

Supragastric belching: Pathogenesis, diagnostic issues and treatment

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Abstract Belching is defined as an audible escape of air from the esophagus or the stomach into the pharynx. It becomes pathologic if it is excessive and becomes bothersome. According to Rome IV diagnostic criteria, there is a belching disorder when one experiences bothersome belching (severe enough to impact on usual activities) more than 3 days a week. Esophageal impedance can differentiate between gastric and supragastric belching. The aim of this review was to provide data on pathogenesis and diagnosis of supragastric belching and study its relationship with gastroesophageal reflux disease and psychological factors. Treatment options for supragastric belching are also presented.

Keywords: Esophageal impedance, esophageal manometry, gastroesophageal reflux disease, ineffective esophageal motility, speech therapy, supragastric belching

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INTRODUCTION

Belching is defined as an audible escape of air from the esophagus or the stomach into the pharynx.^[1-3] Belching is a result of the vagally mediated reflex that relaxes the lower esophageal sphincter (LES) and is activated when the accumulation of air in the stomach increases the gastric volume.^[2-4] At the end of the process, gastric ventilation occurs.

Depending on the anatomical site where the refluxed gas has its origin, esophagus or stomach, belches can be supragastric or gastric. Belching becomes pathologic if

it is excessive and becomes bothersome. Based on Rome IV diagnostic criteria, belching disorders are classified as excessive supragastric belching (SGB) and excessive gastric belching. The following criteria must be fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis: bothersome belching (severe enough to impact on usual activities) from the esophagus or stomach, more than 3 days a week.^[1-3] The observation of frequent, repetitive belching is a supportive criterion for excessive SGB. Nevertheless, intraluminal impedance measurement is required to distinguish supragastric from gastric belching.^[5,6] Ructus and eructation are synonyms for belching.^[2-5]

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Although excessive SGB can be an isolated symptom, in some patients, it is associated with gastroesophageal reflux disease (GERD), functional dyspepsia (FD), bulimia nervosa, obsessive-compulsive disorder, or anxiety disorder.^[4-8] In patients with upper gastrointestinal complaints referred to tertiary care units, SGB was present in 3.4% of patients.^[9] The long-tenured view was that excessive belching was a direct consequence of aerophagia (excessive swallowing of air into the stomach). However, according to Rome IV criteria, aerophagia and excessive SGB are two distinct disorders with different pathogenetic mechanisms. Aerophagia occurs less often than SGB. It is caused by the swallowing of a large quantity of air leading to abdominal distension, bloating, and excessive gastric belching.^[1-3]

Socio-psychological factors have an essential role in the pathogenesis of the disease. Belching is absent during sleep and significantly reduced when patients with excessive SGB are unaware that they are observed or when they are distracted.^[6-8] SGB is a learned and self-induced behavioral phenomenon with no organic substrate. Psychological factors play an important role in symptom frequency and severity. Esophageal impedance is the gold standard for diagnosis. The aim of this review was to provide data on the pathogenesis and diagnosis of supragastric belching and study its relationship with gastroesophageal reflux disease and psychological factors.

PATHOPHYSIOLOGY AND DIAGNOSIS OF BELCHING

The current knowledge we have on belching is based on observations from studies using esophageal impedance monitoring. In addition, esophageal manometry helped to identify the events happening just before or during SGB. Therefore, this part of the review is based on impedance and manometric changes observed in belching. Esophageal impedance monitoring allows the assessment of the intraluminal movement of gas or liquid, the direction of flow, and the nature of esophageal content. Therefore, aerophagia [Figure 1a], SGB [Figure 1b], and gastric belches [Figure 1c] can be differentiated, and the origin of bothersome complaints can be established.^[6] The main components of the SGB reflex are sensory reception, neural pathways, central nervous system integration, and neurochemical mediation.^[4,6,10]

