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Journal search and commentary

Article reviewed: Case-control study of 24 h ambulatory blood pressure in patients with obstructive sleep apnoea and normal matched control subjects ☆

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Objective

To determine if Obstructive Sleep Apnea (OSA) is an independent correlate of systemic blood pressure during the day.

Study Design

Case-control study.

Study Population

Forty-five OSA patients (>4% falls in oxyhemoglobin saturation/h: 29.1 ± 3.4, mean ± SD) and 45 control subjects without OSA (>4% falls in oxyhemoglobin saturation/h: 1.7 ± 1.6), groups matched for age (OSA: 51.7 ± 10.4 years; no OSA: 52.2 ± 10.4), BMI (OSA: 30.9 ± 2.8; no OSA: 30.5 ± 2.4), alcohol consumption, smoking status (never or former/current), treated systemic hypertension, ischemic heart disease (defined as history of angina pectoris and/or myocardial infarction) as well as a diagnosis of diabetes. The study groups were exclusively male.

Groups were not matched for waist/hip or waist/height ratios and these ratios were both greater in the Control group.

Methods

Study participants with OSA were recruited from individuals who had been referred to the Sleep Clinic. They underwent in-laboratory sleep studies during which arterial oxyhemoglobin saturation (S_pO_2) was recorded by pulse oximetry, and pulse rate, movement as well as sound were recorded. Obstructive events were determined from a video recording. OSA was defined by the presence of >10 episodes/h in which S_pO_2 fell by 4%.

Control subjects, initially identified as potentially matching OSA patients, were selected from the local general practice register and were invited by their respective General Practitioner to participate in this research. Each had a home sleep study during which S_pO_2, snoring, chest wall movement, body position, and heart rate was recorded. The exclusion criterion was >5 episodes/h in which S_pO_2 fell by 4%. Symptoms consistent with OSA were not considered to be independently exclusionary.

Twenty-four hour systemic arterial blood pressure was recorded using a validated recorder which measured blood pressure by arm cuff using Korotkov sounds. The cuff was inflated every 30 min throughout

☆ Davies CWH, Crosby J, Mullins R, Barbour C, Davies RJO, Stradling JR. (Thorax 2000;55 (9):736-740).

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the 24-h period. When this method provided unreliable recording the oscillotonometric method was employed.

Results

The OSA group had significantly higher diastolic blood pressure throughout the 24-h period compared with the Control population. Systolic blood pressure was higher in the OSA group only during the night. Nocturnal systolic did not fall to the same degree in the OSA patients as was observed in the Control group. Inclusion or exclusion of individuals receiving anti-hypertensive medication in the analyses did not influence these results.

Conclusion

The investigators concluded that when compared to a closely matched control population OSA patients have significant elevation of systemic blood pressure and that this difference is sufficient to confer increased risk for cardiovascular morbidity.

Comment

As pointed out in the Editorial by Fletcher in the same issue as the above paper [1], this study adds to the increasing body of evidence supporting an independent association between OSA and systemic hypertension. Several recently published papers, examining community populations or large groups of patients referred to a sleep clinic (spanning a range of Sleep-Disordered Breathing severity) have demonstrated such an association employing statistical techniques to adjust for potentially confounding co-variates or identify parameters of Sleep-Disordered Breathing which are independently associated with hypertension [2–5]. The study by Davies et al. examines the issue from another perspective, in a case-control fashion, and reached the same conclusion. The fact that these investigators did not perform 'traditional' polysomnography should not detract from the validity of their results and conclusions. The methodology employed seems reasonable and appropriate to address the question. However, it cannot be assumed that the findings are applicable to women with OSA. The important question of cardiovascular risk associated with OSA in women demands investigation. Additionally, as has been often stated, association should not be presumed to reflect causality. The propriety of initiating treatment for OSA, on the basis of cardiovascular status is best justified after demonstration of such causality, and preferably with identification of pathophysiological mechanism(s). Obvious exceptions are patients with sleep-related angina or potentially life-threatening cardiac rhythm abnormalities.

References

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