

V. Puri · G. V. Kakarlapudi · Z. T. Awad · C. J. Filipi

## Hiatal hernia recurrence: 2004

Received: 20 April 2004 / Accepted: 4 June 2004 / Published online: 6 August 2004  
© Springer-Verlag 2004

**Abstract** *Background:* The incidence of laparoscopic hiatal hernia recurrence is less than ideal. The reasons are more theoretical than objective, as the literature has little data in support of specific mechanisms of recurrence. *Method:* A recent literature review using all Internet-available, English-language articles on laparoscopic hernia repair was completed. *Results:* A multitude of mechanisms of recurrence are suggested, but only surgeon inexperience, postoperative vomiting, heavy lifting, and retention of the hernia sac are supported by data. *Conclusion:* The incidence of hiatal hernia recurrence has stabilized. The role of an onlay mesh prosthesis for the prevention of hiatal hernia recurrence is under investigation, and long-term results are awaited.

**Keywords** Hiatal hernia · Laparoscopy · Recurrence · Mesh

### Introduction

The laparoscopic repair of routine hiatal hernias may be problematic. Clearly the initial experience with repair of large type III hernias was a concern for all laparoscopic surgeons. An unusually high cumulative recurrence rate [1, 2, 3], as compared to the open technique (Table 1), lead some to believe that all paraesophageal hernias should be repaired using the open operative approach. Recurrences are not defined well in some studies, and end points for hernia definition vary between studies.

Presented at the Seventh Annual Scientific Meeting of the American Hernia Society, Orlando, Fla. USA February 24–28, 2004.

V. Puri · G. V. Kakarlapudi · Z. T. Awad · C. J. Filipi (✉)  
Department of Surgery, Creighton University,  
Suite 3700 601 N. 30th St, Omaha,  
NE 68131, USA  
E-mail: cjfilipi@creighton.edu  
Tel.: +1-402-2804213  
Fax: +1-402-2804278

Nevertheless, the available data, as imperfect as it was, convinced many that a problem existed.

Subsequent laparoscopic hiatal hernia repair data have shown an overall improvement in recurrence rates, although length of follow-up is relatively short. Apparently this improvement is a manifestation of the learning curve. Nevertheless, the cumulative recurrence rate remains higher than ideal.

This article will review the previously mentioned causes for the high recurrence rates [4, 5] and elucidate several additional mechanisms for hernia recurrence. Suggested technical alterations will be given.

Prosthetic onlay reinforcement of the crus closure [6, 7] has become a popular alternative. Low recurrence rates have been demonstrated, although long-term follow-up is not available. This alteration in technique will be discussed as well.

### Methods

A Medline review of the English literature was performed. The early and late laparoscopic hiatal hernia repair data are compared against results from open hiatal hernia repair. The early experience from laparoscopic repair is summarized in Table 2 (1997–2000). Subsequent series of laparoscopic repair are included in Table 3. Series with fewer than 15 patients were excluded from the tables. Smaller series have, however, been included for the data on repair using mesh. Articles describing specific mechanisms of hiatal hernia recurrence were reviewed and included.

### Results

Table 1, Table 2, Table 3, and Table 4 show the series that are germane. Table 2 demonstrates the early experience of laparoscopic surgeons and Table 3 the more recent experience with laparoscopic repair of hiatal

**Table 1** Open paraesophageal hernia repair

Source	No. of patients	Recurrence rate (%)	Length follow-up (months)	Surgery
Orringer et al. [8] (1972)	892	14.5	120–180	T
Nicholson and Nohl-Oser [16] (1976)	283	13.1	12	T
Mokka et al. [17] (1977)	50	12	60	A
Gardner et al. [18] (1977)	42	0	38.5	A
Gatzinsky et al. [19] (1979)	105	9.5	48	A + T
Refsum and Nygaard [20] (1979)	43	16.3	35	A
Singh [21] (1980)	238	8.4	84–120	T
Pearson et al. [22] (1983)	53	17	74.4	A + T
Ellis Jr. et al. [23] (1986)	51	8	59	A + T
Williamson et al. [24] (1993)	119	10	61.5	A
Weissberg and Refaely [25] (1995)	55	5.5	108	A + T
Schauer et al. [26] (1998)	25	8	48	A + T
Rogers et al. [27] (2001)	60	1.5	19	T

