Clinical update

Importance and management of chronic sleep apnoea in cardiology

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Sleep apnoea is a common, yet underestimated, chronic disorder with a major impact on morbidity and mortality in the general population. It is quickly becoming recognized as an independent risk factor for cardiovascular impairment. Hypertension, coronary artery disease (CAD), diabetes, cardiovascular rhythm and conduction abnormalities, cerebrovascular disease, and heart failure (HF) have all been linked to this syndrome. This review will explore the critical connection between sleep apnoea and chronic cardiovascular diseases while highlighting established and emerging diagnostic and treatment strategies.

Keywords
Sleep apnoea • Heart failure • Hypoxaemia • Cardiovascular disease

Introduction

Sleep apnoea is a common, yet underestimated, chronic disorder with a major impact on morbidity and mortality in the general population. It is quickly becoming recognized as an independent risk factor for cardiovascular impairment. Hypertension, coronary artery disease (CAD), diabetes, cardiovascular rhythm and conduction abnormalities, cerebrovascular disease, and heart failure (HF) have all been linked to this syndrome. This review will explore the critical connection between sleep apnoea and chronic cardiovascular diseases while highlighting established and emerging diagnostic and treatment strategies.

Classification

Sleep apnoea describes a syndrome of nocturnal respiratory interruptions resulting in sleep fragmentation, daytime hypersomnolence, and oxyhaemoglobin desaturation, usually unrecognized by the patient.¹ The severity of sleep apnoea is commonly quantified by the apnoea-hypopnoea index (AHI), which is the number of apnoeic and hypopnoeic events per hour. An AHI of 5–15 indicates mild disease, 15–30 indicates moderate disease, and >30 indicates severe disease. The prevalence of sleep apnoea in the general population appears surprisingly high, even when bias is taken into account in both the selection of subjects referred for sleep studies and their willingness to participate. Sleep apnoea, of any degree, was reported to be present in 17% of adults according to one study, and moderate to severe sleep apnoea (AHI ≥ 15) in 5.7%.² Other studies found the prevalence of moderate sleep apnoea to be between 1 and 14%.³ It is reasonable to assume that ~1 in 5 adults has at least mild sleep apnoea, and 1 in 15 adults has at least moderate sleep apnoea.³

The sleep apnoea syndrome can be divided into obstructive sleep apnoea (OSA), central sleep apnoea (CSA), and the combination of the two. Obstructive sleep apnoea is caused by nocturnal upper airway collapse with preserved breathing efforts during apnoea. The risk of OSA rises with increasing body weight; active smoking; diabetes; age; and influence of alcohol, sedatives, and muscle relaxants.⁴ Central sleep apnoea, also clinically known as Cheyne-Stokes respiration, is a result of decreased ventilatory drive, mainly due to loss of fine tuning of the breathing control system. It is especially common in patients with heart failure or stroke when the voluntary respiratory control is disabled during sleep.⁵ Patients with CSA have enhanced chemoreceptor sensitivity.⁶ Relatively minor increases in nocturnal PaCO₂ can cause an exaggerated hyperventilatory response, often driving PaCO₂ below the apnoeic threshold. The resulting apnoea causes CO₂ to build up, perpetuating the cycle. A prolonged circulation time may further delay chemoreceptor response to hypoxia and hypercapnia, further desynchronizing the circulatory, respiratory, and neurological systems⁷ (Figure 1).

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Some patients have both types of respiratory sleep disorder. They are, for the most part, patients with long-standing OSA or heart failure who have developed chronic malfunctioning of the respiratory regulatory system.\(^1\)

### Pathophysiology of chronic sleep apnoea

Patients with sleep apnoea suffer a host of haemodynamic and biochemical derangements.\(^8\) Obstructive sleep apnoea causes sharp decreases in intrathoracic pressure which increases venous return, results in an acute leftward intraventricular septal shift, alters transmural cardiac pressures, impairs left ventricular filling, increases myocardial oxygen demand,\(^9\) and causes ischaemia.\(^10\) In addition, haemodynamic derangements and the stress of frequent awakenings lead to increased catecholamine release by stimulating central and peripheral chemoreceptors and eliminating the reflex inhibition of sympathetic activity.\(^11\) Hypoxaemia and retention of CO\(_2\) in sleep apnoea cause chemoreflex-mediated increases in sympathetic activity with consequent vasoconstriction and acceleration of the heart rate.\(^12\) This can lead to marked fluctuations in blood pressure.\(^13\)–\(^15\)

Changes in oxygenation and intrathoracic pressure have also been associated with acute changes in pulmonary vascular resistance. Pulmonary artery pressure increases over the course of an apnoeic episode due to a combination of progressive hypoxia, decreased intrathoracic pressure, increased right heart output, and decreased left heart compliance.\(^16\) (Figure 2).

