Is sleep apnoea syndrome a cardiovascular disease?

Le syndrome d’apnée du sommeil est-il une pathologie cardiovasculaire ?

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Sleep apnoea syndrome (SAS) has become a common disorder with a potential deleterious impact on the cardiovascular system. The prevalence varies from 4 to 14% depending on a number of factors including age (increased prevalence after the age of 50), sex (more common in men), body mass index (BMI; more prevalent in obese individuals), definitions (apnoea-hyperpnoea index per hour) and regions of the world (more common in North America than in Europe) [1,2]. Obstructive sleep apnoea (OSA) occurs more frequently in obese patients and is present in a large proportion of patients with hypertension, coronary artery disease, stroke and atrial fibrillation [3]. In contrast, central sleep apnoea (CSA) occurs mainly in patients with severe heart failure [4].

Repetitive apnoeas expose the cardiovascular system to cycles of hypoxia and exaggerate negative intrathoracic pressure and arousal. These deleterious stimuli can depress myocardial wall stress and parasympathetic activity, provoke oxidative stress and activate systemic inflammation, and impair vascular endothelial function [3]. Negative intrathoracic pressure increases right ventricular preload while apnoea-induced hypoxia causes pulmonary vasoconstriction, thus increasing right ventricular afterload. This could impede left ventricular filling and decrease stroke volume. Hypoxia during OSA might also directly impair cardiac contractility and diastolic relaxation [3].

In this issue, Paulino et al. [5] reported the high prevalence of SAS (80%) in a large cohort of patients with chronic heart failure (CHF) (n = 316). The authors emphasized the high prevalence of severe SAS (≥ 30/h) (41% of the patients with SAS). In line with the literature, patients with SAS were more frequently male, older, with a higher prevalence of atrial fibrillation, and a higher brain-natriuretic peptide (BNP) concentration.
and BMI compared to patients without SAS. However, left ventricular ejection fraction (LVEF) and functional class of heart failure did not differ significantly between the two groups. They also reported that in the SAS group, OSA was more frequent (70%) than CSA (30%). CSA was associated with lower LVEF, higher BNP concentration and prevalence of atrial fibrillation compared with OSA and non-SAS patients. It is of note that the prevalence of SAS in this French study is higher than in other studies (60 to 75%) despite using the same definition for SAS and severity of heart failure [6,7]. Again, the higher prevalence of OSA compared with CSA has not been reported before in CHF (usually a similar or a predominance of CSA is reported) [8]. This discrepancy could be due to the ability of beta-blockers and biventricular pacing to reduce the risk of CSA [6].

OSA has been identified as a possible independent risk factor for the development of heart and vascular disease, while CSA is rather a consequence of cardiovascular disease. Meanwhile, the pathophysiology of CSA has not been elucidated. Whether CSA is an epiphenomenon in CHF or could lead to an increased risk or progression of heart failure remains a matter of debate. However, it is well established that CSA is associated with a poor outcome in CHF [9,10].

Clinical and experimental studies argue for a close relation between sleep apnoea and cardiovascular disease [4,6]. Sleep apnoea could initiate cardiovascular disease and/or accelerate disease progression in patients with established cardiovascular disease. The clinical question is how to diagnose SAS in CHF patients and why? Patients with CHF should undergo dual management involving both cardiologists and physicians involved in sleep apnoea through a network or a specialized in- or outpatient clinic. SAS should be investigated in patients with CHF. Clinical trials evaluating the impact and consequences of SAS on cardiovascular disease are still to be performed [11,12]. Furthermore, we have to move into the era of treatment strategies for SAS (e.g., continuous positive airway pressure [CPAP] or positive airway pressure, mandibular advancement appliances or new drugs with a central nervous effect on sleep apnoea) and evaluate whether these treatments could improve cardiovascular disease by reducing cardiovascular morbidity and mortality. CPAP has already demonstrated acute and long-term benefits in terms of left ventricular function but with no long-term benefit on clinical outcome [13–17]. Finally, the effect of heart failure therapies on CSA (e.g., beta-blockers, renin angiotensin system blockers, cardiac resynchronization) should be also investigated.

References