T = thoracic; A = abdominal

**Table 2** Laparoscopic paraesophageal hernia repair without mesh: Early experience

Author (year)	Patients (n)	Conversions (n)	Recurrences (%)	Complications	Follow-up (months)
Perdikis et al. [28] (1997)	65	2	15.2	Gastric perforation 2, bleed 6	18
Huntington [29] (1997)	58	1	0	Major 4	12
Gantert et al. [30] (1998)	55	6	4	Intraoperative technical 5, pulmonary embolus 1	11
Hawasli and Zonca [31] (1998)	27	0	0	Stoke 1	1–56
Horgan et al. [32] (1999)	41	2	12	Death 1, stomach perforation 1	36
Edye et al. [10] (1998)	55	0	12.7	Dysphagia 2, gastroparesis 1	20–40
Schauer et al. [26] (1998)	70	3	0 symptomatic	Gastrotomy 1, esophageal perforation 4	13
Wiechmann et al. [33] (2001)	60	6	9	Esophageal perforation 2	12
Swanstrom et al. [34] (1999)	52	0	8	Intraoperative 3, postoperative 3	18
Watson et al. [11] (1999)	86	20	1	Early 11	12
Carlson et al. [35] (1999)	16	0	18.8	Pneumothorax 1	12–36
Hashemi et al. [1] (2000)	27	0	42	Major 2	24

**Table 3** Laparoscopic paraesophageal hernia repair without mesh: Recent experience

Author (year)	Patients (n)	Conversions	Recurrences	Complications	Follow-up (months)
Pierre et al. [36] (2002)	203	3	5 (2.5%) needed reoperation	Leaks or perforations 15	18
Jobe et al. [2] (2002)	52	0	32%	—	37
Diaz et al. [3] (2003)	116	0	21 (22%) 3 (3%) reoperation	5 (4.7%) major	30
Balsara et al. [37] (2002)	22	0	0	1 major	3–24
Khaitan et al. [38] (2002)	31	6	7% herniated wrap 33%	2	25.2
Dahlberg et al. [39] (2001)	37	2	12.9%	Intraoperative 6, postoperative 5	15
Velanovich and Karmy-Jones [40] (2001)	31	5	3 (12%)	1 major	?
Mattar et al. [41] (2002)	136	3	3 (2.3%) symptomatic	14 (12.2%)	40
Leeder et al. [12] (2003)	37	4	2 (6%)	1 major	6–89
Keidar and Szold [42] (2003)	20	0	18%	15%	58
Frantzides et al. [7] (2002)	36	0	8 (22%)	0 major	40

hernias. The cumulative recurrence rates for Table 2 and Table 3 are 8.1% and 9.6%, respectively.

#### Proven mechanisms of recurrence for hiatal hernia

Previously only vomiting, surgeon inexperience, and heavy lifting have been conclusively associated with

recurrent hiatal formation [4, 5, 8, 9]. Postoperative vomiting is clearly the most agreed upon mechanism of hiatal hernia recurrence. If the patient experiences dry heaving, the chances of hiatal hernia recurrence also increase. This may occur more often with laparoscopic operations, as the patient does not have nasogastric decompression often utilized with open surgery.

**Table 4** Paraesophageal hernia repair with mesh

Author (year)	Patients (n)	Open/Laparoscopic	Recurrences (%)	Complications	Follow-up (months)
Carlson et al. [43] (1998)	44	O	0	Major 3	52
Carlson et al. [35] (1999)	15	L	0	Major 0	12–36 Keidar
Hui et al. [44] (2001)	12	L	8.5	Major 1	37
Livingston et al. [45] (2001)	10	L	0	Major 0	
Casaccia et al. [46] (2002)	8	L	0	Major 0	8
Frantzides et al. [7] (2002)	36	L	0	Major 0	20.4
Champion and Rock [6] (2003)	52	L	1.9	Major 0	25
Granderath et al. [47] (2003)	24	L	0	Major 0	12
Keidar and Szold [42] (2003)	10	L	10	Major 1	58

O = open; L = laparoscopic

Surgeon inexperience was demonstrated as a factor in a study by Orringer et al. [8]

Another cause of hernia recurrence that appears to be conclusively established is the retention of the hernia sac [10, 11, 12]. The removal of the sac from the mediastinum, if not the complete sac excision, is now a routine part of the laparoscopic repair of large hiatal hernias.

