Mechanical bronchodiilatation for asthmatic airway: role of CPAP therapy

Antonio M. ESQUINAS¹, Ezgi ÖZYILMAZ²

¹ Morales Meseguer Hastanesi Yoğun Bakım Ünitesi, Murcia, Spain,
² Çukurova Üniversitesi Tıp Fakültesi, Göğüs Hastalıkları Anabilim Dalı, Adana.

The mechanisms for nocturnal worsening of asthma are not fully understood. However, circadian rhythms of circulating hormones such as epinephrine, cortisol and melatonin as well as neural mechanisms including cholinergic tone are thought to play a role. In some cases, nocturnal airway narrowing is associated with sleep disorders such as obstructive sleep apnea syndrome (OSAS). Recently, nasal continuous positive airway pressure (n-CPAP), has been considered a potential supplemental therapy for its well known positive effects on lung mechanics as well as its effect on airways, inspiratory/expiratory flows and gas exchange; in essence functioning as a mechanical bronchodilator.

We read with interest the recent original study by Busk et al. looking at the effect of n-CPAP on airway hyper-responsiveness in a group of asthmatic patients (1). This article does provide important and relevant information; however, we would like to add the following comments in relation to its application in clinical practice;

1. Experimental studies show that n-CPAP may contribute to lower airway hyper-responsiveness by altering airway-parenchymal interdependence and this is considered to be an early adaptive and reversible mechanism on the airways (2,3). Complex interactions of immune modulation, acetylcholine response challenge, excitation-contraction coupling mechanisms of the smooth muscle cells that eventually result in changes to the structure and composition of the airway wall tissue. These responses are time dependent, hence, factors such as duration of asthma, chronic airway hyper-responsiveness, airway reversibility, fixed airway structural alterations due to asthma chronicity could influence n-CPAP response. In this study, no information is provided regarding the chronicity of asthma or the n-CPAP settings versus responses in the study subjects.

2. It is also important to know the interactions between n-CPAP and asthma medications such as bronchodilators and anti-inflammatory agents as they could have either a synergistic or antagonistic effect. In a previous report, Wang et al. showed that application of n-CPAP decreased airway hyper-responsiveness in patients with asthma and concomitant use of n-CPAP during inhalation of a low dose salbutamol significantly enhanced the bronchodilator effect (4). Medication information is lacking in this study.

3. One of the rationales to use n-CPAP in this study is due to the airway strain in asthmatic subjects. Airways strain could potentially be influenced by variables such as anthropometric index, polysomnography results,
forced expiratory volume in one second (FEV₁) and severity and duration of asthma. Unfortunately, this information is not available from this study. Hence it would be difficult to comment on n-CPAP therapy.

4. We feel the methodology of n-CPAP use could have been strengthened by the following considerations;

a) Before n-CPAP therapy: It would be useful to obtain information related to lung function, such as, pulmonary function tests, lung mechanics, gas exchange, spirometry, breathing patterns and subjective sensations during tidal breathing.

b) During n-CPAP: titration of pressures for n-CPAP and symptom monitoring should be carried out for adequate control of symptoms and for long-term recommendations of therapy.

In conclusion, we consider that n-CPAP in asthma may provide a potential physical therapy to hyper-responsive airways by acting as a mechanical bronchodilator. We do feel that a larger study is warranted for identifying ideal patient population and for understanding the n-CPAP methodology for the ultimate benefit of the patient.

CONFLICT of INTEREST

None declared.

REFERENCES


