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Effect of Hiatal Hernia on Esophageal Manometry and pH-Metry in Gastroesophageal Reflux Disease

PANAGIOTIS KASAPIDIS, MD, JOHN SOPHOCLES VASSILAKIS, MD, GEORGE TZOVARAS, MD, EMMANUEL CHRYSOS, MD, and EVAGHELOS XYNOS, MD

An increased frequency of reflux events and a prolonged acid clearance have been shown in gastroesophageal reflux (GER) patients with a hiatal hernia as compared to those without. The objective of the present study was to further investigate esophageal motility and patterns of reflux in GER patients, in relation to the presence or absence of hiatal hernia. Esophageal manometry and ambulatory 24-hr esophageal pH-metry were used in 42 patients with GER and 18 controls. Eighteen of the patients were considered to have a nonreducing hiatal hernia on endoscopy. Hiatal hernia patients showed a higher extent of reflux (total composite score, $P = 0.016$; total reflux time, $P = 0.008$, reflux time in supine position, $P = 0.024$; reflux time in upright position, $P = 0.008$), a lower frequency of reflux events ($P = 0.005$), a more severe esophagitis on endoscopy ($P < 0.01$) and a lower amplitude of peristalsis at 5 cm proximal to LES ($P = 0.0009$) as compared to patients without hiatal hernia. The amplitude of peristalsis at the distal esophagus was inversely related to the extent of reflux ($P = 0.024$). Acid clearance was also significantly prolonged in the hernia subgroup ($P = 0.011$). Although LES resting pressure did not differ significantly between the two subgroups of patients, it was inversely related to the extent of reflux in the patients with hiatal hernia ($P = 0.0005$). It is concluded, that GER patients with hiatal hernia present with an increased amount of reflux and more severe esophagitis, which results in more severely impaired esophageal peristalsis as compared to patients without hernia. Prolonged acid clearance and impaired esophageal emptying observed in patients with hiatal hernia could be the result of both the presence of the hernia itself and the reduced peristaltic activity of the esophagus.

KEY WORDS: gastroesophageal reflux; hiatal hernia; esophageal acid clearance; esophageal emptying; esophageal peristalsis; esophageal manometry.

The importance of hiatal hernia in the pathogenesis of gastroesophageal reflux disease (GERD) has been controversial. Before 1970, the presence of hiatal hernia was considered to be the key factor of the disease. However, the observations, according to which (a) the presence or absence of hiatal hernia failed to distinguish patients with GERD from asymptomatic subjects (1); (b) half of the normal population over the age of 50 years had radiologically proven hiatal hernia (2); (c) only 9% of the individuals with proven hiatal hernia had typical symptoms suggesting GER, of whom only the one third had esophagitis (3); and (d) a significant percentage of patients with GERD and esophagitis have no hiatal hernia whatsoever (4), shifted the interest towards other pathogenetical factors.

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From the Department of Gastroenterology of the Athens Naval and Veterans Hospital and the Department of General Surgery, Medical School, University of Crete, Heraklion, Greece.

Address for reprint requests: E. Xynos, University Hospital of Heraklion, Stavrakia, Heraklion, Crete, GR-711 10, Greece.

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For almost the last two decades, incompetence, primarily of the lower esophageal sphincter (LES) (4, 5), as well as impaired esophageal body motility (6, 7) and delayed gastric emptying (8, 9) are considered to be the main contributing factors in GERD, by promoting esophageal mucosa contact with acid, pepsin, or even duodenal secretions. However, there have been some recent data that refocus current concepts of GERD pathogenesis on the incompetence of diaphragmatic crura and the presence of hiatal hernia. First of all, more than 80% of patients with reflux esophagitis and peptic esophageal stricture have hiatal hernia, as compared to only 13% of patients without esophagitis (10, 11). Furthermore, in an analysis of 184 patients with reflux symptoms, the presence of hiatal hernia was strongly associated with low LES pressure, increased acid exposure of the esophagus, and endoscopic esophagitis (12). In addition, it has been shown that the hiatal hernia delays esophageal acid clearance (13), whereas reflux events related to intraabdominal pressure increases are most likely to occur in the presence of a large hiatal hernia and a low LES pressure (14).