A variety of other suspected mechanisms have been associated with recurrence, and additional risk factors for laparoscopic hiatal surgery have been suggested. The latter prove important because the follow-up by en large is shorter for the laparoscopic series. Thus these factors, although theoretical, still may be of importance.

Possible mechanisms of recurrence common to both open and laparoscopic operations

#### *Coughing, smoking, and collagen-metabolic disorders*

The inevitable secondary coughing from smoking is the probable reason for the adverse relationship; however, Read has theorized and Jorgensen has proven a relationship between collagen-metabolic disorders and smoking [13]. Regardless, increased intra-abdominal pressure from coughing is a known factor for wound dehiscence [14], and it is recommended that all patients observe smoking-cessation measures preoperatively.

#### *The short esophagus*

For years surgeons have debated the existence of the short esophagus. There is agreement that some patients suffer from severe reflux disease that results in deep ulcerations and, in some circumstances, panmural fibrotic stricture formation. Many surgeons also attest to paraesophageal inflammation found during mediastinal dissection of the esophagus. The significance of this phenomenon and the many points in favor or against the influence of esophageal shortening are beyond the scope of this paper. It is fair to summarize by saying that the number of publications in favor of the concept exceed those opposed. If indeed the short esophagus exists, it does make sense that the incidence of re-herniation

would be increased as the fundoplication is attached to the distal esophagus.

Possible mechanisms of recurrence unique to laparoscopic operations

#### *Premature return to normal activity*

A suspected cause of recurrent hiatal hernia formation after laparoscopic surgery is a too-rapid return to a normal lifestyle. There is a shorter hospital stay because a paralytic ileus is not experienced, plus pulmonary complications are less likely. With decreased postoperative pain, the laparoscopic operation allows the patient to be discharged from the hospital in 1–2 days and usually to be back to their place of work in 7–10 days. The patient expects this, the physician expects this, and the employer expects this, and in many respects, there is no apparent reason not to return to work. The incisional pain has diminished, if not disappeared, normal eating habits have resumed, and physiologically the body has resumed normal functions in every respect. However, the adhesions and eventual fibrosis are still poorly formed at 1 week. Normal activity means normal stresses on the crural closure with only early adhesions to secure the stomach in its anatomical position.

#### *Decreased adhesion formation*

The intraperitoneal reoperative findings and the technical disparities between open and laparoscopic hiatal hernia are considerable, not the least of which is the difference in adhesion formation. In the senior author's experience after performing over 100 laparoscopic reoperative procedures for failed Nissen fundoplication, the patients with open repairs had far more adhesions than the laparoscopic patients. The open operative adhesions almost always involved the anterior abdominal wall binding the stomach to the peritoneum (a pseudogastropexy) but also the under surface of the liver. Although the strong majority of the laparoscopic operations have adhesions to the posterior surface of the lateral segment of the left lobe of the liver, they are in

general grade 2–3 adhesions. The gastric liver adhesions are grade 3 or 4 adhesions.

The grade of adhesions in the hiatus is often similar between the open and laparoscopic repair with the posterior hiatal adhesions being the most dense. Previous dissection in the mediastinum and the amount of gastric or adipose tissue incarcerated within the mediastinum also governs the difficulty of the dissection but is not obviously different between the two techniques.

#### *Less tissue inclusion in the closure*

*Distortion of hiatal structures* The configuration of the hiatus during surgery is different for the laparoscopic approach. With insufflation, there is always an anterior and lateral stretch of abdominal wall and the attached diaphragm, creating a more vertical orientation and attenuation of the diaphragmatic crura. As a result, there is less crural tissue available for suture inclusion, as with the open repair, the crura lie in a more horizontal and relaxed position. In addition, the senior author has noticed when performing an open Nissen fundoplication that Penrose drain retraction is in a more caudad direction rather than anterior and to the left, as when performing a laparoscopic operation. This results in a more anterior stretch of the crura with the laparoscopic technique.

*Different needles* The ski-shaped needle is often used for the laparoscopic operation because the needle passes through a smaller trocar. The needle is shaped from a curvilinear needle; therefore, its length is the same. Sometimes it is awkward to back the needle into the posterior gastroesophageal window because of the needle length. This can result in a more superficial inclusion of the posterior left limb of the right crus. Secondly, the straight configuration makes it more likely to puncture the aorta with the most posterior positioned suture, so the natural tendency is to include less tissue, especially on the left side; the right limb of the right crus is easier to position the suture in because it usually is less bulky and the aorta is out of harm's way. The vena cava is to the right of the right limb but is almost always easy to avoid.

