Risk of cardiovascular diseases and obstructive sleep apnea in children and adults

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COMMENTARY

The complicated interrelationships between sleep-disordered breathing and cardiovascular illness are being highlighted in new research, bringing therapeutic and scientific opportunities as well as obstacles. Obstructive and central sleep apnea, as well as Cheyne-Stokes respiration, is common in patients who visit cardiology clinics. Sleep disruptions have been linked to a variety of factors that influence cardiovascular structure and function. Obstructive sleep apnea has been linked to an increased risk of coronary heart disease, heart failure, stroke, and atrial fibrillation, according to epidemiological studies. Central sleep apnea with Cheyne-Stokes respiration predicts heart failure and atrial fibrillation, and it highly predicts death in individuals with heart failure. Thus, a strong literature provides the mechanistic and empirical bases for considering obstructive sleep apnea and central sleep apnea associated with Cheyne-Stokes respiration as potentially modifiable risk factors for cardiovascular disease. Small trials show that using continuous positive airway pressure to treat obstructive sleep apnea improves not only patient-reported outcomes like sleepiness, quality of life, and mood, but also transitional cardiovascular end points like blood pressure, cardiac ejection fraction, vascular parameters, and arrhythmias.

Positive pressure medicines, on the other hand, do not appear to have a function in reducing cardiovascular mortality, according to findings from large-scale randomised controlled studies. Although one trial supported the beneficial impact of continuous positive airway stress on quality of life, mood, and work absences, the results of two recent large randomised controlled trials, published in 2015 and 2016, raise questions about the effectiveness of pressure therapies in reducing clinical end points. The provides a context for interpreting the findings of recent studies, 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 implementation of trials with sufficient power to target end points and support subgroup analyses. Strengthening collaboration across the cardiology, sleep medicine, and clinical trial communities may be the most effective way to achieve these objectives. In both normal and pathological states, sleep is a key regulator of cardiovascular function. Sleep can affect the autonomic nervous system, systemic hemodynamics, cardiac function, endothelial function, and thrombosis in people who don't have a major sleep issue.

Some of these effects are caused by the normal circadian cycle of various physiological processes, while others are caused by particular modulatory effects of sleep stages per se. There is a link between the occurrence of vascular events, irregular heartbeats, and sudden death with physiological sleep. Primary sleep irregularities (sleep deprivation, shift work, and sleep-disordered respiration) may be linked to cardiovascular disease, including hypertension, atherosclerosis, stroke, heart failure, cardiac arrhythmias, sudden death, obesity, and the metabolic syndrome, according to epidemiological and neuropathological studies. Finally, sleep abnormalities can arise as a result of a variety of medical disorders (including obesity, chronic heart failure, and menopause), potentially contributing to cardiovascular morbidity.

Further research into the specific pathophysiological processes that link sleep disturbances to cardiovascular disease is critical for establishing therapeutic techniques, and it could have significant implications for cardiovascular chronotherapeutics. After birth, the cardiorespiratory system in babies undergoes significant functional development, which is sleep-state dependent. Given the immaturity of these systems, it's no surprise that newborns are susceptible to cardiorespiratory instability, particularly during sleep. The last event of Sudden Infant Death Syndrome is thought to be caused by a failure of cardiovascular control mechanisms in particular (SIDS). SIDS is defined by the "triple risk model" as an occurrence that occurs when three overlapping elements collide: (1) a vulnerable newborn, (2) a critical developmental stage in homeostatic regulation, and (3) an exogenous stressor. This paper summarises the normal development of cardiovascular control in newborns during sleep and describes the link between impaired cardiovascular control and the three overlapping variables implicated in SIDS pathogenesis. When sleep is inadequate or disrupted, a variety of mental and physical diseases develop, including cardiovascular (CV) illness, which raises health-care expenses. Insomnia, short (7h) or extended (>9h) sleep, and other sleep disorders are linked to an elevated risk of hypertension, metabolic syndrome, infarction, heart problems, arrhythmia, CV disease risk, and/or death, according to several observational studies and meta-analyses. Inflammatory, immunological, neuro-autonomic, endocrinological, genetic, and microbiome disturbances may all play a role in how insomnia and other sleep disorders enhance CV risk.

For optimal CV health, guidelines are developing that recommend that all persons over the age of 18 obtain at least 7 hours of sleep. Sleep disorders are treated with benzodiazepine receptor agonists binding to gamma aminobutyric acid type-A (benzodiazepine and non-benzodiazepine medicines) and antidepressants, with cognitive-behavioral therapy being the mainstay of non-pharmacologic care of persistent insomnia. However, observational studies and meta-analyses show that anxiolytics and hypnotics increase mortality risk; however bias may be present due to confounding and significant heterogeneity in these researches. However, it appears that the danger posed by non-benzodiazepine hypnotic medicines (Z medications) is lower than that posed by anxiolytics, with evidence suggesting that at least one of these agents, zolpidem, may even pose a lower risk.

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