Furthermore, hypoxia activates nucleated cells in the body, which sense and respond to reduced oxygen tension.\(^17\) The condition stimulates transcription factors such as hypoxia-inducible factor 1 (HIF-1) and nuclear factor \(\kappa\)B (NF-\(\kappa\)B) which regulate the expression of genes that mediate inflammatory responses.\(^8\)–\(^17\) This process generates reactive oxygen species that consume intravascular nitric oxide (NO), and impair endothelial-mediated vasodilation.\(^18\) Nuclear factor \(\kappa\)B orchestrates the expression of cytokines [tumour necrosis factor (TNF)-alpha, IL-6, and IL-8], adhesion molecules (VCAM-1, E- and L-selectin, ICAM-1, and CD15), and cyclo-oxygenase enzymes (cyclo-oxygenase 1).\(^19\)–\(^20\)
Hypoxia-inducible factor 1 promotes angiogenesis and can potentiate atherosclerosis by stimulating vascularization within the atherosclerotic plaque. Inflammatory cytokines enhance the survival of myeloid inflammatory cells, such as granulocytes, monocytes, and macrophages, increasing their functional longevity and inflammation. Sleep apnoea is independently associated with higher levels of C-reactive protein, IL-6, IL-8, IL-18, TNF-alpha, homocysteine, leptin, and matrix metalloproteinase.

The severity of sleep apnoea, and, particularly, the severity of oxyhaemoglobin desaturation, is linked to increased levels of TNF-alpha and IL-6, cytokines which relate to hyperglycaemia, increased basal beta-cell function, insulin resistance, and overt type 2 diabetes. In patients with type 2 diabetes, OSA is associated with poorer glucose control measured by HgB A1C, independent of adiposity and other confounders.

Cross-sectional studies have shown a correlation between hyperlipidaemia and sleep apnoea, with obesity as a possible confounder. Animal models have demonstrated a direct link between hypoxia and elevated cholesterol and liver triglyceride content. Sleep apnoea may even play a role in the development of non-alcoholic steatohepatitis (NASH), with one study finding the prevalence of sleep apnoea to be near 50% in patients with NASH.

Inflammation provoked by chronic sleep apnoea might cause considerable endothelial damage. Sleep apnoea patients have increased numbers of circulating apoptotic endothelial cells compared with controls. These levels correlate with abnormal brachial artery flow-mediated dilation, a marker of endothelial dysfunction. Increased intima-media thickness, associated with higher levels of C-reactive protein, IL-6, and IL-18, is also strongly related to the duration of nocturnal oxyhaemoglobin desaturation. This vascular compromise is a common pathway for the development of multi-organ dysfunction (Figure 3).

Hypertension

Large cross-sectional and longitudinal studies have shown a strong correlation between chronic sleep apnoea and arterial hypertension, independent of potential confounders. A large prospective study also demonstrated a strong association between the presence of sleep apnoea and the development of hypertension. A distinct feature of sleep apnoea-induced hypertension is loss of the normal nocturnal decrease in blood pressure. This indicates a link between these two disorders.

Intermittent hypoxia and reoxygenation, as well as the stress of haemodynamic derangements and frequent awakenings, lead to
decreased NO levels, higher catecholamine levels, and increased blood pressure. These changes are partially reversible with continuous positive airway pressure (CPAP) therapy as demonstrated by numerous well designed randomized, controlled studies. Treatment is especially important in cases of resistant hypertension since more than 80% of these patients have been reported to have sleep apnoea.

The relationship between sleep apnoea and hypertension may be even more complex. Resistant hypertension is highly correlated with hyperaldosteronism, which promotes accumulation of fluid within the neck and can worsen sleep apnoea. Moreover, increasing evidence suggests that blocking aldosterone in patients with resistant hypertension can both improve the severity of sleep apnoea while decreasing blood pressure.

