LEARNING THE ROPES: SLEEP DISORDERS AND CVD

Focus on Sleep Disorders in Cardiovascular Disease

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Sleep disorders are common in the general population, and are associated with obesity, the metabolic syndrome, hypertension, and with problems related to mental health. Furthermore, sleep disorders and hypertension share several risk factors, although their causal relations remain unclear. Poor sleep quality is associated with impaired quality of life, and an increased risk for coronary artery disease, stroke, and all-cause mortality¹. Obstructive sleep apnea is the most common form of sleep disordered breathing (which constitute of obstructive sleep apnea, central sleep apnea, and their combination named complex sleep apnea). There are several pathophysiological mechanisms by which obstructive sleep apnea could promote hypertension and cardiovascular disease, including autonomic alterations with sympatho-adrenal activation, endothelial dysfunction, coagulopathy, metabolic disturbances, increased inflammation, and increased oxidative stress [2-5]. Thus, it is not surprising that sleep disordered breathing is a common finding among people with cardiovascular disease, and may be a potential target for reducing cardiovascular risk. Also, studies show improvement in quality of life, and benefits in patients with hypertension and arrhythmias.

Given that sleep disordered breathing is common and may promote cardiovascular disease, this is a condition associated with considerable direct and indirect costs for health care. More important, however, studies suggest that diagnosis and treatment of obstructive sleep apnea is cost effective⁶. There are several useful and simple questionnaires to use for screening of obstructive sleep apnea (e.g. STOP-Bang, NoSAS)^{7,8}. However,

it appears that screening for sleep disorders in general, and sleep disordered breathing such as obstructive sleep apnea in particular, is seldom performed in clinical cardiology. This may be a missed opportunity for diagnosis, potential treatment, and cardiovascular risk reduction in many of the patients we see every day in our practice.

Learning the ropes in this issue of Hypertension News provides you we three short reviews on sleep disorders and cardiovascular risk, covering 1) the risk for cardiovascular disease, 2) the pathophysiology, and 3) the management of obstructive sleep apnea in patients with cardiovascular comorbidities. Focus on sleep disorders in cardiovascular disease is warranted.

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Sleep Disorders and Risk of CVD

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According to Thomas Dekker quote “Sleep is that golden chain that ties health and our bodies together”.

Insufficient and inadequate sleep quality is a growing health problem affecting more than one in three adults. Sleep disorders are critical contributors to obesity, diabetes, cardiovascular (CV) disease, heart failure (HF), stroke and depression. Sleep debt increases the risk of motor vehicle crashes, fatal injury, and disability each year.

Sleep can be disrupted by ‘sleep and circadian rhythm disorders’ (i.e., sleep apnoea, insomnia, narcolepsy, restless leg syndrome, parasomnias, sleep bruxism), work, social or lifestyle behaviours. Unhealthy sleep patterns defined as short (<7 hours) and long sleep (>8 hours), late chronotype, insomnia symptoms, snoring and excessive daytime sleepiness (EDS) have been associated with an increased risk of coronary heart disease (CHD), CV disease, stroke, and all-cause mortality. While patients with a healthy sleep score (Figure 1a) demonstrated a reduced risk of CV disease by 35%, CHD by 34% and stroke by 34% independently of genetic risk score¹, and HF² suggesting that treating sleep disorders should be considered for CV prevention.

Obstructive sleep apnoea (OSA) is the most common type of sleep-disordered breathing [3]. Nearly 1 billion of the worldwide adults’ population aged 30–69 years have OSA, and the number of people with moderate to severe OSA, for which treatment is generally recommended, is estimated 425 million⁴. Given the already high and increasing prevalence of obesity and aging, the number of people presenting with OSA symptoms continues to grow.

OSA is characterized by recurrent episodes of closure (apnoea) and hypopnea (narrowing) of the upper respiratory airflow resulting in shallow or paused breathing (Figure 1b), intermittent hypoxemia, and arousal associated with maintained or increasing ventilator efforts³. Apnoea is defined as at least 90% airflow limitation through the respiratory tract which is maintained for 10 seconds and associated with increased inspiratory efforts from chest and/or abdomen, whereas hypopnea refers to a drop in the airflow of $\geq 30\%$ for ≥ 10 seconds and is associated with a $\geq 3\%$ oxygen desaturation or arousal. Based on the index denoting average number of apnoeic and hypopneic episodes per hour of sleep (apnoea-hypopnea-index [AHI], OSA is classified as: mild AHI ≥ 5 and <15 events per hour, moderate AHI ≥ 15 and ≤ 30 events per hour, and severe AHI >30 events per hour.