

Editorial

The Upper Airway Resistance Syndrome: Fact or Fiction?

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Daytime fatigue and sleepiness caused by inspiratory flow limitation throughout NREM sleep (with associated arousals), in the absence of the clinical and polysomnographic features of obstructive sleep apnea hypoapnea syndrome (OSA/HS) has been described as the upper airway resistance syndrome (UARS) [1]. Some authors had tried to assign uniqueness to this relatively "young" syndrome, including polysomnographic features (increased alpha frequency during NREM sleep in patients with UARS) and palatal sensory function (intact in patients with UARS) [1,2]. Recent studies have shown that patients with UARS are more somnolent than patients with OSA/H, leading to the hypothesis of a cause-and-effect relationship between airway collapse and physiologic hyperarousals [3]. In one such study, pharyngeal critical pressure (Pcrit) was used as the index of upper airway collapsibility (the airway pressure at the flow-limiting site below which the flow-limiting site collapses). Patients with UARS have been characterized to have lower values of Pcrit when compared with patients with OSA/HS, and just mildly increased when compared to normal individuals [3].

In this issue of *Current Respiratory Medicine Reviews*, Dr. Guillemnault, the leading expert in UARS presents a comprehensive and up-to-date review on the clinical and pathophysiological variables that define this syndrome [4]. The correct characterization of this syndrome is at times quite difficult for practicing clinicians. There is still some controversy as to whether UARS is a true pathology or an artifact resulting from improper measurement of airflow [3]. The symptoms of UARS include insomnia, daytime sleepiness, sleep fragmentation, and fatigue. In many instances these symptoms may be confused with other pathological entities such as major depression, or chronic fatigue syndrome, or OSA/HS. The UARS has been described to be more frequent among women, in contrast with OSA/HS which is more common among men [1]. The long-term outcome between these two entities is also quite different. When a patient is diagnosed with OSA/H, they get immediate

treatment with the lifelong use of a continuous positive airway pressure device. On the other hand, patients that are diagnosed with UARS, in many instances are left untreated [5].

Controversy also arises as whether or not, these patients have accurate hypopnea scores, specifically in patients that have being diagnosed at centers that use thermal sensors [6]. Clearly, more evidence is needed from randomized, controlled trials, to attribute this syndrome its uniqueness and etiopathogenesis. Preliminary studies have tried to outline a theory that would explain if these patients have an abnormal central nervous system response in an attempt to adjust to changes in the upper airway patency during sleep, and that these responses may be genetically predisposed [7].

Whether or not the UARS when properly diagnosed and treated changes the long-term outcome of patients is subject to some debate. However, we believe that this syndrome is still at its infancy and we must address treatment to these patients and look at long-term outcomes.

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