The implication of the above observations is that under normal conditions the contraction of the crural diaphragm, during increases of the intraabdominal pressure, adds to LES pressure and thus to the competence of gastroesophageal junction. Failure of this mechanism, due to the presence of a gaping hiatus, leads to gastroesophageal reflux. In an effort to further elucidate the issue, we investigated the effect of hiatal hernia on esophageal motility and on the parameters of reflux, as measured by pH-metry, in patients with symptoms suggestive of GERD.

MATERIALS AND METHODS

Patients. Studies were performed to 18 healthy volunteers (male/female: 14/4, mean age: 38 ± 13 SD years, range: 21–73 years) and 42 patients (male/female: 38/4, mean age: 37 ± 13 SD years, range: 22–72 years). All patients were consecutively included in the study after having had abnormal 24-hr ambulatory esophageal pH monitoring results. In addition, they had to experience GER symptoms for at least four months prior to investigation and to have not received antireflux treatment other than antacids. All subjects had upper alimentary endoscopy, esophageal manometry, and 24-hr ambulatory esophageal pH-metry within a three-day period. The patients were divided into two groups according to the presence or absence of hiatal hernia as noted on endoscopy. A hiatal hernia on endoscopy was defined as a pouch of gastric mucosa longer than 2 cm, confined between the endoscopic squamocolumnar junction and the diaphragmatic hiatal dentation on the stomach, at rest. When in doubt of the diagnosis of hiatal hernia on endoscopy, the subjects were not included in the study (15–17). The study was approved by the Ethical Committee of the Athens Naval and Veterans Hospital.

Upper Alimentary Endoscopy. At upper alimentary endoscopy, the presence of a sliding hernia, as mentioned above, and the grading of esophagitis, according to Savary-Miller criteria (18), were identified. Grading of esophagitis was as follows: grade 0 for normal; grade I for reddening of the distal esophagus; grade II for linear, longitudinal, or circumferential confluent erosions not involving the whole periphery of the lumen; grade III for grade II lesions that involve the whole periphery of the distal esophageal lumen; and grade IV for extensive grade III lesions with true ulceration. Cases with strictures were excluded. Because of difficulties in interpreting grade I esophagitis, grades 0 and I were considered to be normal, while grades II, III, and IV were considered to be abnormal.

Manometric Recordings. A four-lumen manometric catheter, 4 mm in external diameter, constantly perfused by water (0.6 ml/min, Arndorfer capillary perfusion system), was used. The lateral openings were circumferentially oriented at 90° to each other and placed at 3, 8, 13, and 18 cm from the distal tip of the assembly. A pressure transducer, incorporated to each perfusion line, was connected to an amplifier and a chart recorder (Beckman). The station pull-through technique was applied twice to measure the resting lower esophageal sphincter pressure (LESP) (mean maximal value of pressures from each of the four side holes at the mid-expiratory sphincter pressure minus the end-expiratory intragastric pressure) and the total and intraabdominal length of the LES. Then the assembly was positioned, with the distal recording site situated at the upper limit of the LES and the other three openings in the lumen of the esophageal body. At this position, subjects were instructed to swallow 5 ml of water on 10 occasions, with an interval of 30 sec between swallows. The duration of the postdeglutition LES relaxation; the amplitude of peristaltic contractions at 5, 10, and 15 cm proximal to LES; the velocity of peristalsis at 5 cm proximal to LES; and the duration of peristalsis at 10–5 cm proximal to LES were calculated as the mean values obtained from the 10 swallows.