One of the first studies on belching using esophageal impedance was the study of Bredenoord *et al.*^[11] In gastric belches, air originates from the proximal stomach and is swallowed while eating or drinking. Gastric belches prevent

the accumulation of important amounts of air in the stomach. The swallowed air, distends the proximal stomach, which induces transient relaxations of the LES (TLESR). TLESRs allow the passage of air in the esophagus with distention of the esophageal body, which induces upper esophageal sphincter (UES) relaxation, and air escapes in the pharynx.^[11] In impedance, this oral movement of gas is seen as an increase in impedance starting in the distal channel (close to LES because the air comes from the stomach), followed by an increase in the impedance in more proximal channels; therefore, a retrograde rise in impedance corresponding to the retrograde movement of air is observed [Figure 1c]. In SGB, the air is “swallowed” (not literally, but it comes from the pharynx) and does not reach the stomach. There is a rapid antegrade flow of air, followed by a retrograde movement. The impedance catheter records an increased impedance in the proximal channel, moving toward LES (the swallowed air goes down), followed by a rapid retrograde return of impedance to baseline (the drop in impedance begins distally and progresses proximally, being determined by the air moving towards the mouth to be expelled)^[11] [Figure 1b].

Esophageal manometry performed at the same time with esophageal impedance showed both pharyngeal contractions and decreases in esophageal body pressure immediately before the increase in electrical impedance. Based on these observations, the authors suggested two possible mechanisms for SGB: 1. a pharyngeal contraction pushing the air in the esophagus and 2. the negative esophageal pressure gradient, sucking the air in. In addition, an increase of abdominal pressure was observed, attributed to abdominal strain, probably the mechanism used to evacuate the esophageal air.^[11] The pharyngeal contraction described using conventional manometry (seen as a pressure rise) proved to be a pharyngeal intrabolar pressure in high-resolution esophageal manometry and not a peristaltic contraction.^[6] The authors hypothesized that the retraction of the tongue base pushes air in the pharynx, and through opened UES into the esophagus, like a piston, while the nasopharynx is closed.^[6]

The sequence of events leading to SGB was further completed and confirmed by the study of Kessing *et al.*^[12] The first phenomenon that takes place in SGB is contraction and movement of the diaphragm toward the abdominal cavity, creating a negative intraesophageal pressure, followed by the opening of the UES, which allows the air to be sucked into the esophagus. The entering of air in the esophagus is confirmed by an antegrade rise in esophageal impedance. LES pressure remains unchanged or even increases.^[11,12] In comparison, most gastric belches

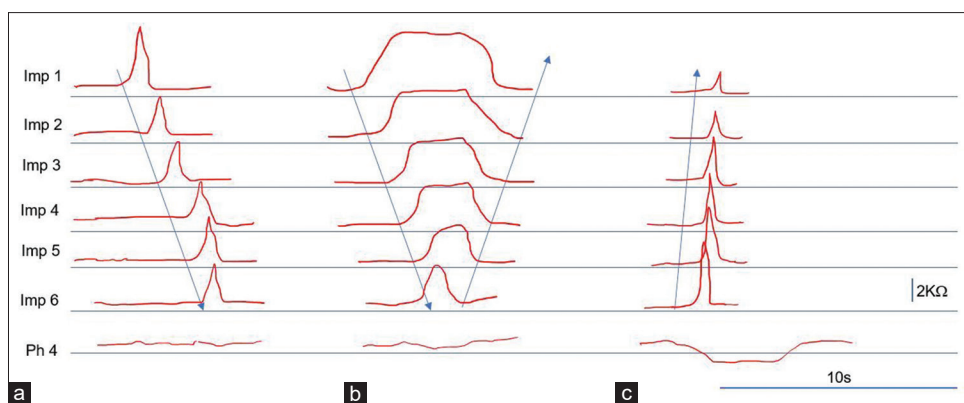


Figure 1: a. Air swallowing results in an increase in impedance starting in proximal impedance channels Imp 1 and going down to the stomach; b. Supragastric belch – swallowed air (air going down), returns rapidly towards the mouth, from the distal esophagus (Imp 6) to proximal esophagus (Imp 1); c. gastric belch – the air originates from the stomach, the increase in impedance is observed first distally (channel Imp 6), and afterwards, air moves rapidly to the proximal esophagus (Imp 1)

are associated with TLESR episodes or a decrease of LES pressure that allow gastric air to be vented from the stomach. Another important difference between SGB and gastric belches refers to the timing of the UES opening. The UES opens early in SGB to allow passage of air in the esophagus, while it opens late in gastric belches, after the onset of the retrograde airflow, to allow air to pass from the esophagus to the pharynx.^[12]

