Defining GERD

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"It is not the death of GERD that I seek, but that it turns from its evil ways and follows the path of righteousness."

The reflux world is fully aware of what GERD is and what GERD does. What the world does not know, however, is the answer to the most important yet least asked question surrounding GERD’s raison-d’etre: Why is GERD here and why do we have it?

What GERD is: abnormal gastric reflux into the esophagus that causes any type of mischief.

What GERD does: causes discomfort and/or pain with or without destroying the mucosa; causes stricture or stenosis, preventing food from being swallowed; sets the stage for the development of esophageal adenocarcinoma; invades the surrounding lands to harass the peaceful oropharyngeal, laryngeal and broncho-pulmonary territories; reminds us that we are not only human, but that we are dust and ashes.

Why GERD is here: We propose three separate and distinct etiologies of GERD, and we offer the following three hypotheses to explain why, after 1.5 million years of standing erect, we have evolved into a species (specifically Homo sapiens sapiens) that is destined to live with the scourge of GERD.

Hypothesis 1: congenital. The antireflux barrier, comprising the smooth-muscled lower esophageal sphincter, the skeletal-muscled right crural diaphragm and the phreno-esophageal ligament does not completely develop due to a developmental anomaly or incomplete gestation.

Hypothesis 2: acute trauma: The antireflux barrier in adults suffering acute traumatic injury to the abdomen or chest is permanently disrupted by unexpected forces, such as motor vehicle accidents (with steering wheel crush impact), blows to the abdomen (from activities such as boxing, etc.), heavy lifting or moving (e.g., pianos, refrigerators) or stress positions (e.g., hand stands on parallel gym bars). The trauma creates a hiatal hernia that renders the antireflux mechanism useless and incapable of preventing GERD.

Hypothesis 3: chronic trauma: The antireflux barrier in children and adults is gradually weakened over time as a result of chronic straining to defecate and straining in an unphysiologic position, both of which stem from our modern day habits of eating a low-fiber diet and living on the high-seated toilet.

We suggest that the chronic traumatic hiatal hernia is (a) the cause of more than 90 percent of the GERD that stalks the Western world; (b) is a direct result of abandoning the popular and worldwide practice of squatting to socialize, eat and defecate; and (c) our just reward for adopting the "civilized" high sitting position on chairs and modern toilets.

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bAbbreviations: GERD, gastroesophageal reflux disease; LES, lower esophageal sphincter.
INTRODUCTION

Abdominal distress has been recorded ever since humans began to express themselves on stone. Throughout the ages, the symptoms have been recognized by almost every civilization. The exact origins of these distresses will never be known, and modern day investigators can feel free to attribute them to the gastrointestinal disease currently being discussed. Indeed, the same abdominal symptoms can be attributed to dyspepsia, duodenal ulcer, gastric ulcer, GERD, chronic constipation and even diarrhea. More than 4000 years ago, the Chinese reportedly used extracts of seminal fluid and baby urine to relieve dyspeptic symptoms. In the first century, Caius Pliny, the Roman elder, effectively treated dyspepsia with coral powder [1] and milk [2]: “if there be an ulcer growne in the stomacke, drinke the milke of an asse or cow, and it will heale it.” By the seventh century, dyspepsia was being managed by Paulus of Aegina with kaolin and by Paracelsus in the sixteenth century with powder of pearls [2]. During the second millennium, sufferers with dyspepsia symptoms were offered, in addition to antacid powders and milk, everything from herbal extracts to religion, witchcraft to starvation, and magic to extremist diets. Some treatments included application of leeches and poultices and internal administration of arsenic, silver nitrate, carbolic acid, cannabis indica and cocaine [3].

In the early nineteenth century, Abercrombie of Edinburgh wrote “...food must be in very small quantities and of the mildest quality, consisting chiefly or entirely of farinaceous articles and milk...” [4]. He advocated the use of oxide of bismuth, lime water and nitric acid for severe abdominal symptoms. By the end of the nineteenth century, milk and antacids had become respectable therapeutic agents for the treatment of indigestion and dyspepsia. In 1915, based on the concept that neutralization of free acid would inhibit gastric juice digestion, Bertram Sippy proposed that ulcers be treated with hourly feeding of milk and antacids [5]. Since that time, and until the early 1960s, Sippy’s antacid-milk regimen remained the principal treatment of peptic diseases.

