AIMS

- to describe the pathophysiological effects of obstructive sleep apnoea on the development of coronary artery disease,
- to describe the epidemiological evidence that links obstructive sleep apnoea with coronary artery disease,
- to describe the effects of treatment for obstructive sleep apnoea on prognosis of the coronary artery disease based on evidence from clinical trials.

SUMMARY

Epidemiological studies suggest that obstructive sleep apnoea (OSA) is over-represented in patients with coronary artery disease (CAD). Studies have also shown that the clinical course of CAD is initiated or accelerated by the presence of a coexisting OSA. A rapidly evolving field of experimental data suggests that OSA, by mechanisms such as hypoxemia and reoxygenation, may trigger a sequence of events subsequently leading to development of atherosclerotic disease. However, it is reasonable to believe that the risk of developing vascular disease, and more specifically CAD, as a consequence of OSA is influenced by several yet unidentified genotypic and phenotypic risk factors. Obstructive events during sleep induce a state of increased cardiac oxygen demand but they are also frequently associated with low oxygen reserve due to lack of ventilation. This generates a situation where nocturnal angina can be triggered by OSA in CAD patients. There is growing evidence that elimination of OSA by continuous positive airway pressure (CPAP) benefits patients at sleep clinic cohorts with concomitant CAD. However, a causal relationship between OSA and CAD is still not fully conclusive. As many of the CAD cases with OSA do not report daytime sleepiness, these patients are not recognized and considered for CPAP treatment, either. In one recent randomized controlled trial, routine prescription of CPAP to CAD patients with nonsleepy OSA did not show significant reduction in long-term cardiovascular outcomes but beneficial effects were observed after adjustment for baseline comorbidities and adherence to CPAP treatment. Results from other ongoing randomized controlled trials in larger clinical cohorts may hopefully give more insights into the causal relationship between OSA and CAD.

REFERENCES