One of the functions of the UES is to prevent esophageal air insufflation during periods of negative intrathoracic pressure, like during inspiration. The factors determining the opening of the UES in SGB are not yet elucidated. During TLESR, UES is opened, allowing the air suction type of SGB during the reflux period.^[13] Esophageal distention determined by air insufflation opens the UES.^[14] However, as mentioned above, in patients with SGB, there is a decrease in esophageal pressure preceding the air sucking. Swallowing also opens the UES, but this aspect was not observed in manometry before SGB.^[14]

Currently, impedance monitoring is the diagnostic tool for excessive SGB. Further, 24-h ambulatory pH-impedance monitoring is used to differentiate SGB from gastric belches and GERD, even if SGB and GERD seem to coexist. Gastric belches determine an increase in impedance starting in the distal channel and progressing to the mouth. SGB is recognized by an increase in impedance, starting in the most proximal channel and rapidly progressing to the distal channel, followed by a retrograde drop in impedance (starting distally and progressing proximally) as air is eliminated.^[6] Based on manometry and impedance studies, two methods used by patients with SGB to bring air into the esophagus were identified: the air-suction method and the air-injection method.^[6] SGBs can be present in healthy subjects. Koukias *et al.*^[9] reported that in

healthy subjects (n = 40), the number of SGB ranges from none to 15/24 h (median value = 0, mean value = 3), the upper limit of normal (95th percentile) being considered 13/24 h. A few years earlier, another group reported fewer SGBs/24 h (median of 2 SGBs per 24 h), but only 10 healthy subjects were evaluated.^[15,16]

PATHOGENESIS OF SUPRAGASTRIC BELCHING

As the diaphragm can be controlled voluntarily, one hypothesis regarding the development of SGB is that in the beginning, belching is self-induced to relieve symptoms such as fullness from the upper part of the abdomen. Afterwards, this action becomes a learned behavior, and the patient is no longer aware that he or she can control the belches.^[12] SGB is almost absent during sleep, supporting the idea that it is a voluntary behavior.^[15] Psychological factors are involved in excessive SGB development, influence its severity, and psychological therapies can be used in treating this disorder. The presumed factors that are involved in the pathogenesis and outcome of SGB are presented in Figure 2.

Psychological factors and SGB

Supragastric belching is a disabling behavioral disorder that can significantly impact a patient's quality of life (QOL) and interfere with daily functioning, including work and school absenteeism, difficulty with household chores, and decreased social and leisure activities.^[8,17-19] Although the association of psychiatric disorders and functional gastrointestinal disorders (FGIDs) is well established, the association between SGB and psychiatric comorbidities has not been adequately studied.^[20] SGB has been described in patients with psychiatric troubles such as obsessive-compulsive disorder, depression, anxiety disorders, and eating disorders (bulimia nervosa and anorexia nervosa).^[21-25]

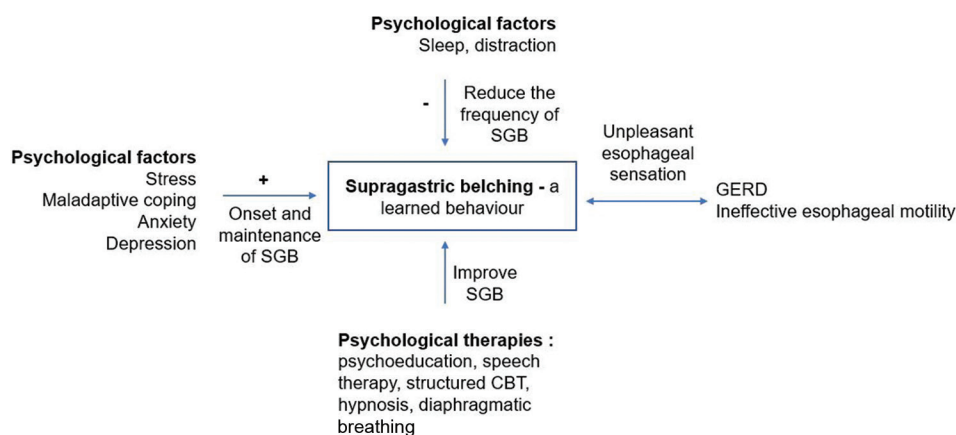


Figure 2: The factors involved in the pathogenesis and outcome of supragastric belching (SGB). GERD, gastroesophageal reflux disease; CBT, cognitive behavioral therapy

The role of psychological factors in SGB is proven by several observations.^[26,27] First, the frequency of belching worsens under stress. Second, there are no SGBs during sleep. It was reported that distracting the patients resulted in a decrease in SGBs frequency. Similarly, when patients were unaware they were studied, fewer SGBs occurred.^[12,15,26,28] These observations support the idea that SGB is a self-induced behavioral phenomenon.