GERD: THE FIRST WORD

Throughout the eighteenth and nineteenth centuries, the recorded causes of esophageal disease included just about everything except reflux of gastric acid into the esophagus. Indeed, in such text books as Heberden’s The History and Cure of Diseases and Osler’s The Principles and Practice of Medicine [6, 7], the causes of esophageal diseases and strictures included mechanical, thermal and chemical irritants; syphilis, tuberculosis and actinomycosis; and neoplasms. A common cause of esophageal perforation was sword swallowing.

Before 1906, only 44 reported cases of “peptic ulcer of the esophagus” had been reported. In 1929, Jackson [8] described 88 cases of esophageal ulcer observed endoscopically. It was not until 1935, however, that Winkelstein [9] first defined reflux esophagitis clinically. Winkelstein described for the first time five patients with a disease caused by reflux of gastric acid into the esophagus.

In 1946, Allison introduced the hiatal hernia as the major villain in the reflux esophagitis battles. He equated the presence of esophageal ulcer with that of hiatal hernia:

Peptic ulcer of the esophagus has usually been considered a rarity and to be found mainly where there is a congenitally short esophagus. It now seems nearer the truth to say that ulcer occurs where there is such a derangement of the mechanism of the cardia that acid gastric juice flows back easily into the lower end of the esophagus. The disorder which predisposes to ulceration is hernia of the stomach through the diaphragmatic hiatus into the poste-
ri or mediastinum. The hernia may be congenital or acquired, and may be reducible or irreducible at operation. Irreducibility may be caused by ulceration, fibrosis, and shortening of the esophagus [10].

Five years later, in 1951, Allison described in anatomical detail the sliding (axial) hernia and suggested that surgical treatment be directed at repair of the anatomical defect (the hiatal hernia) [11]. For the next 20 years, hiatal hernia was a synonym for reflux esophagitis, and the role of the lower esophageal sphincter (LES)b in reflux esophagitis was rarely mentioned.

In 1971, however, the primacy of the hiatal hernia over the LES came to an end with a publication by Cohen et al. in the New England Journal of Medicine [12]. Cohen concluded that the LES pressure, not the hiatal hernia, was the main determinant for symptomatic reflux disease. The hiatal hernia proponents were not deterred, and the debate continued.

Throughout the debate, one particular observation became generally accepted: that hiatal hernia almost always accompanies severe reflux esophagitis. In endoscopic studies, up to 80 percent of patients with esophagitis have hiatal hernias, and more than half the patients with hiatal hernias have esophagitis. Most important, esophagitis is almost always absent if hiatal hernia is absent [13]. Thus, the preponderance of evidence suggests that the finding of reflux-induced severe esophageal mucosal disease almost guarantees the presence of a hiatal hernia.

During the 1980s, evidence accumulated to support the concept of an intricate antireflux mechanism in which the LES was only one component. Hill described a complex antireflux barrier consisting of the LES, the diaphragm, a gastroesophageal "flap" valve, and an anchored gastroesophageal junction [14]. The "flap" valve is thought to consist of the oblique or sling fibers of the stomach itself, which cause the gastric fundus and intra-gastric pressure to impinge on the distal end of the esophagus to enhance the antireflux barrier.

In further studies of the hiatal mechanism, Sontag investigated 184 symptomatic reflux patients who had received endoscopy and 24-hour pH testing as part of their diagnostic workup [15]. The mechanism by which gastric acid caused esophagitis was tested using clinically relevant models of GER. The results strongly supported the concept of a complex hiatal mechanism. Figure 1 shows that although the LES pressure was significantly lower in the presence of a hiatal hernia, the LES pressure, by itself, had little effect on acid reflux or the development of esophagitis. The presence of the hiatal hernia, by increasing the frequency of reflux episodes, was consistently and strongly associated with esophageal mucosal damage. Figure 2 demonstrates clinically the influence of hiatal hernia on the frequency of reflux episodes and esophagitis.

