Swallowing in obstructive sleep apnea syndrome

A deglutição na síndrome da apneia obstrutiva do sono

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ABSTRACT
Obstructive sleep apnea syndrome and primary snoring are associated with the presence of neurogenic lesions and impaired sensory function in the upper airway, which are presumably caused by low-frequency vibrations produced by snoring or intermittent hypoxia. The clinical impact of this peripheral neuropathy on the pharynx has not been thoroughly investigated with respect to the management of patients with obstructive sleep apnea syndrome. Several authors have shown changes in swallowing associated with this syndrome, such as early bolus escape, the presence of pharyngeal residue, laryngeal penetration, and increased latency before triggering of the swallowing reflex. In this article, we review the main features of swallowing that may be altered in obstructive sleep apnea syndrome and the mechanisms involved in its pathophysiology as well as the results of studies that have evaluated swallowing in patients after treatment for this syndrome.

Keywords: deglutition; deglutition disorders; sleep apnea, obstructive; snoring.

INTRODUCTION
Obstructive sleep apnea syndrome (OSAS) is characterized by repeated episodes of partial or complete obstruction of the airway during sleep, resulting from narrowing of the pharynx and a decrease in the tone of the pharyngeal dilator muscles. OSAS may be preceded by an early stage of primary snoring. Neurogenic lesions in the oropharynx and the soft palate are associated with OSAS and primary snoring, but their cause is unknown. Some authors believe that the lesions are triggered by low-frequency vibrations produced by snoring or intermittent hypoxia.

Because the pharynx is the site of the lesions, many authors have suggested that there may be a swallowing dysfunction associated with OSAS. The onset of the swallowing reflex and the propagation of the food bolus are dependent on adequate pharyngeal sensitivity and function. Moreover, continuous OSAS may affect efferent neuromuscular activity and the upper airway function control centers.

The aim of this paper is to review the evidence in the literature regarding swallowing dysfunction in primary snoring and in OSAS.

NEUROGENIC LESIONS IN OSAS
Neurogenic lesions are found in the oropharynx of individuals who snore, and these lesions are thought to be caused by low-frequency vibrations produced by stertor. This assertion is supported by several histological studies, such as that of Friberg et al., in which mucosal biopsies of the soft palate showed an increased number of abnormal nerve endings in people who snore. The same author performed biopsies of the palatopharyngeal muscle and found morphological changes typical of neurogenic involvement, such as grouping of tissues by fiber type, clusters of atrophied areas, and fascicular atrophy, both in primary snorers and in patients with OSAS. Moreover, changes in the neural regulation of...
microcirculation were detected in the soft palate mucosa of snorers and some patients with mild OSAS\textsuperscript{12}. Other authors have also reported findings consistent with peripheral nerve injury in muscle biopsies from apnea patients\textsuperscript{13,14}.

Intense snoring causes stretching and low-frequency vibration of pharyngeal tissues\textsuperscript{15}. Takeuchi et al. demonstrated that long-term exposure to low-frequency vibration due to the occupational use of vibrating tools (chain saw, pneumatic drill) causes peripheral nerve injury and lesions in the microcirculation of the fingers in humans\textsuperscript{16}. It has also been demonstrated in dogs that oscillatory pressure waves (30 Hz and +/- 3 cm H\textsubscript{2}O) applied to the upper airway at the same frequency as snoring, affected pharyngeal receptors, increased local dilator muscle activity, and disrupted sleep\textsuperscript{17}.

Kimoff et al. have demonstrated a selective impairment of sensory function of the upper airway mucosa in OSAS patients. They found both decreased vibration sensitivity and reduced tactile discrimination between two points in the oropharynx of patients with primary snoring and OSAS compared to a non-snoring group without OSAS. Furthermore, these effects did not occur in control areas, such as the lips and hands\textsuperscript{1}. Nguyen et al. also noted a decrease in the sensitivity of the larynx and velopharynx, which positively correlated with the severity of the OSAS\textsuperscript{18}.

These data suggest changes in afferent and/or efferent neural pathways involved in the upper airway reflexogenic mechanism in OSAS patients\textsuperscript{1}. There is speculation about the role of this neuropathy in the progression of pharyngeal collapsibility, which is observed in OSAS\textsuperscript{1}; it is known that the permeability of the upper airway depends on the balance between the negative inspiratory pressure and the action of pharyngeal dilator muscles, which, in turn, requires a fully functioning neural afferent pathway to be stimulated\textsuperscript{1}. A possible causal relationship between snoring and the neurogenic lesions can be argued for. Although peripheral neuropathy can theoretically precede the onset of snoring, this is not universally found among snoring individuals and seems to arise during the course of the disease\textsuperscript{1}.