Known psychological vulnerabilities are related to the onset and exacerbation of gastrointestinal disorders. Early-life adversity, psychopathology, and maladaptive coping have been involved in FGDIs and motility disorders.^[29] Psychosocial factors, such as stress, learned fear of symptoms, and maladaptive coping (e.g., food avoidance), can contribute to the onset and maintenance of excessive belching. As symptoms increase, associated psychosocial stress and reduced coping ability may lead to anxiety and depression symptoms. Maladaptive coping (failure to accept or adapt to symptoms and limitations imposed by them), inability to manage unpleasant adverse effects, or cope with uncomfortable symptoms has been linked with a decrease in QOL.^[30]

The relationship between anxiety, depression, and SGB has been evaluated in the current literature with inconsistent results. Sun *et al.*^[8] conducted a study involving 20 patients that met the diagnostic criteria of belch, reporting no significant relationship between belch severity and anxiety or depression status. Furthermore, Kessing *et al.* observed no significant differences related to anxiety levels between patients with none to mild, moderate, and severe belching symptoms.^[31] Bredenoord *et al.* found no elevation in anxiety and depression levels in patients with SGB.^[19] On the contrary, Punkkinen *et al.*^[32] conducted a randomized trial evaluating the effects of behavioral therapy on SGB.

At baseline, the included participants were assessed for anxiety and depression status. Almost half of the patients reported a Beck anxiety inventory score of ≥ 8 , indicating anxiety, and 30% reported a Beck depression inventory score of ≥ 13 , indicating depression, much more compared with the prevalence observed in the general Finnish population.^[32]

Early recognition and appropriate classification are of cardinal importance for guiding diagnostic reasoning and informing therapeutic decisions. Effective treatment of patients with SGB requires an understanding of the psychosocial background against which symptoms occur. A comprehensive assessment of whether other circumstances such as pain, anger, depressed mood, anxiety can elicit or precipitate the belching is recommended. Psychological factors are linked with the symptoms and prognosis.^[33]

Gastroesophageal reflux disease and SGB

Belches, including SGBs, are often observed in patients with GERD, making researchers ask themselves if SGB was the cause or the consequence of reflux episodes.^[9] Gastric belches are related with meals being often observed postprandially. In contrast, SGBs seem not to be related with meals.^[11]

Koukias *et al.*^[9] analyzed 2950 reports during a period of 4 years. The aim of the study was to establish the prevalence of excessive SGB in a gastrointestinal physiology unit and to analyze the relationship between GERD, esophageal motility, and excessive SGB. One hundred patients (3.4%) with excessive SGB were identified, and 95% of these patients had typical reflux symptoms, while 65% reported dysphagia. Pathological esophageal acid exposure was observed in 41% of patients with SGB. SGBs preceding

reflux episodes were responsible for 27% of the total acid exposure time, supporting the hypothesis according to which SGB plays a role in the pathogenesis of GERD. Esophageal hypomotility was found in 44% of patients. Patients with ineffective motility had more SGBs compared to patients with normal motility (118.3 ± 106.1 vs 80.6 ± 75.7 , $P = 0.020$). Motility changes were frequent failed peristalsis, weak peristalsis with large defects, and weak peristalsis with small defects.^[9] GERD can induce hypomotility of the esophageal body; therefore, it may be a confounding factor. However, patients with normal acid exposure and SGB also had esophageal hypomotility. Excessive SGB was rarely independent from other upper gastrointestinal symptoms (5 of 100 patients). Nevertheless, 14% of patients with excessive SGB did not consider that they were suffering from excessive SGB and did not consider it a health issue, making the prevalence of SGB difficult to establish.^[9] The study did not analyze the effect of parameters involved in the pathogenesis of SGB, such as diaphragmatic movement, hiatus hernia, reflux volume, and psychological factors. The study could not distinguish between cause and effect in the SGB/esophageal hypomotility association, nor in the SGB/GERD association.

