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Children and Adults are at Risk for Cardiovascular Disease and Obstructive Sleep Apnoea

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Editorial

New research is highlighting the complex interrelationships between sleep-disordered breathing and cardiovascular disease, presenting therapeutic and scientific potential as well as challenges. Patients who enter cardiology clinics frequently have obstructive and central sleep apnea, as well as Cheyne-Stokes respiration. Sleep disturbances have been related to a number of issues that affect the development and function of the cardiovascular system. Epidemiological studies have connected obstructive sleep apnea to an increased risk of coronary heart disease, heart failure, stroke, and atrial fibrillation. Heart failure and atrial fibrillation are predicted by central sleep apnea with Cheyne-Stokes respiration, and death is substantially predicted in people with heart failure [1]. There is a strong evidence to include obstructive sleep apnea and central sleep apnea linked with Cheyne-Stokes respiration as potentially modifiable risk factors for cardiovascular disease.

Small studies show that treating obstructive sleep apnea with continuous positive airway pressure improves not only patient-reported outcomes such as sleepiness, quality of life, and mood, but also transitional cardiovascular end points such as blood pressure, cardiac ejection fraction, vascular parameters, and arrhythmias. According to evidence from large-scale randomised controlled research, positive pressure drugs do not appear to have a role in reducing cardiovascular mortality [2]. Although one study found that continuous positive airway stress improves quality of life, mood, and work absenteeism, the findings of two recent major randomised controlled trials published in 2015 and 2016 raise doubts regarding the efficacy of pressure therapy in reducing clinical end points. The provides context for interpreting recent study findings, key clinical messages, and recommendations for future sleep and cardiovascular research, including a greater focus on individual risk factors, the use of existing and new multimodality treatments that also address adhesion, and the use of trials with sufficient power to target end points and support subgroup analyses.

The most effective strategy to achieve these goals may be to strengthen collaboration across the cardiology, sleep medicine, and clinical trial fields. Sleep is a crucial regulator of cardiovascular function in both normal and diseased situations. Even in persons who don't have a serious sleep problem, sleep can alter the autonomic nervous system, systemic hemodynamics, cardiac function, endothelial function, and thrombosis. Some of these impacts are due to the usual circadian cycle of various physiological systems, while others are due to the specific modulatory effects of sleep stages. There is a relationship between physiological sleep and the development of vascular events, irregular heartbeats, and sudden death. According to epidemiological and neuropathological studies, primary sleep irregularities (sleep deprivation, shift work, and sleep-disordered breathing) may be linked to cardiovascular disease, including hypertension, atherosclerosis, stroke, heart failure, cardiac arrhythmias, sudden death, obesity, and the metabolic syndrome. Finally, sleep disturbances can occur as a result of a range of medical conditions (such as obesity, chronic heart failure, and menopause), potentially increasing cardiovascular morbidity [3]. More research into the specific pathophysiological processes that relate sleep disruptions to cardiovascular illness is needed to develop treatment approaches, and it could have major consequences for cardiovascular chronotherapeutics.

The cardiorespiratory system in newborns develops significantly after birth, and this growth is sleep-state dependent. It's no surprise that babies are prone to cardiorespiratory instability, especially during sleep, given the immaturity of these systems. The collapse of cardiovascular control mechanisms in particular is assumed to be the cause of the last event of Sudden Infant Death Syndrome (SIDS) [4]. SIDS is characterised as an occurrence that occurs when three overlapping variables collide: (1) a susceptible baby, (2) a critical developmental stage in homeostatic regulation, and (3) an exogenous stressor, according to the "triple risk hypothesis." This research highlights the link between inadequate cardiovascular control and the three overlapping characteristics implicated in SIDS pathogenesis, as well as the normal development of cardiovascular control in neonates during sleep. When sleep is insufficient or disrupted, a number of mental and physical disorders, including cardiovascular disease (CVD), emerge, increasing health-care costs. According to multiple observational studies and meta-analyses, insomnia, short (7h) or extended (>9h) sleep, and other sleep disorders are connected to an increased risk of hypertension, metabolic syndrome, infarction, heart issues, arrhythmia, CV disease risk, and/or death.

Inflammatory, immunological, neuro-autonomic, endocrinological, genetic, and microbiome changes may all play a part in how insomnia and other sleep disorders increase the risk of cardiovascular disease. Guidelines are being developed that indicate that all people over the age of 18 get at least 7 hours of sleep per night for optimal CV health. Benzodiazepine receptor agonists binding to gamma aminobutyric acid type-A (benzodiazepine and non-benzodiazepine medications) and antidepressants are used to treat sleep disorders, with cognitive-behavioral therapy being the basis of non-pharmacologic treatment for persistent insomnia [5]. Anxiolytics and hypnotics, on the other hand, appear to increase mortality risk in observational studies and meta-analyses; nevertheless, bias may exist due to confounding and high heterogeneity in these investigations. Non-benzodiazepine hypnotic medicines (Z pharmaceuticals) appear to pose a lower risk than anxiolytics, with evidence suggesting that at least one of these compounds, zolpidem, may even pose a lower risk.

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