Ambulatory 24-Hour Esophageal pH-metry. The glass electrode of the pH-meter, along with an intraluminal reference electrode, were transnasally introduced and positioned 5 cm proximal to the upper limit of the manometrically defined LES. Sampling occurred every 6 sec and data were stored in a portable digital data recorder (Oxford Medilog 1010). Recording an esophageal pH less than 4 for at least 18 sec was considered to be a reflux episode. Subjects were instructed to perform their daily activities and to follow a free diet, avoiding only acid juices, chocolate, and alcohol. From pH-metry, the following variables were calculated: (a) the total composite score (19), (b) the total percent of reflux time, (c) the percent of reflux time in the upright position, (d) the percent of reflux time in the supine position, (e) the total number of reflux episodes, (f) the total number of reflux episodes more than 5 min in duration, (g) the duration of longest reflux episode, (h) the acid clearance time (reflux in minutes per total number of reflux episodes), and (i) the frequency–duration index (FDI is the product of the mean number of reflux episodes per

TABLE 1. ENDOSCOPIC GRADING OF ESOPHAGITIS IN PATIENTS WITH GER

	Hiatal hernia	
Endoscopic grading	*Without*	*With*
Grade 0 and 1	18	3
Grade 2	4	10
Grade 3	1	4
Grade 4	1	1

Patients with hiatal hernia versus patients without hiatal hernia significant difference in severity of esophagitis: $P < 0.01$.

hour multiplied by the duration of reflux episodes per hour). A variable greater than the mean value plus 2 SD of the controls was considered to be abnormal.

Statistical Evaluation. All quantitative data were expressed as mean ± SD. The Mann-Whitney U test for unpaired values and the chi-square test were applied, as appropriate, to identify differences between groups. Regression analysis was used to identify any possible correlation between different parameters in the same group. $P < 0.05$ was considered to be statistically significant.

RESULTS

Endoscopy was negative for hiatal hernia or esophagitis in the controls. Hiatal hernia was present in 18 of the 42 patients (43%). Mean age and body weight did not differ significantly between the two groups of patients (without hiatal hernia: mean age: 35 ± 11 years, body weight: 76 ± 12 kg; with hiatal hernia: mean age: 39 ± 16 years, body weight: 77 ± 8 kg). Table 1 shows endoscopic grading of esophagitis in both groups of patients. Esophagitis was significantly more severe in patients with hiatal hernia, as compared to those without ($P < 0.01$).

There were significant differences between controls and patients in most of the manometric variables recorded. However, no significant differences were found between patients without and those with hiatal hernia, as far as the manometric variables characterizing the LES are concerned. Patients with hiatal hernia had a significantly lower amplitude of peristalsis at 5 cm proximal to LES as compared to patients without hiatal hernia (67.3 ± 33 vs 105.9 ± 41 mm Hg; $P = 0.0009$), and this was the only significant manometric difference noted between the two subgroups of patients (Figure 1).

Similarly, almost all esophageal pH-metric variables were significantly higher in the patients with

