Obstructive sleep apnoea, intermittent hypoxia and heart failure with a preserved ejection fraction

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To the Editor

We have read the review article entitled 'Obstructive sleep apnoea, intermittent hypoxia and heart failure with a preserved ejection fraction' by John E Sanderson, Fang Fang, Mi Lu, Chen Yao Ma and Yong Xiang Wei, published in *Heart*, the official journal of the British Cardiovascular Society. Despite the authors' meritorious efforts in the present review, we believe some considerations may take part in an important complementary discussion. The review article has demonstrated that obstructive sleep apnoea (OSA) is likely to be a significant risk factor for heart failure with a preserved ejection fraction (HFpEF). Given this scenario, we think that it is important to stand out that for patients with HFpEF, the HR for OSA was comparable with the HRs for diabetes, coronary artery disease and blood pressure.[1] Sarkar *et al* claim that the intermittent hypoxia observed in OSA leads to oxidative stress, increased sympathetic activation, endothelial dysfunction, blood pressure surges, an increase in the levels of circulating inflammatory markers and hypercoagulability. Large negative intrathoracic swings generated by obstructed breathing efforts also place considerable mechanical stress on the heart and great vessels. Together, these changes create an environment that has the potential to increase the risk of cardiovascular disease, such as HFpEF.[2] The review observed that OSA may be a potential stimulant change in the extracellular matrix and in the development of myocardial fibrosis, and that early treatment of OSA may be particularly relevant for the prevention of HFpEF. Monocytes and granulocytes from patients with OSA have increased levels of reactive oxygen species production when compared with control subjects.[3] Besides, the endothelial cells taken from the veins of the forearm of patients with OSA show signs of increased inflammation and stress oxidation, which correlated with impaired endothelial function.[4] Therefore, there are similarities between the pathophysiology of OSA and the causes of HFpEF. All are associated with the activation of a wide range of inflammatory, metabolic, neural and haemodynamic changes that can affect cardiac function and should be clearly discussed in the context of those findings.

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