**Coronary artery disease and acute coronary syndrome**

The correlation between sleep apnoea and the development of CAD and acute coronary syndrome (ACS) has been well established. In one study, sleep apnoea was found in 65.7% of patients admitted to the hospital for acute myocardial infarction (MI). The co-existence of CAD and sleep apnoea portends a poor prognosis with a large prospective study demonstrating a 70% relative and a 10.7% absolute increase in death, cerebrovascular events, and MIs compared with those having CAD alone. A similar study demonstrated that in patients treated successfully with percutaneous coronary intervention after an ACS, the presence of sleep apnoea was associated with higher mortality (38 vs. 9%), increased rate of stent restenosis (24 vs. 5%), and increased rate of major adverse cardiac events (37 vs. 15%) within 1 year.

Optimal treatment of sleep apnoea may have important clinical implications for patients with refractory angina. A small study done on patients who suffered from nocturnal angina showed that 9 out of 10 patients were suffering from sleep apnoea, with a consistent temporal association between apnoeic episodes and the onset of angina. The administration of CPAP diminished the number of anginal episodes and the number of nocturnal myocardial ischaemic events. There are also case reports of patients suffering...
from typical angina symptoms, without critical CAD seen on cardiac catheterization, who were found to be suffering from sleep apnoea.64

Although treatment of sleep apnoea has not been shown to reverse progression of CAD, it might retard its progression. Treatment of sleep apnoea with CPAP can decrease the rate of occurrence of new cardiovascular events in patients with known CAD.65 The results from the large ‘Randomized Intervention with CPAP in Coronary Artery Disease and Sleep Apnoea’ (RICCADSA trial) are still pending but will soon help to shed light on the benefit of CPAP in this population.66

Sleep apnoea increases blood coagulability and viscosity.67 These patients have increased platelet aggregability, higher levels of clotting factors, and reduced fibrinolytic capacity.68 These findings could, in addition to contributing to CAD progression, predispose to thrombus formation and in-stent thrombosis.

Arrhythmia

Sleep apnoea is associated with hypoxia, autonomic derangements, and cardiac structural changes, all of which predispose to arrhythmia. Cross-sectional studies have indicated a strong association between atrial fibrillation (AF) and sleep apnoea, independent of age, sex, hypertension, HF, and body mass index.69 These studies have reported a sleep apnoea prevalence of 43–73% among those with AF, with high rates of CAD and CSA in this group.70

The degree of nocturnal hypoxia was also shown to be directly correlated with the risk of developing AF.71,72 Hypoxia causes autonomic abnormalities, diastolic dysfunction, and systemic inflammation leading to cardiac remodelling and alterations in cardiac conduction leading to the development of AF.72,73

The presence of OSA portends a 25% greater risk of AF recurrence after catheter ablation.74 Treatment with CPAP prior to an ablation procedure is associated with an eight-fold improvement in the lasting success of this procedure.75,76

Sleep apnoea, especially in association with HF, has been linked to a host of other cardiac dysrhythmias, including nocturnal paroxysmal asystole, bradyarrhythmias, atrioventricular nodal block, supraventricular tachycardia, and non-sustained ventricular tachycardia.77 People with OSA have dramatically increased risk of sudden cardiac death during sleep. This risk was clearly associated with the AHI.78 A study of HF sufferers with implanted defibrillators further demonstrated that sleep disordered breathing is associated with an increased incidence of malignant ventricular arrhythmias.79 A similar study demonstrated a predominance of nocturnal life-threatening ventricular arrhythmias in sleep apnoea patients with no significant increase in daytime arrhythmic activity.80

Treatment with CPAP decreases nocturnal rates of sinus bradycardia, sinus pauses, paroxysmal AF, premature ventricular contractions, and ventricular ectopy.81–83 In one study, CPAP completely eliminated pathologically significant nocturnal rhythm disturbances in seven of eight patients.84

Heart failure

Sleep apnoea is extremely common in people with heart failure. Estimates of prevalence are as high as 47–76%.85–88 Alarmingly, one study reported an average AHI of 44 in HF patients with sleep apnoea.89 The relationship between sleep apnoea and HF is complex. Chronic sleep apnoea causes a series of derangements that could potentially lead to the development or exacerbation of HF. Hypoxaemia, often aggravated by coexisting anaemia, is linked with acute increases in BNP concentrations in patients with HF, independent of the frequency of apnoeic events.89 Furthermore, hypertension, CAD, diabetes—all well established risk factors for HF—are adversely impacted by sleep apnoea.