A recent series of elegantly designed studies by Mittal has greatly clarified the crucial role of the diaphragm as the normal effective antireflux barrier [16-18]. Mittal defines the anatomical antireflux barrier as the fortress comprising the LES, the crural diaphragm phrenoesophageal ligament. The development of a hiatal hernia alters the normal anatomic arrangement by separating the LES from the crural diaphragm and preventing the two from working together to prevent high-pressure-induced GER. Finally, support for the critical role of the entire hiatal mechanism and a non-critical role for the LES is provided by the results of surgery. Surgical restoration of the altered anatomic arrangements improves and heals esophagitis without necessarily resulting in a substantial increase in the LES pressure [18]. Esophageal mucosal damage, therefore, results from the overall effect of an incompetent antireflux barrier in the form of an incompetent crural diaphragm, which promotes frequent reflux episodes, delayed
Figure 1. Final structural model of GER. Numbers represent structural coefficients. All clinically relevant and statistically significant parameters included and all non-significant parameters deleted ($\chi^2 = 2.31$; Tucker-Lewis coefficient [TLC] = 1.009). Although the LES pressure is significantly lower in the presence of a hiatal hernia, the LES pressure, by itself, has little effect on acid reflux or the development of esophagitis. Thus the presence of the hiatal hernia, by increasing the frequency of reflux episodes, is consistently and strongly associated with esophageal mucosal damage. From Ref. [15].

esophageal emptying, and prolonged acid and pepsin contact time.

The preponderance of evidence suggests the following conclusions: (1) the hiatal hernia, by permitting more frequent reflux episodes and greater acid contact time, is the major factor associated with esophagitis; 2) that in patients with symptomatic GER, the presence of a hiatal hernia is the single most important predictor of reflux esophagitis; and (3) that an incompetent LES is not a major etiologic factor in the development of esophagitis.

Based on these conclusions, we propose three separate and distinct etiologies of GERD, and we offer the following three hypotheses to explain why, after 1.5 million years of standing erect, we have evolved into a species (specifically Homosapiens sapiens) that is destined to live with the scourge of GERD.

THREE HYPOTHESES

Hypothesis 1: congenital

The antireflux barrier — comprising the smooth-muscled LES, the skeletal-muscled right crural diaphragm and the phrenoesophageal ligament — does not develop completely due either to a developmental
Figure 2. Influence of hiatal hernia on frequency and esophagitis. Demonstration of the powerful influence of hiatal hernia on frequency and esophagitis and the lack of influence of the LES pressure on esophagitis. The large difference in reflux frequency between two patients of similar age is apparent on the 24-hour ambulatory pH tracing. Although both patients have similar LES pressures (6 mm Hg), patient 1 has a much greater frequency. This is likely due to the presence of the hiatal hernia. The frequent drops below pH 4.0 coincide with hiatal-hernia-induced acid reflux into the esophagus. Patient 1: male, 65 years old, LES pressure 6 mm Hg, hiatal hernia present, esophagitis present, Barrett's not present. Patient 2: male, 62 years old, LES pressure 6 mm Hg, hiatal hernia not present, esophagitis not present, Barrett's not present. From Ref. [15].

Evidence: In 1975, Darling reported that 83 (29 percent) of a total of 285 infants and children had a hiatal hernia or GER [19]. In 1976, Astley et al. reported a long-term prospective followup of 113 children with vomiting due to a small hiatral hernia [20]. When reviewed by the same clinical and radiological observers twenty or more years later, more than half still had the hiatal hernia. Eighteen years later, in 1994, the same group followed up their report on 118 patients with a hiatal hernia in childhood [21]. Hiatral hernia continued to persist in more than half the

anomaly or a gestation period of less than 40 weeks.
patients, although few complained of significant symptoms. Of interest, two patients who requested endoscopic exams were found to have Barrett’s esophagus. In their original population, 27 patients had failure to thrive, 28 had persistent vomiting, 15 had recurrent pneumonias, 16 had esophagitis, nine had apnea due to laryngeal spasm and eight had GI bleeding. These cases were considered to be congenital GER and hiatal hernia. In 1991, Latif Al-Arfaj reported on 10 children with massive hiatal hernias in Saudi Arabia [22].

