Can postural OSA be usefully identified from its severity alone?

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ABSTRACT

Introduction When obstructive sleep apnoea (OSA) does not occur throughout sleep, there must be factors influencing its presence or absence. These are most likely to be sleep stage, posture and presleep alcohol, among others. We hypothesised that as OSA severity increases, the likelihood of postural OSA (POSA) would also decrease.

Methods Laboratory sleep studies of 39 patients with OSA were manually reviewed to calculate supine and non-supine oxygen desaturation indices (ODI). The usual definition for POSA was used, a ratio of supine to non-supine ODI of ≥2.

Results The mean age was 53.2 (SD 12.4) years, the body mass index was 35.0 (SD 8.9) kg/m^2 and 74% were male. The median supine ODI was 54.3 (IQR 25.7–73.5) and non-supine ODI was 18.7 (IQR 8.6–38.4). The overall prevalence of POSA was 56%. The prevalence of POSA for ODIs of <40 was 68%, and 35% if ≥40.

Conclusions An ODI ≥40, compared with <40, halved the likelihood of POSA from 68% to 35%. Although there is clearly a relationship between overall ODI and POSA, it is not strong enough to diagnose an individual with POSA. However the relationship provides a useful way to prescreen trial subjects to enrich for POSA.

Key messages

► As the severity of obstructive sleep apnoea increases, the presence of a postural component decreases.
► Subjects with oxygen desaturation indices under 40/hour have double the prevalence of postural obstructive sleep apnoea compared with indices above 40 (68% vs 35%).

INTRODUCTION

When severe obstructive sleep apnoea (OSA) is present continuously throughout the night, its presence is unlikely to depend on such variables as sleep state, posture or presleep alcohol. As the all-night oxygen desaturation index (ODI) falls from 60 to, say, 30, usually this does not mean that apnoeas are now 2 min long, rather than 1 min, but that there are apnoeas (with the usual cycle time of about a minute) for only half the sleep time. Supine position clearly worsens OSA in many, due to increased compliance and collapsibility of the pharynx.[1] This increase in collapsibility is thought to be due to the direct gravitational effects of the mandible on the upper airway, and indirectly to a reduced functional residual capacity when supine, reducing the caudal traction on the pharyngeal walls, and thus reducing the bracing effect from such traction.[2] Postural treatments for OSA have been used for many years,[3] and their popularity has increased recently with the introduction of posture-control devices

that are somewhat more sophisticated than the tennis-ball-in-the-back-of-the-pyjamas approach.[4] It is usually argued that a patient should have significant postural dependency to make a trial of such a treatment worthwhile. The exact definition of postural OSA (POSA) varies,[5] but most commonly it is arbitrarily defined as a supine to non-supine ratio in the apnoea/hypopnoea index (AHI) or ODI of ≥2. With a view to potential efficacy of treatments, some have argued that the non-supine AHI should also be <5 to be defined as POSA.[6] There are very few robust data on postural therapies for OSA, with no placebo-controlled trials reporting longer term symptom-based primary outcomes.[7] Such multicentre trials in ordinary clinical units are needed[7] and will require simple recruitment strategies if they are to be clinically useful. We wondered if the severity of OSA on its own could be usefully used to prescreen patients for entry into such trials of POSA therapy, since earlier studies have suggested a falling off of POSA prevalence as the AHI increases.[6]

METHODS

Fifty clinical sleep studies (VISI-Lab, Stowood Scientific Instruments, Oxford, UK) between May and July 2016 were sequentially selected from patients diagnosed with OSA. Studies were excluded if technically inadequate, or from patients with significant associated central sleep apnoea (CSA) or hypoventilation, an ODI <5, or those with <30 min of either supine or non-supine sleep. The remaining sleep studies were carefully reviewed to calculate the ODI, both supine and non-supine,
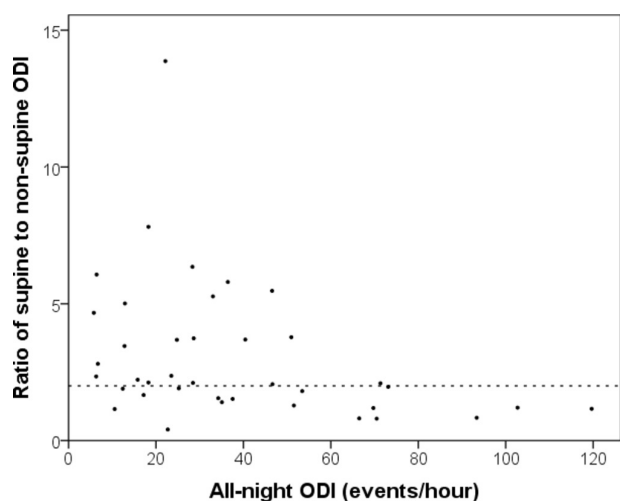
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Figure 1 Relationship between all-night oxygen desaturation index (ODI) and the ratio of supine to non-supine ODI. Horizontal line at a ratio of 2 is the conventional definition of postural obstructive sleep apnoea.

using video recording to accurately define posture. The ratio of supine to non-supine ODI was calculated and plotted against the all-night ODI. In addition, the prevalence of a supine to non-supine ratio of ≥2 was calculated for the two groups, depending on whether the all-night diagnostic ODI was <40 or ≥40.

RESULTS

Forty-six out of 50 sleep studies were technically satisfactory for this analysis. Three were further excluded due to hypoventilation or CSA, three due to <30 min of supine sleep and one due to an all-night ODI <5, leaving 39 for the final analysis. The mean age of this study group was 53.2 (SD 12.4) years, the body mass index was 35.0 (SD 8.9) kg/m^2 and 74% were male. The median all-night ODI was 28.6 (IQR 17.2–51.4), supine ODI was 54.3 (IQR 25.7–73.5) and non-supine ODI was 18.7 (IQR 8.6–38.4). The clear relationship between the all-night ODI and the ratio of supine to non-supine ODI is shown in figure 1 (Spearman's rank correlation, −0.44, P<0.005). Taking the arbitrary definition for POSA as a ratio of ≥2, the overall prevalence was 56%. If the all-night ODI was <40, then the prevalence of POSA was 68%, and if ≥40 then the prevalence was 35%. The prevalence of POSA when the all-night ODI is over 60 appears much smaller (figure 1), but our study numbers do not allow a robust estimate.

DISCUSSION AND CONCLUSIONS

The overall prevalence of POSA was 56%, in line with previous reports.[8] As expected, the likelihood of having POSA increased as the ODI fell, such that if the all-night ODI was <40, then the prevalence was almost double (68%) that of those with an all-night ODI ≥40 (35%). At the conventional ODI cut-off point for mild/moderate versus severe OSA of 30, the prevalences of POSA in the mild/moderate and severe groups were 75% and 37%, respectively. The 32% prevalence of non-POSA in those with an all-night ODI of <40 is presumably due to other factors that only provoke OSA for part of the night, such as rapid-eye-movement sleep. Thus the diagnostic ODI from a sleep study, where posture is not routinely measured, could be used to enrich the likelihood of finding POSA when, for example, screening a patient for entry into a randomised controlled trial of POSA therapy. However, at a clinical level, it may not be useful since, even at ODI values over 40, there is still a one-third chance that a patient will have POSA, using the conventional 2:1 supine to non-supine definition.

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