The use of automatic-suture devices should be avoided. The ones available do not include more than 1 cm<sup>2</sup> of tissue, and although easy to use, tissue inclusion appears to be the most critical factor in an effective crural closure.

*The exclusion of the subdiaphragmatic fascia* we have measured the thickness of the left subdiaphragmatic fascia, as compared to the overlying peritoneum of the right limb of the right crus and found it to be four times thicker. The subdiaphragmatic fascia covers the left limb of the right crus but is divided on the medial surface of the left limb during laparoscopic Nissen fundoplication. This is in contrast to the open repair, when a finger is simply placed around the esophagus to create the posterior window. The cutting of the fascia was found to be necessary, but the reason is now suspect. Formerly

laparoscopic surgeons felt this was the only way to safely create the posterior window. Blunt or sharp dissection from the right side was risky. If one now utilizes a left-side-first technique and divides all the short gastric vessels and posterior attachments of the stomach to the pancreas, a window can be easily made if the posterior aspect of the left limb is identified from the right side.

The problem with dividing the left subdiaphragmatic fascia is that it migrates to the left with the anterior and lateral distention of the insufflated abdomen, which makes suture inclusion less likely. It has been well proven by inguinal hernia surgery that muscle apposition will result inevitably in suture pull out and hernia recurrence. To think in any other way seems short-sighted. Thus fascia and peritoneum inclusion in the hiatal closure is probably crucial to the success of the operation. Peritoneal inclusion on the right side is less problematic, perhaps explaining why most of the suture tear out that I have observed comes from the more bulky left limb of the right crus.

#### *Knot tying*

Another potential difference between the open and laparoscopic techniques is the placement and knot-tying sequence. Most surgeons, when performing the open technique, place their sutures and then attach a hemostat to each subsequent suture but do not tie the sutures until all the sutures have been placed. This is in contradistinction to the laparoscopic operation when, after each suture placement, the same suture is tied or secured in whatever other fashion the surgeon chooses. This is to prevent suture fouling, which can more easily happen during laparoscopic operations. This may make the laparoscopic closure less favorable, as the exposure for needle placement is less ideal, and thus the inclusion of tissue becomes more of an issue as the suture placement progresses from posterior to anterior. Suture tying is somewhat more suspect with the extracorporeal technique, as compared to open tying because tactile feedback is less with the former method. The tye-knot device (Ti-knot LSI Solutions, Victor NY USA) has been our choice for suture apposition and does provide better tactile feedback, plus there is only one application rather than 3–6 half hitches, depending upon what type of nonabsorbable suture is utilized.

#### *No hiatal calibration*

Finally there is the issue of hiatal calibration. Many surgeons have avoided dilator hiatal closure calibration because of an early publication that described frequently experienced intraoperative esophageal perforation from the bougie introduced by the anesthetist. A visual estimate has been used, and this increases the risk of a too-loose closure, as opposed to a too-tight closure, as the esophagus often has attached adipose tissue, making it appear larger than it actually is. This, too, then can

lead to the increased incidence of hiatal hernia recurrence, as the fundoplication can more easily migrate intrathoracically (essentially a paraesophageal hiatal hernia).

### Mesh repairs

The placement of an onlay mesh repair may assist in preventing hiatal hernia recurrence (Table 4). There are short follow-up publications that attest to that fact, yet anecdotal cases of mesh erosion into the esophagus are often mentioned by experienced surgeons. Further follow-up and randomized prospective trials are needed to substantiate the true benefits of the mesh onlay repair. In addition, indications and technical precautions when using mesh reinforcement are yet to be well defined.

---

### Discussion

There is little to suggest a learning curve on this basis of Table 2 and Table 3 cumulative recurrence rates; however, the methods used for detection of a recurrent hiatal hernia vary between series. Some investigators routinely perform endoscopies and barium studies for all patients, while others simply follow them clinically. However, some authors comparing their initial cases and later cases note the sharp decline in recurrence rates, apparent evidence that the learning curve is being surmounted [12].