In 1958, Botha demonstrated unequivocally that hiatal hernia could be congenital and different than the hiatal hernia in adults. He removed the diaphragm, lower esophagus, upper stomach and surrounding tissues en bloc from 115 children, aged 32 weeks to eight years, most of whom were less than one month of age [23]. Detailed dissection of these specimens revealed important information: the anatomic difference between the sliding hiatal hernia of infancy and that of adults was that the former was present at birth as a result of a congenitally short esophagus or a congenitally malformed or underdeveloped diaphragm, whereas the latter is seen in the later decades of life as a result of degenerative changes of normal hiatal structures.

Speculation: Despite the detailed descriptions given in these studies and the long-term follow-up of up to 40 years, it is not certain whether these hiatal hernias were due to incomplete maturation due to developmental anomaly or incomplete gestation. It is certainly clear, however, that most of these hiatal hernias were congenital, as a majority of the children had symptoms of vomiting and failure to thrive from the time of birth. It is possible that the children were born with normal structures and then developed hiatal hernia from persistent vomiting within hours after birth. This possibility isn’t likely, however, since no cause for vomiting was reported in the studies, and in most of the children, the vomiting and the failure to thrive occurred within a short time of birth.

Summary: These studies clearly demonstrate that one of the causes of hiatal hernia in children is congenital and that in half these patients, the hiatal hernia persists into adult life.

Hypothesis 2: acute traumatic etiology

The antireflux barrier in adults suffering acute traumatic injury to the abdomen or chest is permanently disrupted by unexpected forces, such as motor vehicle accidents (with steering wheel crush injury), blows to the abdomen (from activities such as wrestling, boxing, or martial arts), heavy lifting or moving (e.g., pianos, refrigerators) or unphysiologic stress positions (e.g., gymnastic feats). The trauma creates a hiatal hernia that renders the antireflux mechanism useless and incapable of preventing GERD, as seen in the following five cases:

A 67-year-old steroid-dependent asthmatic has severe GER symptoms. At the age of 20 years, a grenade blast propelled him out of his foxhole in Iwo Jima, instantly killing his four comrades. He awoke two weeks later in a U.S. hospital with his stomach in his chest. Several unsuccessful surgeries were attempted to repair the huge rent in the diaphragm and to anchor the stomach back into the abdomen. Severe symptoms of both GER and asthma developed within weeks of the injury. The GER and asthma remained as serious problems until he died at the age of 74 years.

A 25-year-old muscular gymnast has continuous heartburn. At the age of 24 years, he developed sudden, crushing, retrosternal chest pain during a vertical handstand on the parallel bars. Myocardial infarction was ruled out at the local hospital. Within days of the injury, moderate to severe GER symptoms developed and self-treatment with antacid tablets began. Endoscopy revealed a 4 cm hiatal hernia, esophageal erosions and ulcerations.
A 62-year-old man has heartburn and episodic solid food dysphagia. At the age of 59 years, while sitting in the car waiting for the traffic light to turn green, he was hit from behind by a 16-wheel Mac truck. His car was hurled 100 feet over a fence and into a ditch. A steel saw was required to cut away the steering wheel, which was imbedded in his abdomen almost to the spine. Heartburn and dysphagia for solid foods developed immediately after hospitalization. Endoscopy six months after the accident revealed a 3 cm hiatal hernia with a very tight GE junction and a very tight diaphragmatic hiatus.

A 32-year-old man has heartburn and complete loss of dental enamel. At the age of 31 years, while driving home from work, he developed sudden, crushing retrosternal chest pain. Myocardial infarction was ruled out after three days in the C.C.U. Within days of the event, he began taking liquid antacids for heartburn and regurgitation. Within two months of the event, the enamel on his lower canines and incisors was completely eroded — likely due to reflux of gastric acid into the mouth.

A 54-year-old furniture mover has asthma and GER symptoms. At the age of 34 years, while moving a refrigerator up three flights of stairs, he developed retrosternal, crushing chest pain. He did not seek medical attention. Within three weeks of the event he developed symptoms of heartburn, regurgitation, dysphagia and wheezing. He treated himself with liquid antacids for several years before seeing a physician. Endoscopy revealed diffuse erosions, Barrett's esophagus and a 6 cm hiatal hernia with a wide GE junction and a widely patent diaphragmatic hiatus.