Another study^[16] analyzed the relationship between excessive SGB and GERD in 50 patients with reflux symptoms and 10 healthy subjects. All the subjects underwent ambulatory 24-h pH-impedance. SGBs were identified in half of GERD patients and half of healthy subjects, but the median number of belches/24 h was higher in GERD (13 vs. 2). In connection with the pathogenesis of SGB, two main physiopathological patterns were observed: 1/3 of SGBs occurred just before the onset of reflux episode (in less than 1 s); 18% of SGBs occurred during the reflux episode, with the onset being 4–10 s after the start of the reflux episode. This study showed that SGB episodes occurred more frequently in GERD patients, and half of SGB were in close temporal relation to refluxes. In some cases, SGBs elicit reflux episodes, and in other cases, SGBs might be a response to an unpleasant esophageal sensation induced by reflux.^[16]

Kessing *et al.*^[31] analyzed 90 patients with troublesome belching, who performed ambulatory 24-h pH-impedance for reflux symptoms. The severity of belching was classified using a 3-point scale, the belches perceived during the monitoring day were recorded in a diary, and SGB and gastric belches were analyzed. Patients with severe complaints had more SGBs that coincided with reflux episodes compared to patients with moderate and none to mild complaints. Both SGBs and gastric belches were more likely perceived by the patient if they coincided with a

liquid reflux. In a subgroup of patients, one-third of liquid refluxes were preceded by SGB and thus may have caused the refluxes. The authors concluded that the burden of belching in patients with GERD complaining of severe belching was caused by excessive SGBs.^[31]

A recent study by Jeong *et al.*^[13] compared belching and their relationship with reflux events in patients with GERD and >5 belches/day ($n = 10$) and in patients with belching disorder (BD, $n = 10$). Both groups of patients exhibited GBs and SGBs, and interestingly, the SGB/GB ratio was not different between the two groups. The most common type of belching was SGB in both groups. SGBs were related to reflux episodes in GERD patients but not in BD patients. Most of the SGBs in patients with BD were non-acidic.^[13]

Therefore, the relation between SGB and GERD seems to be bidirectional. Forty percent of patients with excessive SGB have pathological esophageal acid exposure,^[9] while 1/3rd of refluxes seem to be induced by SGBs. One hypothesis is that SGBs are a response to an unpleasant esophageal sensation.^[16] This hypothesis is supported by the observation that SGBs occur more often in patients with ineffective esophageal motility.^[9] Ineffective esophageal motility is associated with a delayed esophageal clearance,^[4,6,12] which can be the substrate of the unpleasant esophageal sensation mentioned above.

TREATMENT OF SUPRAGASTRIC BELCHING

As mentioned above, SGB is a learned behavior. Therefore, psychological therapies have a major role in SGB treatment, and more importantly, they are effective, underling once more that psychological factors play an important role in SGB pathogenesis and outcome. Psychological therapies aim to identify the self-learned pattern of behavior in SGB. The first step in SGB treatment is offering reassurance and a careful explanation of its behavioral origin and a thorough explanation of the cause of belching. Patients are encouraged to believe that they can control their symptoms, and belching is used as a hint to remind them to keep the mouth open to avoid swallowing more air.^[33]

One technique is speech therapy, focusing on unlearning the behavior with a speech pathologist. The sessions consist of explanations, creating awareness of esophageal air influx, and exercises (glottis training, breathing, and vocal exercises) to discontinue the SGB mechanism. In an open-label study of 11 patients,^[34] after ten 1-hour sessions of speech therapy, frequency and intensity of SGBs decreased in most patients. Another more recent and retrospective study

analyzed treatment outcomes in 48 patients with SGB and reported improvement of SGBs in 83% of patients after 10 sessions of speech therapy.^[35] These studies measured the effect of therapy by using visual analogue scale (VAS) scores. One small study used an office-based procedure to treat excessive belching. In one session, the patients were thought to breathe slowly and diaphragmatically with the mouth open to prevent belching. Four of the five patients had no complaints one month later.^[36]