The learning curve or the lack thereof and the mechanisms of hiatal recurrence have been reviewed with the purpose of underlining for practitioners additional measures that can be taken to improve results. All of this may be precluded by mesh repair (Table 4); however, there is an inherent and significant apparent risk with mesh reinforcement. Therefore, continued efforts at improving technique should be sought prior to randomly abdicating to mesh reinforcement. Eventually improved biomaterials that avoid esophageal erosion may be the answer. In concert with their development, further long-term results from trials with mesh will become available, perhaps making the correct choice more apparent to clinicians.

Regardless of the operative technical measures taken, the office staff must emphasize that the success of the operation is partly dependent on the success of preventing vomiting, dry heaving, and constipation. The patient's compliance with rapid reporting of excessive nausea is imperative and must be emphasized. At our institution, patients are routinely given a prescription for antiemetics in addition to written instructions concerning nausea and activity. Our preoperative office instructions explain the importance of avoiding heavy lifting, and if patients are required to lift heavy objects, they are excused from work for 6 weeks.

The primary mechanism of hernia recurrence for inguinal hernias and ventral hernias is closure under tension. This can probably be said for hiatal hernias as well. However, the tension applied to the hiatal closure by normal physiologic forces may be due to the decreased influence of gravity when in the upright position. An additional and confounding factor is the negative pressure of the thoracic cavity and the increased movement in the hiatus and its surrounding organs. Every heartbeat and breath is part of the hiatal dynamic. Peristaltic movement of the gastroesophageal junction may be a factor as well.

Although these considerations are perhaps of interest, there are essentially no data concerning the vector-force analysis experienced in the hiatus. Much additional work is needed to determine the optimal hiatal hernia closure. An intraperitoneal mesh repair, as performed with laparoscopic ventral hernia repair, has been demonstrated to work. Strong lateral attachments are necessary; however, this is difficult to achieve with mesh placed upon the crural closure. Perhaps glue will suffice, but it would be helpful to understand the range of the disruptive forces applied to the hernia repairs mentioned, the adhesive resistant forces created by various meshes, the mesh-tissue interface surface-area ratio resistance, and the fixation devices currently used.

Collagen metabolism remains another significant unknown influence on the recurrence of hiatal hernias. Recent work by Dr. Schumpelick's group demonstrates that patients with recurrent incisional hernia showed the lowest ratios of collagen types I to III in their tissue [15]. The altered collagen ratio might be the result of the decreased activity of MMP-1. The observed alterations in the expression of collagen-interacting proteins again indicate the possibility of a fundamental connective-tissue disease as the causal factor in the pathogenesis of hernias. Obviously, further scientific investigation is required to assist us in understanding the ideal repair and the additional considerations mentioned.

---

### Conclusion

Hiatal hernia recurrence is a problem that must be solved. There are abundant opportunities for further research. The theorized mechanisms need to be proved or disproved. The role of prosthetic mesh in the prevention of hiatal hernia formation is unclear.

---

### References

1. Hashemi M, Peters JH, DeMeester TR, Huprich JE, Quek M, Hagen JA, Crookes PF, Theisen J, DeMeester SR, Sillin LF, Bremner CG (2000) Laparoscopic repair of large type III hiatal hernia: objective followup reveals high recurrence rate. *J Am Coll Surg* 190:553-560

