience two patients with upper dysphagia. Now, what do we have to do in case of upper dysphagia, do we have to dismantle the fundoplication or do we have to do a cricopharyngeal myotomy? What is your suggestion?

Dr M. Migliore: In case of patients with postoperative pharyngoesophageal dysphagia, I think, first of all, we should perform the oesophageal manometry of the entire oesophagus. If we demonstrate, as we found in our study, a high pressure in the lower oesophageal sphincter, we should take down the fundoplication, which was in our patients a Nissen type fundoplication. If the pressure in the lower oesophageal sphincter is normal a myotomy of the upper sphincter can be justified only if there are no motor abnormalities in the oesophageal body.

Dr Lerut: But if you don't have high sphincter pressure in the lower sphincter, can you then speak about a tight Nissen? Probably not.

Dr Migliore: No, I agree with you. Tight fundoplication was defined when the postoperative lower oesophageal sphincter pressure was above or equal to 30 mmHg.

Dr Lerut: So the mechanism will be different then?

Dr Migliore: Yes. A patient with dysphagia but without tight fundoplication can develop the symptom as a consequence of oesophageal clearance failure.

Mr K. Jeyasingham (*Bristol, UK*): I think you've documented what you have said in manometric terms. But tell me, in your patients, did the pharyngoesophageal dysphagia come up before the lower oesophageal dysphagia, and how long before, or did they come after the lower oesophageal dysphagia, in which case you would probably have investigated it earlier?

Dr Migliore: Our patients developed the pharyngo-esophageal dysphagia in the early follow-up and without the association of the lower oesophageal dysphagia. The symptom is the most predominant and generally is associated with chest pain and weight loss. Concerning your second question, in our institution before this study we used to investigate earlier the patient with lower oesophageal dysphagia.

Mr J. Thorpe (*Leeds, UK*): It's very rare to have to take down a Nissen fundoplication. I would make a plea for a conservative approach, like a dilatation, in the first instance, before going on to myotomies and undoing the plication. Do you think the motility changes are secondary to the operation and due to disturbed denervation? What do you think the mechanism of these changes are, particularly in the upper sphincter? Is this just a normal response to a more obstructive sphincter?

Dr Migliore: All the three patients with pharyngo-esophageal dysphagia underwent oesophageal dilatation. We operated on two of them because there was no response and the third patient is on follow-up after two sessions of dilatation. Regarding the pathphysiological mechanism is not known, we demonstrated that the tight wrap in the distal oesophagus can also cause a functional obstruction of the upper oesophageal sphincter, which is explained with the crico-esophageal inco-ordination. We can speculate that the mechanism of these changes is a vagus nerve injury due to the presence of the tight wrap, but we have currently no data which can confirm this hypothesis.