

LETTERS TO THE EDITOR

Pleural pressure during sleep in Marfan syndrome: details about the CPAP effect

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We read with great interest the original study by Sowho et al¹ in which the authors evaluated the effect of continuous positive airway pressure (CPAP) on pleural pressure (Ple-P) in patients with Marfan syndrome (MFS) during sleep. It is well known that CPAP improves sleep patterns and reduces the Ple-P in various forms of sleep breathing disorders. However, we consider that it would be interesting to know the CPAP limits and pathways in these complex situations. Ple-P measurement and the CPAP effect depending on body position should have been addressed in the study, since there are studies indicating that increased Ple-P affects the mechanics of breathing.² Moreover, there are other factors that can influence esophageal pressure (Pes) and we consider that monitoring the effect of body position on Pes results could make this study more interesting. The study was performed in the supine position, and as position-dependent Pes measurements were not made in the study we do not know whether there are variations in these measurements with respect to left or right lateral decubitus. It is known that CPAP induces tidal volume variations. Therefore, variations in tidal volume could influence Pes measurements and CPAP effects. It is interesting to consider that CPAP induces variations in tidal volume distribution between ventral and dorsal lung regions and this aspect could influence Pes measurements.² We are of the opinion that the study did not take body mass index into account. Patients with MFS are often not obese as in our series and their neck circumference is normal; accordingly, it could have been useful to understand the possible presence of laxity of the soft palate or of the upper airways. It has been shown that nonventilatory treatment of apneas or flow limitation causes no effect on Ple-P and Pes.

Another important aspect to know is how these results can be extrapolated to patients with MFS with varying body mass indexes. This is supported by a recent study that found that, in patients with a body mass index of ≥ 50 kg/m², the CPAP results may be contradictory. We consider that it could be interesting to know the authors' opinion on the accuracy of Pes–Ple-P measurements in patients with MFS with respect to variations in body mass index.² There is a correlation between the MFS autonomic response and CPAP effect. We know that CPAP has an impact on autonomic response and reduces the sympathetic reflex activation and arousal in obstructive sleep apnea.³ On the other hand, the information on hemodynamic information

(blood pressure/heart rate) variations⁴ is unknown. The use of CPAP can provide an additional protective effect against hypertension that occurs in MFS. Authors have observed that, after CPAP treatment, patients were more bradycardic and their blood pressure and its standard deviation decreased as SaO₂ improved in non-rapid eye movement stage 2 sleep⁵ or the arousal-induced reflex sympathetic activation response was reduced. Finally, it would be interesting to know the CPAP limits in Ple-P control during sleep and its residual effect. This observation should be evaluated with the results of another study emphasizing that the alteration in Pes persists in patients with upper-airway sleep-disordered breathing and those with obstructive sleep apnea syndrome and can even last more than 20 minutes without an arousal on electroencephalography.⁶

We believe that further clinical trials based on Pes measurements are needed to determine the limits of CPAP levels appropriate with apnea intensity in MFS.

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