Sleep apnoea induced increase in sympathetic tone and heart rate are especially detrimental in HF. The heightened level of stimulation is maladaptive to an already failing heart and can lead to myocyte injury, cardiac beta-adrenoreceptor desensitization, and functional and structural abnormalities.90 Studies have also shown that higher sympathetic activity is associated with worse outcomes in HF.91,92 Updated HF guidelines are beginning to stress the importance of carefully screening and treating HF patients for sleep apnoea.93

Heart failure can both cause and exacerbate OSA and CSA, initiating a potentially devastating cycle. The severity of HF, as measured by cardiac index, correlates with the degree of CSA.4,95 Fluid shifts while supine result in increased neck vein congestion, neck circumference, pharyngeal airflow resistance, and pharyngeal collapsibility, all of which cause obstructive symptoms.96 HF is also linked with Cheyne-Stokes respiration; animal models suggest that pulmonary congestion activates lung vagal irritant receptors, which causes hyperventilation and decreased CO2, as well as stretch receptors, which elicits a strong inhibitory reflex (Hering–Breuer inflation reflex). This reflex results in a cessation of breathing followed by a period of arousal and hyperventilation that further destabilizes breathing.97,98 In addition, prolonged circulation time may play a role, with the increase in circulatory delay in HF patients altering physiological feedback mechanisms99 (Figure 1).

Cheyne-Stokes respirations are a powerful independent predictor of poor prognosis in patients with CHF.100 Patients with severe heart failure can even experience Cheyne-Stokes respirations during daytime. This phenomenon is associated with higher levels of biomarkers of cardiac dysfunction and is an independent predictor of mortality.101,102 It is important to note that the classic symptoms of Cheyne-Stokes respiration, which are fragmented sleep, paroxysmal nocturnal dyspnoea, orthopnoea, and daytime fatigue, can easily be interpreted as worsening HF symptoms.103 Treatment of CSA may improve HF symptoms and quality of life.

Diastolic dysfunction is also highly correlated with sleep disordered breathing and nocturnal hypoxaemia.104–107 The prevalence of sleep apnoea in patients who have HF with preserved ejection has been reported to be as high as 70%, with a predominance of OSA.108 The cause of the link between OSA and diastolic dysfunction is not well defined, and could be related to hypertension, obesity, ischaemic heart disease, or other factors.
Pulmonary hypertension

Classically, sleep apnoea has been associated with pulmonary hypertension (PH) and cor pulmonale. Indeed, after excluding people with primary lung diseases, the prevalence of PH is ~15–20% in sleep apnoea patients. The development of PH is not, however, related to the severity of sleep apnoea symptoms, and it is uncertain how often the sleep apnoea or the concomitant abnormalities are the cause. For example, nearly half of sleep apnoea patients with PH have increased pulmonary capillary wedge pressure, suggesting left heart dysfunction. In other patients, the presence of PH appears to be related to the amount of sleep time with oxygen saturation <90%. Continuous positive airway pressure shows some benefit in reversing pulmonary arterial hypertension. In the only controlled trial of CPAP which specifically looked at pulmonary artery pressures, effective CPAP reduced average pulmonary systolic pressure by 5 mmHg.

Diagnosis

The wide range of possible severe consequences of untreated sleep apnoea mandates prompt diagnosis. The gold standard is monitored polysomnography in a sleep laboratory. This modality uses multiple biometric recording devices to accurately quantify the number of apnoea (a 90% reduction in tidal volume lasting >10 s) and hypopnoea (a reduction in tidal volume of 50–90%, lasting >10 s accompanied by >3% decrease in oxyhaemoglobin saturation) episodes occurring during a night’s sleep.

Despite the high sensitivity and specificity of polysomnography referral for a sleep study is often difficult, inconvenient, and expensive. For this reason, a number of ambulatory screening systems have been devised. Some devices can record information on multiple data sets including respiratory pattern, respiratory effort, pulse oximetry, heart rate, and peripheral arterial tone. Other devices record only one signal. In general, the more complex devices can obtain very high positive predictive values at the expense of negative predictive values, whereas more basic devices, such as nocturnal oximetry, are highly effective at ruling out sleep apnoea (negative predictive value 96.9%) but suffer in terms of their ability to reliably confirm the diagnosis.