Several studies reported that structured cognitive-behavioral therapy (CBT) was effective on patients' symptoms. CBT sessions were focused on recognizing early warning signals and preventative exercises; a decrease in healthcare utilization and improvement in the overall QOL was noted, and the favorable effects lasted up to 12 months.^[37,38] One recent study including 39 patients that completed five CBT sessions reported that the number of SGBs decreased significantly in parallel with improved social and daily activities.^[38] These patients were followed for the next 12 months, and the authors reported that the positive effects of CBT persisted at 6- and 12-months follow-ups. The factors associated with a better outcome after CBT were a lower number of SGBs, a lower hypervigilance score, and a higher proficiency score. The proficiency score assessed the patient's understanding and implementation of the CBT exercises, starting with the acceptance that SGB is a behavioral phenomenon, continuing with the ability to identify warning signals, and then to adhere to exercises.^[39] All these studies were rather small. Nevertheless, the available data and experts' opinion^[40] suggest that psychological therapies are effective and represent the main treatment option in SGB. However, the major drawback in clinical practice is the availability of experts trained in speech therapy or CBT.

Diaphragmatic breathing exercises represent another form of behavioral therapy that can reduce belching and restore the gastroesophageal pressure gradient.^[32,41,42] In one study, patients with PPI-refractory GERD (36 patients) and belching were either included in the diaphragmatic breathing exercises group (15 patients) or remained on a waitlist (the control group).^[42] Sixty percent of patients in the treatment group and none in the control group achieved a reduction by 50% in belching VAS. In addition, QOL improved in the treatment group. The favorable results were maintained at 4-month follow-up.^[42] Similarly, a more recent study on 42 patients with SGB reported that after five sessions of behavioral therapy (diaphragmatic breathing exercises), the frequency and severity of SGB at 6-month follow-up decreased in the treatment group compared with the control group (follow-up without

intervention). After 6 months, patients from the control group were offered behavioral therapy. Of the 36 patients evaluated after 12 months, an improvement of SGB was observed in 75% of patients. Depression scores also decreased, and mental well-being improved. The effects on anxiety scores and on health-related QOL were modest.^[32]

Pharmacological treatment for SGB is limited to the use of baclofen, an agonist of the γ -aminobutyric acid B receptor which inhibits TLESRs. An open-label study reported that baclofen improved SGB by reducing all flow events. The reduction was correlated with the increase in LES sphincter pressure and the decrease of swallowing rate.^[43] These data were not confirmed by a randomized double-blind, placebo-controlled, crossover study that included 20 patients with rumination syndrome and SGB.^[44] After fundoplication for reflux surgery, the number of GBs and SGBs decreased.^[45]

CONCLUSION

SGB is a self-induced learned behavior. The "swallowed" air is rapidly eliminated without reaching the stomach. Ph-esophageal intraluminal impedance is the gold standard in differentiating between gastric and SGBs. Psychological factors play a major role in SGB development, and psychological therapies are efficient in treating this disorder. Patients with excessive belches are likely to suffer from SGB. The relation between SGB and GERD seems to be bidirectional. SGBs induce some of the reflux episodes, while patients with SGB were found to have pathological reflux and gastro-esophageal reflux symptoms.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Drossman DA. Functional gastrointestinal disorders: History, pathophysiology, clinical features, and Rome IV. *Gastroenterology* 2016;150:1262–79.e2.
2. Drossman DA. Functional gastrointestinal disorders: What's new for Rome IV? *Lancet Gastroenterol Hepatol* 2016;1:6–8.
3. Drossman DA. Rome IV Diagnostic Algorithms for Common GI Symptoms. 2nd ed. Raleigh, NC: Rome Foundation, Inc; 2017.
4. Bredenoord AJ, Smout AJ. Physiologic and pathologic belching. *Clin Gastroenterol Hepatol* 2007;5:772–5.
5. Salazar Quero JC, Moya Jiménez MJ, Rubio Murillo M, Roldán Pérez S, Rodríguez Martínez A, Valverde Fernández J. Supragastric belches. An entity to know. *Gastroenterol Hepatol* 2017;40:396–7.
6. Kessing BF, Bredenoord AJ, Smout AJ. The pathophysiology, diagnosis and treatment of excessive belching symptoms. *Am J Gastroenterol* 2014;109:1196–204.

