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EDITORIAL

Sleep apnoea and hypertension: time for recommendations

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Hypertension can be caused by obstructive sleep apnoea (OSA), as now recognised in international guidelines [1, 2]. The relationship between OSA and hypertension is well established, which has important consequences for the cardiovascular system [3, 4]. Many studies have shown an increase in the incidence and prevalence of hypertension in patients with OSA, independent of confounding factors, such as anthropometric parameters, alcohol consumption and smoking [5–8]. OSA is a recognised cause of hypertension that should be considered in any patient with high blood pressure, particularly in those with resistant hypertension [1, 2]. More than 50% of patients with OSA are hypertensive [7, 9] and more than 80% of patients with resistant hypertension also have OSA [10]. Hypertension related to OSA is predominantly nocturnal with a frequent non-dipper profile (which corresponds to a limited fall in nocturnal blood pressure) and an increase in diastolic blood pressure in the majority of patients [9, 11].

The mechanisms linking sleep apnoea and hypertension have been extensively reviewed [3, 4, 12]. There is increased sympathetic activity, which has been demonstrated in patients with OSA using sympathetic micro-neurography of the nerves supplying muscles, and also using plasma and urinary catecholamine assays. Potential mechanisms contributing to OSA-related hypertension include endothelial dysfunction leading to inhibition of nitric oxide production, decreased vasodilatation, and increased vasoconstriction; systemic inflammation, which favours endothelial dysfunction; oxidative stress, which results in the production of reactive oxygen species and causes vasoconstriction as a result of nitric oxide synthase blockade, increased generation of endothelin-1, and activation of angiotensin II; activation of the renin–angiotensin–aldosterone system, which increases plasma aldosterone levels; and metabolic anomalies leading to hyperinsulinism and resistance to the metabolic effects of leptin, the adipocyte-derived hormone. Activation of the endothelin system results in vasoconstriction and depressed baroreflexes. A genetic contribution to the association between OSA and hypertension might also exist, but there is currently only a limited amount of data available to support this possibility. Although not fully understood, the role of hypoxia in promoting an increase in blood pressure appears prominent, as evidenced both in animal models [13] and more recently in a model developed in normal volunteers [14, 15]. In this latter model, it is notable that intermittent hypoxia during the night did not produce an immediate increase in blood pressure, presumably due to vasodilation occurring in response to intermittent hypoxia counteracting the effects of sympathetic activation [15]. However, there was a sustained increase in sympathetic activity that seems to be responsible for the daytime increase in blood pressure observed in these subjects after only one night of intermittent hypoxia, which is more pronounced after 13 nights, and still tending to persist after 5 days of intermittent hypoxia exposure withdrawal [15]. Blood pressure lowering response to continuous positive airway pressure (CPAP) treatment appears to be dependent on sleep apnoea severity [16–18]. Whether sleepiness is important in predicting the CPAP-associated reduction in blood pressure is still debated [18–20], but probably unlikely [21]. In any case, the reduction in blood pressure obtained when treating OSA seems relatively limited [18, 21, 22] and far less than that previously reported in earlier studies [17], even when including only hypertensive patients [21, 22]. More recently, however, it has been clearly demonstrated that a reduction in blood pressure, as well as a reduced incidence of hypertension, cannot be achieved unless a minimum of 4–6 h of CPAP usage is achieved [8, 23].

In this issue of the *European Respiratory Journal*, PARATI *et al*. [24] report the work performed by a panel of experts participating in the European Union COST (Cooperation in Scientific and Technological research) Action B26 on OSA, with the endorsement of the European Respiratory Society (ERS) and the European Society of Hypertension (ESH). This group of experts has produced, between 2005 and 2010, a number of recommendations in the fields of sleep apnoea and cardiovascular disease [3, 25], management of OSA [26], and driving [27, 28]. Since OSA is now a recognised cause of hypertension, this group, which includes respiratory physicians, sleep experts and cardiologists, considered that there was a need to review the current knowledge and propose recommendations for clinicians and researchers. Importantly, this position paper is not only endorsed by the two scientific societies concerned, *i.e.* the ESH and ERS, but is also jointly published [24, 25].

The document is aimed at addressing the current state of the art in epidemiology, pathophysiology, diagnostic procedures and treatment options for optimum management of OSA in cardiovascular patients, as well as for the management of arterial hypertension in OSA patients. The recommendations have the objective of reminding cardiovascular experts to

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consider the occurrence of sleep-related breathing disorders in patients with high blood pressure, and also for sleep and breathing physicians to consider the occurrence of hypertension in patients with sleep-related breathing disorders. We believe that this cooperative work provides a highly valuable body of knowledge, which stresses the need to continuously update the current national and international guidelines on the relationships between these highly prevalent disorders.

STATEMENT OF INTEREST

None declared.

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