Speculation: We cannot prove from these five patients that GER with or without a hiatal hernia was not present before their acute traumatic event. According to their histories, none of the patients had reflux symptoms before the trauma, and

Figure 3. Result of acute traumatic hiatal hernia. Separation of the smooth muscle LES (intrinsic sphincter) and the skeletal muscle crural diaphragm (extrinsic LES) so that the two sphincters do not function together to prevent GER.
all had moderate to severe reflux symptoms beginning soon after the traumatic incident. The patient on Iwo Jima clearly had traumatic injury to the diaphragm and the chest from the grenade. The gymnast and the refrigerator mover both developed crushing chest pain during strenuous activity, and both required antacids within a short period of time after their chest pain. The patient who lost the enamel of his teeth due to reflux clearly had a rapid onset of acid reflux into the mouth, since his teeth were nearly perfect until he was hospitalized with chest pain. The patient who was hit by a Mac truck was a clear victim of a crush injury to the abdomen, which resulted in dysphagia. The literature is filled with case reports of patients who required reduction of hiatal hernias and closures of diaphragmatic tears due to acute injuries. How much contribution acute injury to the diaphragm makes is unknown, but it may be higher than realized since many patients do not recall the exact injury, especially if it did not require them to seek hospitalization. Figure 3 demonstrates an acute traumatic event that potentially might result in development of hiatal hernia.

Summary: We have presented five patients who clearly developed GER symptoms after an acute injury. Although we cannot say for certain that asymptomatic hiatal hernia and asymptomatic reflux were not present before their acute event, it is certain that the symptoms began after the acute event. We can only speculate that their hiatal hernia and reflux began with the acute injury.

Hypothesis 3: chronic traumatic etiology

The antireflux barrier in children and adults is gradually weakened over time as a result of years of straining to defecate and straining in an unphysiologic position, both of which stem from our modern day habits of eating a low-fiber diet and living on the high-seated toilet. Insufficient dietary fiber results in small caliber stools that can be expelled only by generating and maintaining very high abdominal pressures. The high-seated toilet promotes a physiologically unsound sitting position which, during straining at defecation, directs the abdominal forces upward through the esophageal hiatus. Thus, the repeated straining results in a weakening of the anti-reflux barrier, development of a hiatal hernia and a useless anti-reflux mechanism that is incapable of preventing GERD.

Evidence: In 1981, Rains and Ritchie postulated that the etiology of hiatal hernia included: (1) muscular degeneration with increasing age; (2) increased intra-abdominal pressure as a result of pregnancy, obesity or large ovarian cysts; and (3) an increase of fatty tissue in the hiatus with decreased elasticity of the crus, as a result of the obesity [24]. Such factors at best could only partially explain the contrasting situations found in Africa, Asia and the Westernized countries. Six years earlier, in 1975, Denis Burkitt put together all the information known about the effect of diet on the physiology of the gastrointestinal tract, including the influence of fiber on stool volume, water content and transient time. He postulated that abdominal straining during efforts to evacuate hard feces would increase intra-abdominal pressure to a point that would force the gastroesophageal junction upward into the thoracic cavity and produce or exaggerate the herniation through the diaphragmatic hiatus [25]. Burkitt’s theory was substantiated by the work of Light and Rutledge who, in 1965, measured the pressures in the sigmoid colon [26]. The values reflected the intra-abdominal pressures, which were highest during the Valsalva manoeuvre. In 1979, four years after Burkitt proposed his theory, Fedail et al. measured the intra-abdominal and intra-thoracic pressures and the gradient between them during defecation [27]. They also compared pressures in the sitting position with those in the squatting position. The results of their study demonstrated that the intra-abdominal pressures when straining maximally to defecate always exceeded the intra-thoracic pressures. The investigators
Figure 4. Toilet vs. squatting. Squatting may decrease the gradient across the abdominal-thoraco junction and potentially prevent the forceful thrust of the stomach up through the diaphragmatic hiatus.