Recent studies show that for selected patients with a high pre-test probability of having sleep apnoea, home-based diagnostic and monitoring systems are not inferior to conventional hospital-based polysomnographic diagnosis, with significantly lower cost. Moreover, a home-based approach to diagnosis and treatment may facilitate remote monitoring and may promote greater compliance.

The Portable Monitoring Task Force of the American Academy of Sleep Medicine recommends that unattended portable monitoring for the diagnosis of OSA should be performed in conjunction with a comprehensive sleep evaluation. They also recommend that portable monitoring should be used as an alternative to polysomnography only in patients with high pre-test probability of moderate to severe OSA.

Management

Once sleep apnoea has been diagnosed, the first remedial steps involve eliminating aggravating factors. This includes avoidance of alcohol or sedating medications, elevation of the head of the bed, and change of sleeping position to one’s side to prevent the tongue from falling back and obstructing the airway. Patients should also be encouraged to lose weight. A 10% weight reduction correlated with a 26% decrease in AHI in a population-based study. HF treatment should be optimized with an emphasis on minimizing extravascular fluid accumulation with effective diuresis. Angiotensin-converting enzyme inhibitors and beta-blockers have both been shown to reduce CSA in CHF, presumably by improving the underlying HF.

The standard treatment for patients diagnosed with OSA is nocturnal CPAP usually delivered via a tight fitting nasal mask. Continuous positive airway pressure should be offered to all patients diagnosed with OSA, as the benefits of CPAP in this population are unquestionable. Continuous positive airway pressure therapy has been shown to lower blood pressure, reduce cardiac rhythm disturbances, decrease cholesterol, improve insulin resistance and management of diabetes, reduce the levels of inflammatory cytokines, reverse autonomic derangement, and improve endothelial function. Several long-term studies have also reported that CPAP reduces the incidence of cardiovascular disease and improves long-term cardiovascular morbidity and mortality among patients with severe sleep apnoea.

Heart failure patients require special consideration due to the high incidence of both OSA and CSA. In those with a predominance of OSA, CPAP once again has shown some benefit, although evidence of long-term improvements in morbidity and mortality from randomized controlled trials is lacking. Studies have shown that CPAP decreases sympathetic activation, improves systolic function, significantly reduces the left ventricular end-systolic diameter, and improves quality of life. In one study, CPAP improved the left ventricular ejection fraction from an average of 25 to 33.8%.

The treatment of CSA in HF is controversial. Since CSA is likely a manifestation of advanced HF, optimizing medical management of HF may improve symptoms of CSA. The largest randomized study, ‘Canadian Continuous Positive Airway Pressure for Patients With CSA and HF’ (CANPAP), demonstrated no effect on heart transplant-free survival, but this trial was underpowered. It did, however, demonstrate improvements in apnoea frequency, blood oxygenation, norepinephrine levels, and LV function.

Other assisted breathing devices have shown promising results. Flow-targeted dynamic bilevel positive airway pressure (BPAP), which modulates inspiratory pressure in order to maintain a target inspiratory airflow has been shown to be especially effective in CSA. That is likely due to the associated pulmonary oedema and reduced lung compliance which can be impacted by BPAP. Adaptive servo-ventilation (ASV) therapy is a new positive airway pressure technology that tracks a patient’s breathing pattern and adjusts breath-by-breath pressure support to counterbalance the fluctuations in respiratory rate. It will decrease the rate of pressure supported ventilation during periods of hyperventilation and...
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Table 1  Summary of treatment modalities and outcomes in obstructive and central sleep apnoeas