2. Jobe BA, Aye RW, Deveney CW, Domreis JS, Hill LD (2002) Laparoscopic management of giant type III hiatal hernia and short esophagus. Objective follow-up at three years. *J Gastrointest Surg* 6:181–188
3. Diaz S, Brunt LM, Klingensmith ME, Frisella PM, Soper NJ (2003) Laparoscopic paraesophageal hernia repair, a challenging operation: medium-term outcome of 116 patients. *J Gastrointest Surg* 7:59–66
4. Kakarlapudi GV, Awad ZT, Haynatzki G, Sampson T, Stroup G, Filipi CJ (2002) The effect of diaphragmatic stressors on recurrent hiatal hernia. *Hernia* 6:163–166
5. Mittal SK, Filipi CJ, Anderson PI, Fenton SJ, Cummings JE, Cornet D, Quinn TH, Fitzgibbons RJ (1999) Additional mechanisms of hiatal hernia recurrence and prevention. *Hernia* 3:215–220
6. Champion JK, Rock D (2003) Laparoscopic mesh cruroplasty for large paraesophageal hernias. *Surg Endosc* 17:551–553
7. Frantzides CT, Madan AK, Carlson MA, Stavropoulos GP (2002) A prospective, randomized trial of laparoscopic polytetrafluoroethylene (PTFE) patch repair vs simple cruroplasty for large hiatal hernia. *Arch Surg* 137:649–652
8. Orringer MB, Skinner DB, Belsey RH (1972) Long-term results of the Mark IV operation for hiatal hernia and analyses of recurrences and their treatment. *J Thorac Cardiovasc Surg* 63:25–33
9. Caniano DA, Ginn-Pease ME, King DR (1990) The failed antireflux procedure: analysis of risk factors and morbidity. *J Pediatr Surg* 25:1022–1025
10. Edye MB, Canin-Endres J, Gattorno F, Salky BA (1998) Durability of laparoscopic repair of paraesophageal hernia. *Ann Surg* 228:528–535
11. Watson DI, Davies N, Devitt PG, Jamieson GG (1999) Importance of dissection of the hernial sac in laparoscopic surgery for large hiatal hernias. *Arch Surg* 134:1069–1073
12. Leeder PC, Smith G, Dehn TC (2003) Laparoscopic management of large paraesophageal hiatal hernia. *Surg Endosc* 17:1372–1375
13. Jorgensen LN, Kallehave F, Christensen E, Siana JE, Gottrup F (1998) Less collagen production in smokers. *Surgery* 123:450–455
14. Makela JT, Kiviniemi H, Juvonen T, Laitinen S (1995) Factors influencing wound dehiscence after midline laparotomy. *Am J Surg* 170:387–390
15. Rosch R, Junge K, Knops M, Lynen P, Klinge U, Schumpelick V (2003) Analysis of collagen-interacting proteins in patients with incisional hernias. *Langenbecks Arch Surg* 387:427–432
16. Nicholson DA, Nohl-Oser HC (1976) Hiatus hernia: a comparison between two methods of fundoplication by evaluation of the long-term results. *J Thorac Cardiovasc Surg* 72:938–943
17. Mokka RE, Punto L, Kairaluoma MI, Laitinen S, Larmi TK (1977) Surgical treatment of axial hiatal hernia-reflux complex by Nissen fundoplication. A cineradiologic and manometric study. *Acta Chir Scand* 143:265–269
18. Gardner RJ, Bonnabeau RC Jr, Warden HE (1977) Surgical repair of gastroesophageal reflux with sliding hiatal hernia. *Am J Surg* 133:554–556
19. Gatzinsky P, Sandberg N, Sihlbom H (1979) Gastro-oesophageal reflux after surgical treatment of hiatal hernia with and without severe reflux complications. A follow-up study. *Acta Chir Scand* 145:45–53
20. Refsum S Jr, Nygaard K (1979) Repair of hiatus hernia by an abdominal semi-fundoplication technique. *Acta Chir Scand* 145:39–44
21. Singh SV (1980) Surgery for oesophageal reflux and hiatus hernia. Long-term results in 250 patients. *Scand J Thorac Cardiovasc Surg* 14:311–315
22. Pearson FG, Cooper JD, Ilves R, Todd TR, Jamieson WR (1983) Massive hiatal hernia with incarceration: a report of 53 cases. *Ann Thorac Surg* 35:45–51
23. Ellis FH Jr, Crozier RE, Shea JA (1986) Paraesophageal hiatus hernia. *Arch Surg* 121:416–420
24. Williamson WA, Ellis FH Jr, Streitz JM Jr, Shahian DM (1993) Paraesophageal hiatal hernia: is an antireflux procedure necessary? *Ann Thorac Surg*. 56:447–451
25. Weissberg D, Refaely Y (1995) Symptomatic diaphragmatic hernia: surgical treatment. *Scand J Thorac Cardiovasc Surg* 29:201–206