7. Halland M, Katzka DA. Editorial: The problem of supragastric belching needs to be heard. *Aliment Pharmacol Ther* 2019;50:832-3.
8. Sun X, Ke M, Wang Z. Clinical features and pathophysiology of belching disorders. *Int J Clin Exp Med* 2015;8:21906-14.
9. Koukias N, Woodland P, Yazaki E, Sifrim D. Supragastric belching: Prevalence and association with gastroesophageal reflux disease and esophageal hypomotility. *Neurogastroenterol Motil* 2015;21:398-403.
10. Lang IM. The physiology of eructation. *Dysphagia* 2016;31:121-33.
11. Bredenoord AJ, Weusten BL, Sifrim D, Timmer R, Smout AJ. Aerophagia, gastric, and supragastric belching: A study using intraluminal electrical impedance monitoring. *Gut* 2004;53:1561-5.
12. Kessing BF, Bredenoord AJ, Smout AJ. Mechanisms of gastric and supragastric belching: A study using concurrent high-resolution manometry and impedance monitoring. *Neurogastroenterol Motil* 2012;24:e573-9. doi: 10.1111/nmo. 12024.
13. Jeong SO, Lee JS, Lee TH, Hong SJ, Cho YK, Park J, et al. Characteristics of symptomatic belching in patients with belching disorder and patients who exhibit gastroesophageal reflux disease with belching. *J Neurogastroenterol Motil* 2021;27:231-9.
14. Shaker R, Ren J, Kern M, Dodds WJ, Hogan WJ, Li Q. Mechanisms of airway protection and upper esophageal sphincter opening during belching. *Am J Physiol* 1992;262:G621-8.
15. Karamanolis G, Triantafyllou K, Tsiomoulos Z, Polymeros D, Kalli T, Misailidis N, et al. Effect of sleep on excessive belching: A 24-hour impedance-pH study. *J Clin Gastroenterol* 2010;44:332-4.
16. Hemmink GJ, Bredenoord AJ, Weusten BL, Timmer R, Smout AJ. Supragastric belching in patients with reflux symptoms. *Am J Gastroenterol* 2009;104:1992-7.
17. Disney B, Trudgill N. Managing a patient with excessive belching. *Frontline Gastroenterol* 2014;5:79-83.
18. Tack J, Talley NJ, Camilleri M, Holtmann G, Hu P, Malagelada JR, et al. Functional gastroduodenal disorders. *Gastroenterology* 2006;130:1466-79.
19. Bredenoord AJ, Smout AJ. Impaired health-related quality of life in patients with excessive supragastric belching. *Eur J Gastroenterol Hepatol* 2010;22:1420-3.
20. Fadgyas Stanculete M, Dumitrascu DL. Psychiatric comorbidities in IBS patients. *J Psychosom Res* 2016;100:81.
21. Zella SJ, Geenens DL, Horst JN. Repetitive eructation as a manifestation of obsessive-compulsive disorder. *Psychosomatics* 1998;39:299-301.
22. Jones WR, Morgan JF. Eructophilia in bulimia nervosa: A clinical feature. *Int J Eat Disord* 2012;45:298-301.
23. Hadley S, Walsh B. Gastrointestinal disturbances in anorexia nervosa and bulimia nervosa. *Curr Drug Targets CNS Neurol Disord* 2003;2:1-9.
24. Appleby BS, Rosenberg PB. Aerophagia as the initial presenting symptom of a depressed patient. *Prim Care Companion J Clin Psychiatry* 2006;8:245-6.
25. Kim HY, Bang BW, Kim CE. A psychiatric approach to a patient with excessive belching. *J Korean Neuropsychiatr Assoc* 2014;53:327-31.
26. Bredenoord AJ, Weusten BL, Timmer R, Smout AJ. Psychological factors affect the frequency of belching in patients with aerophagia. *Am J Gastroenterol* 2006;101:2777-81.
27. Bredenoord AJ, Weusten BL, Timmer R, Smout AJ. Air swallowing, belching, and reflux in patients with gastroesophageal reflux disease. *Am J Gastroenterol* 2006;101:1721-6.
28. Rommel N, Tack J, Arts J, Caenepeel P, Bisschops R, Sifrim D. Rumination or belching-regurgitation? Differential diagnosis using oesophageal impedance-manometry. *Neurogastroenterol Motil* 2010;22:e97-104.
