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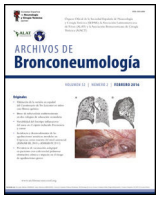

Editorial

Positional nasal congestion. Sleep apnea's forgotten cousin☆

La congestión nasal posicional. El hermano olvidado de la apnea del sueño

Nasal obstruction as an independent cause of sleep apnea is a controversial topic[1]. However, there is no doubt about its role in the subjective quality of sleep or in the tolerance and acceptance of CPAP[2] or mandibular advancement devices[3].

The sensation of nasal obstruction after going to bed is a common complaint among patients attending otolaryngology and pulmonology consultations, especially among patients with sleep disorders[4]. This phenomenon is known as a postural effect and is explained by inferior turbinate hypertrophy in the supine position.

The pathophysiology behind this phenomenon is still unclear. Three main hypotheses have been proposed. The first suggests that nasal venous stasis occurs in a supine position. This hypothesis is supported by the fact that nasal resistance has been shown to increase when the internal jugular vein is compressed[4]. The second hypothesis suggests a reflex phenomenon mediated by deep baroreceptors, since nasal resistance increases when the axillary artery or the sides of the body are compressed, without needing to be a supine position[5,6]. The third hypothesis, based mainly on animal studies, suggests that parasympathetic tone increases in the supine position[7–9].

There are several validated methods for measuring nasal ventilatory function, the gold standard being active anterior rhinomanometry[10]. Rhinomanometry calculates nasal resistance, i.e., the effort the subject has to make to breathe through the nose, from nasal airflow and the difference in pressure between the two nostrils. In the study of positional nasal congestion, De Vito et al. recommend performing both seated and supine rhinomanometry in a procedure they called positional rhinomanometry[11].

So far, 12 authors have explored changes in nasal resistance in a supine position using positional rhinomanometry, all of whom reported an increase in nasal resistance when patients lie down. Five groups included a total of 266 patients with snoring or sleep apnea[3,11–14]. The 4 studies that were suitable for pooling in a meta-analysis showed a combined effect of worsening nasal resistance of 0.20 Pa s/cm^3. Another 5 authors who studied healthy volunteers found a worsening of 0.10 Pa s/cm^3 in this subgroup. To date, only 2 controlled studies have been conducted. One compared healthy controls with patients with sleep apnea[13], while the other compared snorers with non-snorers[14]. In the first, Virkkula et al. found no statistically significant differences in increased nasal resistance between the two groups[13]. In contrast, Desfonds et al. did not perform this comparison, but they did provide data from which it could be calculated, revealing statistically significant differences, with a greater increase in nasal resistance being observed in patients who snore[14].

The reader unfamiliar with rhinomanometry might wonder if this effect is clinically relevant. Normal nasal resistance varies between 0.3–0.5 Pa s/cm^3. A level of resistance higher than 0.80 Pa s/cm^3 is considered severe nasal obstruction. Thus, it is understood that a worsening of 0.20 Pa s/cm^3 on the scale presented is clinically relevant. However, it is interesting to note that the relationship between nasal resistance and nasal airflow is exponential. In patients with borderline nasal resistance (0.3–0.5), small variations in nasal resistance can cause large variations in airflow. This may cause the patient to switch from nasal breathing to mouth breathing. Few authors have explored this variable. In their series, De Vito et al. found that 31% of patients with apnea, with normal baseline nasal resistance, developed pathological resistance in a supine position[11]. In this study, the authors set 0.5 Pa s/cm^3 as the limit, so this percentage may have been higher if a stricter standard, such as 0.3 Pa s/cm^3, had been used.

Little information is available on the treatment of this phenomenon. Topical nasal corticosteroids have been shown to normalize positional nasal congestion after 2 weeks in patients with allergic rhinitis[7]. A study in patients following radiofrequency turbinate reduction was performed in which positional nasal congestion was followed up in patients with vasomotor rhinitis[15]. However, no information is currently available on treatment in patients with sleep-disordered breathing.

In our opinion, the available evidence is insufficient to make clear recommendations. However, there is a clear imbalance between risk and benefit. First of all, we should ask our patients if nasal obstruction occurs when they go to bed, as we might be surprised by how common this complaint is. This possibility should even be explored in patients who are unaware of the problem, but who report that during the day they breathe through their nose, but at night they sleep with their mouth open. This effect can be confirmed by a risk-free examination that is available in many specialized centers. We therefore believe that, with the current evidence, we have an obligation to explore this possibility in our patients. The treatment of this condition may be more controversial. There is little doubt surrounding the use of topical nasal corticosteroids, which have a high safety profile, and radiofrequency turbinate reduction, a minimally invasive though more controversial surgical technique, may be worth exploring.

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Christian Calvo-Henríquez,[a,b,*] Romina Abelleira,[c] Francisco J. González-Barcala[c]

[a] *Rhinology Study Group of the Young-Otolaryngologists of the International Federations of Oto-rhino-laryngological Societies (YO-IFOS), París, France*

[b] *Service of Otolaryngology, Hospital Complex of Santiago de Compostela, Santiago de Compostela, La Coruña, Spain*

[c] *Service of Pneumology, Hospital Complex of Santiago de Compostela, Santiago de Compostela, La Coruña, Spain*

[*] Corresponding author.
E-mail address: Christian.Ezequiel.Calvo.Henriquez@sergas.es (C. Calvo-Henríquez).

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