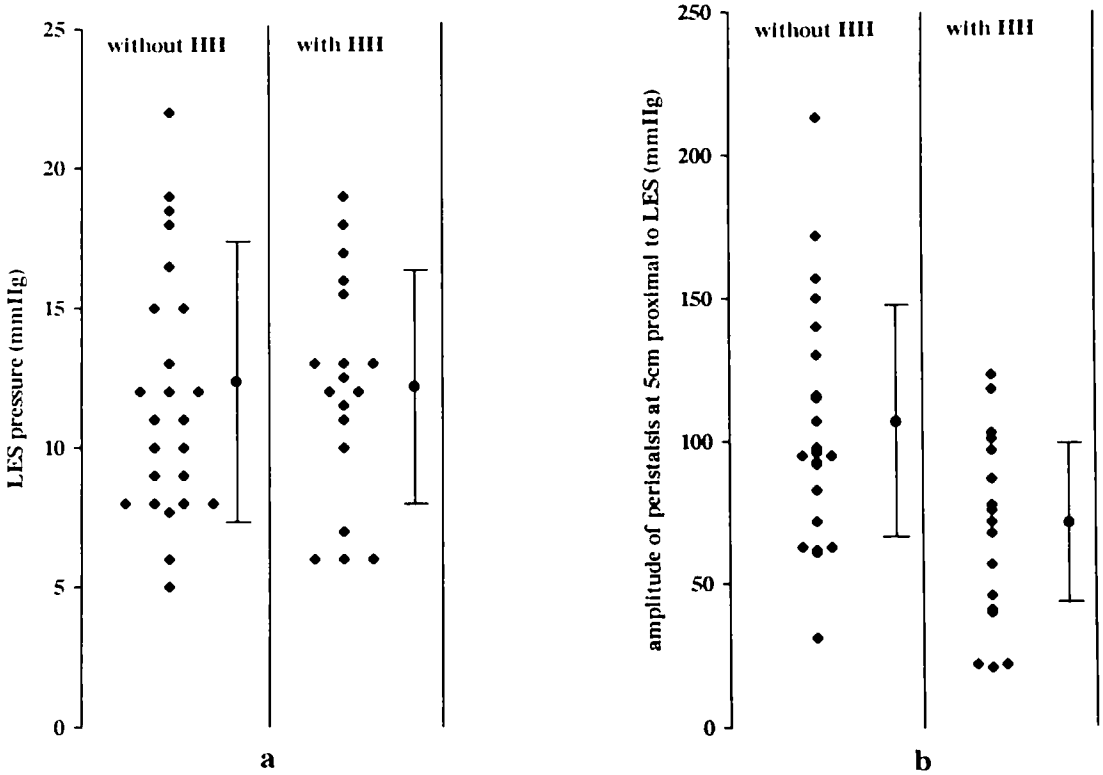

Fig 1. (a) There was no significant difference in the resting pressure of the LES between the patients with and the patients without hiatal hernia. (b) Despite the overlap of values, the patients with hiatal hernia showed a significantly lower amplitude of peristalsis at 5 cm proximal to LES ($P = 0.0009$) as compared to those without hernia.

GER as compared to the controls. In addition, patients with hiatal hernia presented significantly higher: (a) total composite score (142 ± 163 vs 50.9 ± 25.9; $P = 0.016$), (b) total reflux time (28.1 ± 25.3% vs 11.8 ± 5.2%; $P = 0.008$), (c) reflux time in both upright (27.9 ± 22.3% vs 13.4 ± 6.5%; $P = 0.008$) and supine (27.6 ± 37.1% vs 8.6 ± 8.2%; $P = 0.024$) position, (d) number of reflux episodes lasting for more than 5 min (8.1 ± 3.8 vs 5.2 ± 3.4; $P = 0.008$), and (e) frequency–duration index (21.2 ± 20.5 vs 11.8 ± 12.3; $P = 0.05$) as compared to those without hiatal hernia. Furthermore, although the number of reflux episodes was significantly higher in the nonhiatal hernia patients (51.2 ± 23.9 vs 35.1 ± 14.5; $P = 0.005$), the acid clearance time was significantly prolonged in the hiatal hernia subgroup of patients (16 ± 20.5 min/*N* vs 4.1 ± 3 min/*N*; $P = 0.011$) (Figure 2).

A significant inverse correlation was found between the LES resting pressure and the total composite score on pH-metry in the subgroup of patients with hiatal hernia ($r = 0.66$, $P = 0.0005$). Furthermore, the amplitude of peristalsis 5 cm proximal to LES was significantly inversely related to the total composite score in the same subgroup of patients ($r = 0.53$, $P = 0.024$). In other words, the higher the extent of gastroesophageal reflux, the lower the LES resting pressure and the amplitude of esophageal peristalsis in patients with hiatal hernia.

DISCUSSION

Much of the controversy as to whether the presence of hiatal hernia is of significant pathogenetic importance in GERD should be attributed to the definition of hernia. In patients with a reducing hiatal hernia between swallows, esophageal emptying and clearance is not impaired as compared to normal subjects. In contrast, patients with a hiatal hernia longer than 2 cm that is not reduced between swallows present with significantly delayed esophageal emptying and acid clearance of the esophagus (13, 20, 21). In the present study, the patients allocated to the hiatal hernia group had a gastric pouch above the diaphragmatic dentation of at least 2 cm in length at rest (between any incidental swallows) that persisted throughout the endoscopic study. We believe that these patients should be considered as having a nonreducing hiatal hernia, according to the definition of Sloan and Kahrilas (13), although this was not confirmed on barium swallow.