26. Schauer PR, Ikramuddin S, McLaughlin RH, Graham TO, Slivka A, Lee KK, Schraut WH, Luketich JD (1998) Comparison of laparoscopic versus open repair of paraesophageal hernia. *Am J Surg* 176:659–665
27. Rogers ML, Duffy JP, Beggs FD, Salama FD, Knowles KR, Morgan WE (2001) Surgical treatment of para-oesophageal hiatal hernia. *Ann R Coll Surg Engl* 83:394–398
28. Perdakis G, Hinder RA, Filipi CJ, Walenz T, McBride PJ, Smith SL, Katada N, Klingler PJ (1997) Laparoscopic paraesophageal hernia repair. *Arch Surg* 132:586–589
29. Huntington TR (1997) Short-term outcome of laparoscopic paraesophageal hernia repair. A case series of 58 consecutive patients. *Surg Endosc* 11:894–898
30. Gantert WA, Patti MG, Arcerito M, Feo C, Stewart L, DePinto M, Bhojru S, Rangel S, Tyrrell D, Fujino Y, Mulvihill SJ, Way LW (1998) Laparoscopic repair of paraesophageal hiatal hernias. *J Am Coll Surg* 186:428–432
31. Hawasli A, Zonca S (1998) Laparoscopic repair of paraesophageal hiatal hernia. *Am Surg* 64:703–710
32. Horgan S, Eubanks TR, Jacobsen G, Omelanczuk P, Pellegrini CA (1999) Repair of paraesophageal hernias. *Am J Surg* 177:354–358
33. Wiechmann RJ, Ferguson MK, Naunheim KS, McKesey P, Hazelrigg SJ, Santucci TS, Macherey RS, Landreneau RJ (2001) Laparoscopic management of giant paraesophageal herniation. *Ann Thorac Surg* 71:1080–1086
34. Swanstrom LL, Jobe BA, Kinzie LR, Horvath KD (1999) Esophageal motility and outcomes following laparoscopic paraesophageal hernia repair and fundoplication. *Am J Surg* 177:359–363
35. Carlson MA, Richards CG, Frantzides CT (1999) Laparoscopic prosthetic reinforcement of hiatal herniorrhaphy. *Dig Surg* 16:407–410
36. Pierre AF, Luketich JD, Fernando HC, Christie NA, Buenaventura PO, Litle VR, Schauer PR (2002) Results of laparoscopic repair of giant paraesophageal hernias: 200 consecutive patients. *Ann Thorac Surg* 74:1909–1915
37. Balsara KP, Shah CR, Bhandari M (2002) Laparoscopic surgery for reflux esophagitis and paraesophageal hernia. *Indian J Gastroenterol* 21:102–104
38. Khaitan L, Houston H, Sharp K, Holzman M, Richards W (2002) Laparoscopic paraesophageal hernia repair has an acceptable recurrence rate. *Am Surg* 68:546–551
39. Dahlberg PS, Deschamps C, Miller DL, Allen MS, Nichols FC, Pairolo PC (2001) Laparoscopic repair of large paraesophageal hiatal hernia. *Ann Thorac Surg* 72:1125–1129
40. Velanovich V, Karmy-Jones R (2001) Surgical management of paraesophageal hernias: outcome and quality of life analysis. *Dig Surg* 18:432–437
41. Mattar SG, Bowers SP, Galloway KD, Hunter JG, Smith CD (2002) Long-term outcome of laparoscopic repair of paraesophageal hernia. *Surg Endosc* 16:745–749
42. Keidar A, Szold A (2003) Laparoscopic repair of paraesophageal hernia with selective use of mesh. *Surg Laparosc Endosc Percutan Tech* 13:149–154
43. Carlson MA, Condon RE, Ludwig KA, Schulte WJ (1998) Management of intrathoracic stomach with polypropylene mesh prosthesis reinforced transabdominal hiatus hernia repair. *J Am Coll Surg* 187:227–230
44. Hui TT, Thoman DS, Spyrou M, Phillips EH, David T (2001) Mesh crural repair of large paraesophageal hiatal hernias. *Am Surg* 67:1170–1174
45. Livingston CD, Jones HL Jr, Askew RE Jr, Victor BE, Askew RE Sr (2001) Laparoscopic hiatal hernia repair in patients with poor esophageal motility or paraesophageal herniation. *Am Surg* 67:987–991

46. Casaccia M, Torelli P, Panaro F, Cavaliere D, Ventura A, Valente U (2002) Laparoscopic physiological hiatoplasty for hiatal hernia: new composite "A"-shaped mesh. Physical and geometrical analysis and preliminary clinical results. *Surg Endosc* 16:1441–1445
47. Granderath FA, Kamolz T, Schweiger UM, Pointner R (2003) Laparoscopic refundoplication with prosthetic hiatal closure for recurrent hiatal hernia after primary failed antireflux surgery. *Arch Surg* 138:902–907