SWALLOWING DYSFUNCTION IN OSAS

Swallowing is a process divided into four distinct phases. In the oral preparatory phase, voluntary chewing and bolus formation take place. The oral phase itself consists on the elevation and posterior impulsion of food to the back of the oral cavity, featuring the final voluntary activity of swallowing. The pharyngeal phase is represented by the pharyngeal reflex, in which the most complex part of swallowing takes place in a rapid and coordinated fashion. The soft palate rises to seal off the nasopharynx, the larynx closes to protect the lower airway, and the caudal propulsion of the food bolus and relaxation of the cricopharyngeal muscle take place. In the esophageal phase, after the food passes through the upper esophageal sphincter, it is pushed through the esophageal muscles by primary and secondary peristalsis. The process is finalized with the relaxation of the lower esophageal sphincter and the arrival of food in the stomach\textsuperscript{19}.

Normal evocation of the swallowing reflex and the propagation of the food bolus through the pharynx are dependent on adequate pharyngeal sensitivity and function. The presence of sensory lesions in the pharyngeal mucosa of snorers may compromise the mechanism of swallowing\textsuperscript{10,11}.

Teramoto et al. performed a swallowing provocation test and showed a delayed triggering of the swallowing reflex (the food in the pharynx took longer to evoke the pharyngeal reflex) and the need for a greater volume of food bolus to initiate it in OSAS patients compared to a control group. This finding implies an increased risk of tracheal aspiration among OSAS patients\textsuperscript{5}.

On the other hand, Jobin et al. reported a significant reduction in the latency of the swallowing reflex in OSAS patients who were younger and obese, patients of a caucasian ethnicity (while Teramoto et al. studied Japanese patients), and patients with more severe OSAS. This study suggests an impairment of the reflex inhibitory modulation and central control of swallowing\textsuperscript{17}.

In studies using barium videofluoroscopy, Jaghagen et al. detected subclinical abnormalities in swallowing in more than half of patients with untreated primary snoring and OSAS compared to only 7% in the controls. The patients were significantly older than the controls, but the risk of swallowing dysfunction was not positively correlated with the severity of OSAS. The most frequent alteration was premature spillage of the food bolus (48%) to different levels of the pharynx before onset of the swallowing reflex. This finding corroborates the hypothesis that the neurogenic lesions of the oropharynx in snorers impair the sensory function of the mucosa and the triggering of the swallowing reflex. When early spillage occurs, chewing and breathing are not inhibited, and this may result in laryngeal penetration, when food reaches the laryngeal vestibule but does not pass through the glottis, or tracheal aspiration, when food passes through the glottis\textsuperscript{10,19}.

Another observed abnormality is the presence of residual food in the pharynx after complete swallowing and recovery of breathing, which occurs in 11% of patients. This also implies a risk of penetration/aspiration because the patient is not aware of the presence of the residual food, and the lower airway is unprotected. Laryngeal penetration was observed in 5% of the OSAS cases, but there was no tracheal aspiration, which may explain why many patients do not report dysphagia. Meanwhile, in the control group, there were no cases of pharyngeal residue, laryngeal penetration, or trache-
al aspiration. The only change observed was early escape of the bolus in one control individual (7%)\(^9\).

Jaglhagen et al. evaluated swallowing in primary snoring and OSAS patients who were selected for surgical treatment (uvulopalatopharyngoplasty and uvulopalatoplasty). During the preoperative evaluation, 17% of the patients had symptoms of dysphagia (i.e. clinical dysphagia). Among the asymptomatic patients (83%), more than half (51%) showed swallowing disorders during videofluoroscopic testing (i.e. subclinical dysphagia). In the postoperative period, considering the asymptomatic group, no significant difference was observed between patients with or without pharyngeal swallowing dysfunction with regards to the risk of developing clinical dysphagia. For those who were asymptomatic before the surgery, 29% reported dysphagia symptoms afterwards, but only half had the diagnosis confirmed by videofluoroscopy\(^20\).

Okada et al. reported two cases of patients with severe OSAS and swallowing dysfunction who improved after treatment with nasal continuous positive airway pressure CPAP and weight loss. They performed the swallowing provocation test before the treatment and one year after, and observed decreased latency of the onset of the swallowing reflex and also disappearance of tracheal aspiration in one patient. However, it was not possible to determine whether the patient’s improvement was due to weight loss or the use of CPAP\(^21\).

Recently, Valbuza et al. used nasal fiberoptic examination and observed subclinical swallowing abnormalities in patients with moderate to severe OSAS compared to a control group. Early escape of the food bolus occurred in 64% of patients, and food residue in the pharynx was found in 55% of patients. No cases of laryngeal penetration or tracheal aspiration were reported\(^22\).

Pharyngeal swallowing dysfunction is often a slowly progressive disorder in which the individual develops compensatory mechanisms, such as changes in the diet or chewing rate. Thus, symptoms may appear only when the compensatory strategies are overcome by the severity of the disorder. Before this point is reached, active medical intervention can detect swallowing impairment\(^23\).

We observe that in most cases, the complaint of dysphagia is not mentioned spontaneously by OSAS patients, but its perception reveals the potential impacts of OSAS on the patients’ quality of life, which is an additional motivator for seeking and adhering to treatment. This aspect is often ignored in the management of OSAS, and specific treatments (maneuver orientation, postural adjustments, facilitating therapies, and changes in the diet) may also have a positive impact on the quality of life of these patients.

REFERENCES