The extent of gastroesophageal reflux, as mainly expressed by the total composite score and the total and during either upright or supine position reflux time on ambulatory esophageal pH-metry, was significantly higher in patients with hiatal hernia as compared to those without. Furthermore, reflux esophagitis on endoscopy was significantly more severe in the group of patients with a hiatal hernia, as also shown by others (22–24). This finding should be attributed to the fact that the severity of reflux esophagitis is related to prolonged and infrequent reflux episodes rather than to short and frequent ones (7, 25–27). Indeed, the patients of the present study with a hiatal hernia exhibited a significantly increased duration of the longest reflux episode and a significantly smaller number of reflux episodes as compared to those without hiatal hernia.

The amplitude of peristalsis at 5 cm proximal to LES was found to be significantly lower in patients with a hiatal hernia as compared to those without, and this was an original finding of the present study. Although peristalsis of esophageal body is, at least to a degree, primarily impaired in the GERD (5, 9, 22), motor function of the esophagus further deteriorates with severity of esophagitis, because of the persistent reflux (6, 9, 28). The degree of impairment of peristalsis at the distal esophageal body is related both to the extent of the reflux and to the severity of esophagitis (7). Therefore, it can be speculated that an increased extent of reflux, observed in patients with hiatal hernia, results in more severe inflammation of the esophageal mucosa and thus to further impairment of peristalsis of the distal esophagus. This hypothesis is additionally supported by the finding of the present study, according to which the extent of reflux is significantly inversely related to the amplitude of peristalsis of the distal esophagus in patients with hiatal hernia.

An opposite hypothesis has been also stated to interpret the association of severe esophagitis with hiatal hernia. Instead of hiatal hernia being the cause of increased GER and severity of esophagitis, it has been speculated that hiatal hernia may be the consequence of shortening of the esophagus as a result of esophagitis (29). However, this hypothesis cannot explain the presence of hiatal hernia in some of the patients with GER and without esophagitis. The role of hiatal hernia in the severity of esophagitis would be strengthened, provided the aforementioned differences in amplitude of peristalsis and pH-metric variables had been demonstrated in patients with similar grades of esophagitis and with or without hiatal hernia. However, the number of patients included in the present study with esophagitis grade III or IV was

