

Sleep Apnea and Hypertension

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Introduction

How many of us, when we are first taking a history from a hypertensive patient, ask about sleep habits and snoring? My guess is, not many. Sleep disordered breathing is not on the radar of most clinicians, and for good reasons. In the sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI) report,^[1] which is the standard guideline for the evaluation of hypertension in the United States, sleep apnea is not mentioned as a possible cause of hypertension, and most major textbooks of hypertension also pay little attention to it. Despite this, there is convincing evidence for an association and recent evidence that sleep apnea is a primary risk factor for the development of hypertension. We spend about one third of our lives asleep; we are all aware that the blood pressure (BP) falls in most people during sleep, and yet our mindset is that hypertension is a daytime disease. Numerous studies using 24-hour monitoring of BP have demonstrated that the nocturnal fall of BP is not a universal finding, and that variants of the normal dipping pattern may have pathologic consequences.

A wide spectrum of severity of sleep-disordered breathing has been recognized, with snoring at the benign extreme, and obstructive sleep apnea at the other. These conditions are relevant to the present discussion not only because they affect the changes of BP occurring during sleep, but also because they are significant risk factors for cardiovascular disease. The study of sleep apnea has largely been undertaken by pulmonologists, and as a result of the compartmentalization of contemporary medical science, their significance has not been fully appreciated by researchers or clinicians in the field of cardiovascular disease.

Obstructive sleep apnea is a disorder in which there are repeated episodes of partial (hypopnea) or complete (apnea) cessation of breathing during sleep. Such episodes may be of central origin, or due to mechanical obstruction of the airways, or to a combination of the two. By definition, apneas or hypopneas lasting at least 10 seconds are considered to be of clinical significance, although they usually last for 20-30 seconds and can last for more than a minute. The apnea/hypopnea index (AHI, also known as the Respiratory Distress Index, or RDI) is the average number of apneas and hypopneas per sleep hour.

Most apneic episodes are caused by collapse of the pharyngeal airway.^[2] This is the narrowest part of the airway, and the most dependent on muscle tone to maintain its patency. Muscle tone relaxes during sleep, and this may result in narrowing of the upper airway during inspiration, and hence snoring. This collapse of the airway also leads to increased respiratory effort and arousal. The arousal restores the muscle tone of the upper airways, allowing the subject to fall asleep once more. In this way the cycle of sleep and arousal may be repeated throughout the night. Not surprisingly, agents that relax skeletal muscle such as alcohol and benzodiazepines tend to exacerbate sleep apnea.