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• *LETTERS TO THE EDITOR* •

Is the severity of obstructive sleep apnea or the magnitude of respiratory effort associated with gastroesophageal reflux?

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TO THE EDITOR

In a recent issue of *World Journal of Gastroenterology*, Demeter *et al*.[1], reported that in patients having both gastro-esophageal reflux disease (GERD) and obstructive sleep apnea (OSA), there was a positive correlation between endoscopic findings of GERD and the number of apneas and hypopneas per hour, namely apnea hypopnea index. They proposed that the severity of OSA and GERD are parallel to each other. The study of Demeter and colleagues is very important, not only for assessing reflux-induced esophageal damage in OSA, but also for determining therapeutic approaches for patients with OSA and GERD. Because both conditions are very common in general population and are associated with morbidity and deterioration of quality of life[2,3].

However, causative relationship underlying the association may be complicated. One proposed mechanism is that greater respiratory effort increases the pressure gradient across the lower esophageal sphincter and eventually facilitates the retrograde movement of gastric content[4]. The point is that classification of OSA is traditionally based on the apnea-hypopnea index (AHI) and, severity of OSA, which is defined by higher score of AHI, does not necessarily reflect the magnitude of respiratory effort during obstruction. Therefore, it is not easy to conclude that the occurrence of gastro-esophageal reflux is related to the number of apneas and hypopneas rather than respiratory effort during each breathing cessation period. Besides the magnitude of respiratory effort, repetitive stimulation of lower esophageal sphincter via phreno-esophageal ligament may also be linked with a threshold value of respiratory effort. Interestingly, there is another sleep-disordered breathing called "Upper Airway Resistance Syndrome", which is described by excessive daytime sleepiness, increased respiratory effort without oxyhemoglobin desaturation during sleep and absence of apneas or hypopneas. Inspiratory effort is an important characteristic and determinant of sleepiness and other symptomatology in UARS patients[5]. Considering the same mechanism, patients with UARS should also complain from reflux-related symptoms. We are not aware of any study reporting GERD in UARS. Another mechanism involves increased arousals in patients with OSA, together with reduced sleep efficiency, which may trigger transient lower esophageal sphincter relaxation and thus, promote acid reflux[6]. We have recently reported that there is no sufficient evidence to accept arousals and obstructive respiratory events during sleep as primary causes of gastroesophageal reflux and vice versa[7]. Indeed, we failed to find any timely relation between reflux episodes and respiratory events on the simultaneous recordings of dual probe esophageal pH monitoring and polysomnography. We think that controversy will remain until placebo-controlled studies focusing on the timely relation between respiratory effort and reflux episodes will be performed.

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