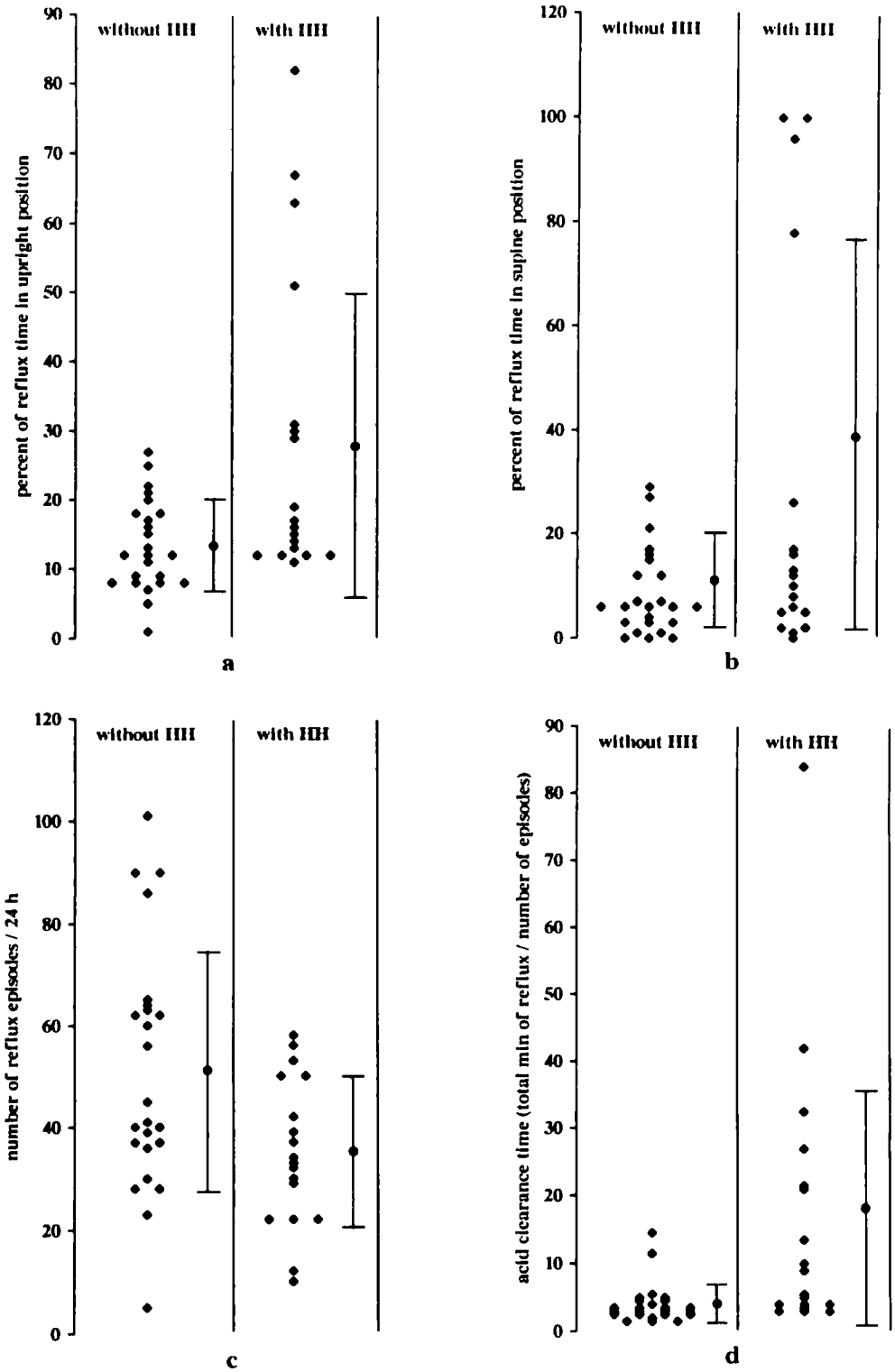

Fig 2. Significant differences in most of the pH-metry variables were found between the two subgroups of patients. The patients with hiatal hernia had a higher percentage of reflux time in both upright ($P = 0.008$) (a) and supine ($P = 0.024$) (b) positions, a decreased number of reflux episodes ($P = 0.005$) (c), and a prolonged acid clearance time ($P = 0.011$) (d), as compared to the patients without hiatal hernia.

small, and hence such comparisons were not possible. Furthermore, patients with hiatal hernia tended to exhibit the more severe grades of esophagitis, while patients with GER but without hiatal hernia showed less severe esophagitis.

It has been shown that esophageal acid clearance is severely prolonged in GER patients with hiatal hernia (13, 15, 16), and this was also confirmed by the findings of the present study. As mentioned above, impaired acid clearance and delayed esophageal emptying in hiatal hernia patients with reflux has been attributed to the presence of the hernia itself because of an early or late retrograde flow, depending on whether the hernia is a nonreducing or a reducing one (15). However, considering that the impaired motility of distal esophagus parallels the delay of esophageal transit and emptying in GERD (9), it is conceivable that the delayed esophageal clearance in hiatal hernia is, at least partly, the result of the inability of the impaired distal esophageal peristalsis to promptly reject the refluxing material back to the stomach.

The amplitude of peristalsis at 5 cm proximal to LES was the only esophageal manometric parameter that was found to be impaired in patients with GER and hiatal hernia. The mean resting LES pressure, although lower than in controls, did not differ between the two subgroups of patients. This means that we failed to confirm the findings of other authors, according to which the presence of hiatal hernia is associated with a significantly lower LES resting pressure (13, 14). This discrepancy could be the result of differences in manometric methodology. However, the importance of LES incompetence in reflux associated with hiatal hernia cannot be overlooked, even by the findings of the present study, as it was shown that the lower the LES resting pressure, the higher the extent of reflux.

In conclusion, GER patients with hiatal hernia present with a greater extent of reflux and more severe esophagitis that results in further impairment of distal esophagal body motility as compared with GER patients without hiatal hernia. It remains uncertain whether the delayed acid clearance and emptying of the esophagus in this subgroup of patients is the result of hiatal hernia, the severely impaired peristalsis of the distal esophagus, or both.

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