Aerophagia is a symptom related to excessive belching secondary to air swallowing during eating or drinking. Air swallowing per se is considered a normal physiological consequence of swallowing and is estimated that as much as 30 mL of air is ingested with most swallows. Distention of the stomach results in a transient relaxation of the lower esophageal sphincter (LES) allowing the ingested air to be vented into the esophagus. Further distention of the esophagus triggers a relaxation of the upper esophageal sphincter (UES) which then allows the air to escape in the form of a belch or burp. Although no formal data seem available with regard to incidence, aerophagia is a symptom noted in some patients treated with CPAP. In this issue of the journal Watson and Mystkowski have provided an interesting clinical observation regarding the occurrence of the symptom of aerophagia and its relationship to symptomatic gastroesophageal reflux disease (GERD) in patients treated with CPAP for obstructive sleep apnea. In a case control study they have shown that in patients treated with CPAP those with aerophagia had a significantly greater incidence of symptom related medications.

The physiological mechanisms of aerophagia have been described in a study which utilized esophageal impedance to measure the antegrade and retrograde flow of gas in the esophagus. It is helpful in understanding the mechanisms of aerophagia secondary to CPAP treatment to understand the pathophysiology described in this study. Two types of aerophagia were noted in this study. The first, and perhaps the most common under normal circumstances, is characterized by the venting of gas from the stomach into the esophagus. The authors refer to this as “gastric belching.” This term is important since “belching” and abdominal bloating are very common symptoms in patients with GERD. The second type is characterized by a rapid ingestion of air into the esophagus followed by a rapid expulsion of the ingested air. Since this air never reaches the stomach it is referred to as “supragastric belching.” In this study, gastric belching was noted in both normals and patients with aerophagia, while the supragastric belching was noted only in the aerophagia patients. The evacuation of the esophageal air is presumably via an abdominal strain which forces air in a retrograde direction. Watson and Mystkowski have proposed that CPAP results in air which is inadvertently injected into the stomach resulting in gastric distention and a reflex inhibition of the LES pressure creating a susceptibility to gastroesophageal reflux (GER). The authors speculate that this pathophysiology creates an environment conducive to the development of aerophagia. This speculation needs further discussion in the light of the above noted study on the mechanisms of aerophagia.

It is presumed that those with GERD have greater susceptibility to aerophagia due to a lower threshold of induction of transient relaxations of the LES secondary to gastric distention occurring via the CPAP treatment. Although very plausible on the surface, this speculation has several drawbacks. First, it makes the assumption that the aerophagia is of the “gastric” type noted above and there is no evidence that CPAP, no matter the pressure, results in substantial amounts of air in the stomach. In point of fact, if this is an issue of air in the stomach, it would seem that the symptom of aerophagia would be more prevalent in patients with higher CPAP pressures. The authors present no data that this is the case. More likely in fact, is that the air would expand the esophagus, but it seems obvious that this could not result in symptoms the subsequent day since there is no way the air could be retained in the esophagus. Furthermore, it is not clear how the relatively small amount of pharyngeal positive pressure noted in these data (approximately 10 cm H2O) could breach the upper esophageal sphincter whose pressure is commonly in the range of 100 mm Hg. Also, as noted above, belching and abdominal bloating are common complaints among GERD patients, and one could speculate that the “aerophagia” in the CPAP patients with GERD may simply be a manifestation of their GERD. This is quite plausible since the aerophagia patients were identified as anyone who had “…painful abdominal bloating, eructation, or flatulence related to CPAP treatment…”
No symptoms of nighttime heartburn were assessed in any of the patients. It is interesting to speculate that nighttime symptoms might be more prevalent in patients whose aerophagia is due to true gastric distention secondary to CPAP. According to the speculations by Watson and Mystkowski, these individuals would be expected to have more LES transient relaxations during sleep thus making them more likely to develop aerophagia secondary to CPAP treatment. It would be useful to know if the presence of nighttime heartburn would indeed be an accurate predictor of risk for the development of aerophagia in CPAP patients and if higher CPAP pressures would also create a risk of aerophagia.

The issue of treatment raises several interesting physiologic speculations. As the authors point out, it is well established that CPAP provides some improvement in heartburn symptoms in CPAP patients and CPAP itself has been shown to reduce nocturnal GER. Assuming some resolution of GERD symptoms with CPAP treatment, the fact that aerophagia develops or remains would suggest that the aerophagia is not a manifestation of GERD, but rather is an independent symptom manifestation of CPAP treatment. Unfortunately the authors do not present any data on the resolution of GERD symptoms with CPAP treatment. The authors speculate that aerophagia may be treated via gastric fundoplication which has the effect of increasing LES pressure. The authors state that this will “…mitigate aerophagia and allow ongoing CPAP treatment.” It is not clear why this treatment would work, since the effect of fundoplication on transient LES relaxations is unclear. Furthermore one of the most common complications of fundoplication is gas/bloat syndrome. A more reasonable treatment would a drug which has the effect of reducing transient LES relaxations such as baclofen.

The comments noted in this editorial are intended to augment physiologic speculation initiated by a particularly provocative and interesting clinical observation. The authors have provided us not only with the first real data on the symptom of aerophagia in CPAP treatment, but also some very interesting thoughts concerning mechanisms which deserve our attention and further speculation. One cannot ask more of an initial case controlled study of this type.

**DISCLOSURE STATEMENT**

Dr. Orr has indicated no financial conflicts of interest.

**REFERENCES**