29. Fadgyas-Stanculete M, Dumitrascu DL, Pojoga C, Nedelcu L. Coping strategies and dysfunctional cognitions as predictors of irritable bowel syndrome diagnosis. *J Evid-Based Psychother* 2015;15:111-20.
30. Cheng C, Hui WM, Lam SK. Coping style of individuals with functional dyspepsia. *Psychosom Med* 1999;61:789-95.
31. Kessing BF, Bredenoord AJ, Velosa M, Smout AJ. Supragastric belches are the main determinants of troublesome belching symptoms in patients with gastro-oesophageal reflux disease. *Aliment Pharmacol Ther* 2012;35:1073-9.
32. Punkkinen J, Nyyssönen M, Walamies M, Roine R, Sintonen H, Koskenpato J, et al. Behavioral therapy is superior to follow-up without intervention in patients with supragastric belching-A randomized study. *Neurogastroenterol Motil* 2021:e14171. doi: 10.1111/nmo. 14171.
33. Riehl ME, Kinsinger S, Kahrilas PJ, Pandolfino JE, Keefer L. Role of a health psychologist in the management of functional esophageal complaints. *Dis Esophagus* 2015;28:428-36.
34. Hemmink GJ, Ten Cate L, Bredenoord AJ, Timmer R, Weusten BL, Smout AJ. Speech therapy in patients with excessive supragastric belching--a pilot study. *Neurogastroenterol Motil* 2010;22: 24-28, e2-e3. doi: 10.1111/j. 1365-2982.2009.01371.x.
35. Ten Cate L, Herregods TV, Dejonckere PH, Hemmink GJ, Smout AJ, Bredenoord AJ. Speech therapy as treatment for supragastric belching. *Dysphagia* 2018;33:707-15.
36. Katzka DA. Simple office-based behavioral approach to patients with chronic belching. *Dis Esophagus* 2013;26:570-3.
37. Bredenoord AJ. Management of belching, hiccups, and aerophagia. *Clin Gastroenterol Hepatol* 2013;11:6-12.
38. Glasinovic E, Wynter E, Arguero J, Ooi J, Nakagawa K, Yazaki E, et al. Treatment of supragastric belching with cognitive behavioral therapy improves quality of life and reduces acid gastroesophageal reflux. *Am J Gastroenterol* 2018;113:539-47.
39. Sawada A, Anastasi N, Green A, Glasinovic E, Wynter E, Albusoda A, Hajek P, Sifrim D. Management of supragastric belching with cognitive behavioural therapy: factors determining success and follow-up outcomes at 6-12 months post-therapy. *Aliment Pharmacol Ther* 2019;50(5):530-7.
40. Zad M, Bredenoord AJ. Chronic Burping and Belching. *Curr Treat Options Gastroenterol* 2020 doi: 10.1007/s11938-020-00276-0.
41. Halland M, Parthasarathy G, Bharucha AE, Katzka DA. Diaphragmatic breathing for rumination syndrome: Efficacy and mechanisms of action. *Neurogastroenterol Motil* 2016;28:384-91.
42. Ong AM, Chua LT, Khor CJ, Asokkumar R, S/O Namasivayam V, Wang YT. Diaphragmatic Breathing Reduces Belching and Proton Pump Inhibitor Refractory Gastroesophageal Reflux Symptoms. *Clin Gastroenterol Hepatol* 2018;16:407-16.e2.
43. Blondeau K, Boecxstaens V, Rommel N, Farré R, Depuyper S, Holvoet L, et al. Baclofen improves symptoms and reduces postprandial flow events in patients with rumination and supragastric belching. *Clin Gastroenterol Hepatol* 2012;10(4):379-84.
44. Pauwels A, Broers C, Van Houtte B, Rommel N, Vanuytsel T, Tack J. A Randomized Double-Blind, Placebo-Controlled, Cross-Over Study Using Baclofen in the Treatment of Rumination Syndrome. *Am J Gastroenterol* 2018;113:97-104.
45. Oor JE, Broeders JA, Roks DJ, Oors JM, Weusten BL, Bredenoord AJ, et al. Reflux and Belching after Laparoscopic 270 degree Posterior Versus 180 degree Anterior Partial Fundoplication. *J Gastrointest Surg* 2018;22:1852-60.