Obstructive sleep apnoea and hypertension

Prof James Ker, Senior Lecturer, Department of Internal Medicine, University of Pretoria

Obstructive sleep apnoea/hypopnoea syndrome (OSAHS) is one of the most important medical conditions documented in the last 50 years.[1] OSAHS is a major cause of morbidity and a significant cause of premature death.

OSAHS raises the mean 24-hour blood pressure (BP) by 5mmHg to 10mmHg and the blood pressure increase is more in those patients with >20% arterial oxygen desaturation per hour of sleep. This rise in mean BP can lead to 20% increased risk of myocardial infarctions and 40% increased risk of stroke.

The rise in BP is a result of surges of BP accompanying each arousal when the apnoea/hypoponea episode is terminated and as a result of an increased 24-hour sympathetic tone due to the repetitive episodes of hypoxaemia. These changes are associated with increased urinary levels of catecholamines, especially in those with more severe OSAHS, which reduces with continuous positive airway pressure (CPAP) treatment.

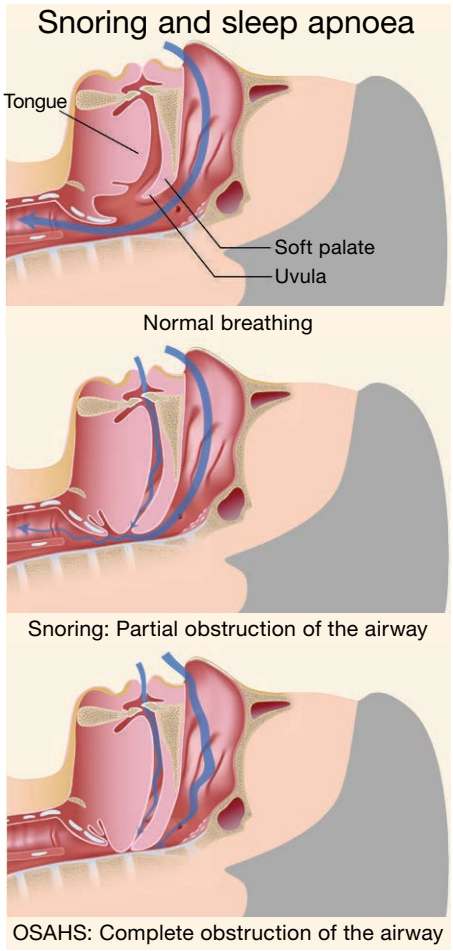
OSAHS and hypertension frequently co-exist. It is estimated that about 20% of patients with hypertension may have OSAHS and in people with OSAHS the prevalence of hypertension could be 50%.

There are some epidemiological data linking the presence of OSAHS with hypertension. In a recent epidemiological study over a mean of more than 12 years, OSAHS and hypertension were associated independent of age or obesity as confounders. This also showed that the severity of OSAHS increased the probability of hypertension.[2]

An association between OSAHS and elevated BP has also been reported in specific groups of patients such as elderly women, pre-hypertensive patients, primary care subjects, patients after spinal cord injury and in patients after stroke. The relationship between OSAHS and elevated BP is also evident in children, however as in adults, the challenge remains to determine what portion of the elevated BP is due to obesity or to the OSAHS or to a combination of both.[3]

A decrease of BP at night is a normal physiological occurrence and this dipping pattern is associated with health. OSAHS changes this pattern and the nocturnal dipping is lost in OSAHS and this increases the risk of cardiovascular events in OSAHS. However, there is not enough research data available to be absolutely certain.[3]

It has been shown that the possible mechanism linking OSAHS and an increased cardiovascular morbidity could be due to an increased burden of systemic inflammation and higher levels of high-sensitive C-reactive protein (hs-CRP), interleukin-1, interleukin 6 and 8 and tumour necrosis factor-. It is unknown if these markers truly increase morbidity/mortality and if treatment of these biomarkers could alter the prognosis. It is also unknown whether salt-retention and hyperaldosteronism could alter the OSAHS-hypertension relation.



OSAHS is an important cause of impaired sleep quality and short sleep duration has been postulated as a potential contributor to an elevated BP.

There are not much data on OSAHS and structural changes of the heart and blood vessels. Treatment of OSAHS has shown that both right and left ventricular function to improve over the short term. It was recently recognised that OSAHS is prevalent in ischaemic and dilated cardiomyopathy and also thoracic aorta dilatation. OSAHS has been shown to cause endothelial dysfunction and this could be reversible with CPAP treatment.

EFFECTS OF TREATMENT OF OSAHS ON BP

It is still not very clear whether treatment of OSAHS with CPAP could really make a difference as far as BP reduction is concerned but a meta-analysis showed a weighted reduction in systolic and diastolic BP of 2.58mmHg and 2.01mmHg with CPAP treatment. It is not yet known which factors could lead to a significant reduction of BP in OSAHS with CPAP therapy. In a pilot study in diabetic patients with hypertension and OSAHS the use of CPAP led to a substantial decrease in BP. Much more studies on different patient groups are needed with CPAP therapy.

CONCLUSIONS

1. Epidemiological data implicates OSAHS as one of the modifiable and prevalent factors in the development of hypertension.
2. A nocturnal non-dipping pattern of BP has been shown in adults with OSAHS but not in children.
3. Patients with OSAHS have higher diastolic BPs during exercise.
4. OSAHS is an important cause of resistant hypertension.
5. The role of CPAP in the treatment of OSAHS is still controversial, whether it will reduce the development of hypertension in patients with OSAHS.
6. CPAP treatment has been shown in studies to be a worthwhile treatment modality in patients with resistant hypertension.

REFERENCES

1. Harrison's Principles of Internal Medicine.18th Edition 2012; vol. 1: 623.
2. Marin JM, *et al.* Association between treated and untreated obstructive sleep Apnoea and risk of hypertension. *JAMA* 2012; 307: 2169-2176.
3. Konecny T, *et al.* Obstructive sleep Apnoea and hypertension: an update. *Hypertension* 2014; 63: 203-209. SF