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Obstructive sleep apnoea</th>
<th>Central sleep apnoea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight loss</td>
<td>Improvement in sleep apnoea symptoms and AHI</td>
<td>No proven benefit</td>
</tr>
<tr>
<td>Optimization of heart failure</td>
<td>No clear benefit</td>
<td>Improvement in central sleep apnoea and decrease in heart failure-related morbidity and mortality</td>
</tr>
<tr>
<td>CPAP</td>
<td>Proven benefit in terms of AHI as well as cardiovascular morbidity and mortality</td>
<td>No proven decrease in morbidity and mortality; likely improvements in apnoea frequency, blood oxygenation, norepinephrine levels, and LV function</td>
</tr>
<tr>
<td>BiPap</td>
<td>Comparable efficacy to CPAP</td>
<td>Questionably more beneficial than CPAP in reducing symptoms and AHI; no data on relative improvements in morbidity and mortality</td>
</tr>
<tr>
<td>ASV</td>
<td>No clear advantage over CPAP</td>
<td>Favourably compares to CPAP in terms of markers of cardiac function as well as symptom reduction; no definitive data on improvement in morbidity and mortality</td>
</tr>
<tr>
<td>Nocturnal oxygen supplementation</td>
<td>Less effective than CPAP in reduction of symptoms or AHI (currently not recommended)</td>
<td>Some benefit reported in nocturnal desaturation and periodic breathing, but no proven benefit in cardiac function, morbidity, or mortality (currently not recommended)</td>
</tr>
<tr>
<td>Behavioural modifications, muscle training, surgery</td>
<td>Some benefit in decreasing AHI and symptoms, no proven improvement in cardiovascular morbidity or mortality</td>
<td>No proven or theoretical benefit</td>
</tr>
<tr>
<td>Phrenic nerve stimulation</td>
<td>No proven or theoretical benefit</td>
<td>Immediate decrease in apnoea frequency and nocturnal desaturations: long-term data lacking</td>
</tr>
</tbody>
</table>

increase the rate during apnoeas. In non-blinded studies, ASV improved markers of cardiac function (LVEF, NYHA class, NT-pro BNP, C-reactive protein) as well as quality of life, exercise capacity, and respiratory stability in HF patients with Cheyne-Stokes respiration, comparing favourably to CPAP.134–139 A study of ASV in people with complex sleep apnoea (HF patients who developed CSA while being treated with CPAP for OSA) showed similar benefits in terms of cardiac function and respiratory stability.140 Although long-term outcome data for ASV are currently lacking, trials are underway.

Since the use of nocturnal supplemental oxygen significantly reduces apnoea-related hypoxia in CSA,141,142 theoretically this might lead to long-term benefit. Studies to date have shown some benefit in terms of ameliorating nocturnal desaturations and periodic breathing, but no benefit in terms of cardiac function, morbidity, or mortality.143,144 Current guidelines do not recommend the use of oxygen therapy in sleep apnoea, as supplemental oxygen alone may prolong apnoeas and may potentially worsen nocturnal hypercapnia in patients with co-morbid respiratory disease.99

For patients unable to tolerate CPAP therapy, less effective options have shown some benefit, including behavioural treatments, dental appliances that prevent the jaw from sliding backwards, and surgery of the soft palate and nasoskeleton to open the posterior airway passage.99 Voice exercises, neck muscle strengthening manoeuvres, and even playing the didgeridoo (an indigenous Australian wind instrument) have been shown to improve the symptoms of moderate sleep apnoea.145 An exciting new modality in the pipeline is transvenous phrenic nerve electrostimulation to stimulate diaphragmatic motion in CSA.146 While clinical trials are still ongoing, preliminary data appear promising (Table 1).

Conclusion

Sleep apnoea is a common chronic disorder with potentially devastating consequences. It induces numerous physical and biochemical derangements. Sleep apnoea is pro-inflammatory, with nocturnal oxygen desaturations and hypercapnia appearing to play a pivotal role in this process. It is independently associated with an increased risk of death of any cause.147 Sleep apnoea even poses a societal risk since sufferers are prone to sleep-deprived driving accidents.

Diagnosis and management of sleep apnoea can be difficult. Symptoms of sleep apnoea are not easily indentified, and as many as 80% of sufferers remain undiagnosed.148 This diagnostic gap should begin to close as awareness rises of the high prevalence of sleep apnoea in many common diseases. High-risk patients for sleep apnoea include the obese, those with treatment refractory hypertension, type 2 diabetes, CAD, stroke, MIs, congestive HF, AF, nocturnal dysrhythmias, and PH.

Even the gold standards of diagnosis and treatment suffer from major drawbacks. Polysomnography is expensive and inconvenient, and successful CPAP treatment is often hindered by frequent intolerance and widespread non-compliance. Sleep apnoea is one of the most prevalent and dangerous cardiac risk factors, and future research will hopefully shed more light on the effects of this disease, as well as provide more effective options for management and treatment.

Conflict of interest: none declared.

References