Suggested that if this pressure gradient across the diaphragm occurred often and for prolonged periods the stomach might gradually be pushed up into the chest. Although the difference was not statistically significant, the authors found that squatting during defecation was more protective than sitting on a raised toilet seat, because the pressure gradient across the diaphragm was less during squatting. It is well known that Western populations sit on high toilets and strain to move out hard feces, whereas populations in developing countries eagerly squat and pass out large, soft, bulky stools. Figure 4 demonstrates the differences between squatting and sitting on high toilet seats.

Speculation: It is virtually impossible to prove that years of straining to defecate on high toilets is the cause of hiatal hernia. However, because continuous straining increases intra-abdominal pressures sufficiently to propel the stomach up into the diaphragmatic hiatus, it is not unreasonable to suggest that constant straining may result in a partial thoracic stomach otherwise known as hiatal hernia. Indeed, if straining to defecate does weaken the antireflux mechanism over time, then the incidence of hiatal hernia should increase with age — and it does! Figure 5 demonstrates the increasing incidence of hiatal hernia with increasing age.

Summary: Westernized countries have a low-fiber diet that results in hard feces and difficult defecation. Developing countries have a high-fiber diet, which results in large soft feces and ease of defecation. Western countries rarely assume the squatting position to defecate, whereas developing countries squat to defecate as a way of life. These factors are extremely important in the development of certain diseases, including hiatal hernia.

THE NEED TO KNOW

The long-ranging debate between the role of hiatal hernia and that of the LES in the development of and contribution to GERD has been termed "a tired argument." Although the argument may be tired, the need to know the mechanisms behind GERD should not be underestimated. If the development of a hiatal hernia is truly the major cause of GERD in Western civilization, then the causes of hiatal hernia should be sought and if possible prevented. From our hypotheses, the congenital hiatal
hernia and acute traumatic hiatal hernia would be difficult to prevent, unless all accidents and acute traumas could be predicted and prevented. The chronic traumatic etiology of hiatal hernia may well be responsible for up to 90 percent of reflux in Western civilization. Our hypothesis, if true, would require a change in lifestyle in order to prevent the development of hiatal hernia. This change in lifestyle may be of greater benefit than just prevention of hiatal hernia and GERD. It may also reduce the incidence of other diseases associated with low-fiber diets such as coronary artery disease, gall stones, diverticular disease, hemorrhoids, aortic aneurysms and venous thromboses. We believe that the development of hiatal hernia is the major factor in the production of GERD and that rational recommendations can be made to prevent its development. An additional benefit from such a change might be a decrease in incidence of inguinal hernias as well.

CONCLUSION

We have presented a brief history of abdominal symptoms throughout the ages and suggested that these symptoms could be found with dyspepsia, gastric ulcer, duodenal ulcer, as well as GERD. We then discussed the origins of peptic esophagitis and the development of GERD in the early part of the twentieth century. We followed the path of its origins through the hiatal hernia to the LES and then back to the hiatal hernia. The overwhelming evidence suggests that the presence of a hiatal hernia is almost mandatory for severe esophagitis to be present and that half the patients with hiatal hernia have reflux esophagitis. The importance of hiatal her-

Figure 5. Radiological prevalence of hiatal hernia. The prevalence of hiatal hernia appears to increase for small as well as for medium to large hiatal hernias, but is especially evident for small hernias. Adapted from Ref. [25].
nia is probably best exemplified by noting the following: in the absence of a hiatal hernia, there is almost never severe esophagitis. Finally, we presented three hypotheses that might explain the origins of GERD through the mechanism of hiatal hernia, which could result from any of three etiologies: (1) congenital, (2) acute traumatic and (3) chronic traumatic. We suggested that the congenital etiology is either a developmental anomaly or an incomplete gestation; that the acute traumatic etiology is a sudden acute trauma to the abdomen or chest; and that the chronic traumatic etiology (a) is the cause of more than 90 percent of the GERD that stalks the Western world; (b) is a direct result of abandoning the popular and worldwide practice of squatting to socialize, eat and defecate; and (c) is our just reward for adopting the “civilized” high sitting position on chairs and modern toilets.

We recommend that Western civilization seriously consider returning to some of the practices handed down to us by our ancestors.

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