Editorial

EFFECTS OF SLEEP DEPRIVATION ON CARDIOVASCULAR SYSTEM

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Sleeping is a basic human need, like eating, drinking, and breathing. Sleep is a vital part of the foundation for good health and well-being. It plays a vital role in a person's mental, emotional, and physiological health. People who don't sleep enough are at higher risk for cardiovascular disease and coronary heart disease regardless of age, weight, smoking and exercise habits. According to a recent statement from the American Heart Association, an irregular sleep pattern is linked to a host of cardiovascular risks, including obesity, high blood pressure, diabetes, and coronary artery disease and stroke. A 2011 European Heart Journal review of 15 medical studies involving almost 475,000 people found that short sleepers had a 48% increased risk of developing or dying from coronary heart disease (CHD) in a seven to 25-year follow-up period (depending on the study) and a 15% greater risk of developing or dying from stroke during this same time. Interestingly, long sleepers -- those who averaged nine or more hours a night also showed a 38% increased risk of developing or dying from CHD and a 65% increased risk of stroke. This article summarizes findings of known effects of insufficient sleep on cardiovascular risk factors including blood pressure, glucose metabolism, hormonal regulation and inflammation.

WHAT IS SLEEP DEFICIENCY/DEPRIVATION?

Sleep deficiency is a broader concept. It occurs if you have one or more of the following

- You don't get enough sleep (sleep deprivation) i.e. sleeping less than 6 hours a night.
- You sleep at the wrong time of day (you're out of sync with your body's natural clock/circadian rhythm)
- You have a sleep disorder that prevents you from getting enough sleep or causes poor quality sleep i.e. Restless Leg Syndrome, sleep apnea syndrome (30 or more than 30 awakenings during night sleep) and Narcolepsy etc.

There are four specific variations of sleep deficiency: insomnia, acute total sleep deprivation (TSD), partial sleep deprivation (PSD), and night shift workers where Acute TSD meaning lack of sleep for a period of at least one night, PSD meaning reduction in the total sleep time relative to one's usual baseline during a 24-hour period, Insomnia meaning dissatisfaction with sleep quantity or quality, associated with one or more of the following symptoms: difficulty initiating sleep, difficulty maintaining sleep characterized by frequent awakenings or problems returning to sleep after awakenings, or early morning awakenings with inability to return to sleep and a Shift Worker referring to a person who follows a work schedule that is outside the typical "9 to 5" business day. Millions of people are considered shift workers,

including doctors and nurses, pilots, bridge builders, police officers, customer service representatives, and commercial drivers.

Effect of Sleep deprivation on Sympathetic activity, Endothelial Function and atherosclerotic plaque initiation and progression:

Endothelial cells elicit relaxation and contractions of the underlying vascular smooth muscle cells releasing vasoactive substances. Among those substances, nitric oxide (NO) plays a key role[1]. When an imbalance of the actions of the endothelium toward reduced vasodilation and increased vasoconstriction as well as increased prothrombotic properties occurs, it is said that endothelial dysfunction is present. Arterial endothelial dysfunction is an important event central to the pathogenesis of atherosclerosis. Continued endothelial dysfunction contributes to plaque initiation and progression [2].

Effect of Sleep Deprivation on Sympathetic activity:

Short sleep duration is associated with increased markers of autonomic tone that indicate lower levels of cardiac parasympathetic (vagal) tone and/or higher levels of sympathetic tone[3]. Several factors lead to sympathetic overactivity including autonomic system dysregulation and increased catecholamine levels in blood.

Effect of Sleep Deprivation on Blood Pressure:

BP should be decreased during sleep normally which is called Normal dipping (BP fall of more than 10% of the day time BP at night).Several studies have found that experimental sleep deprivation leads to increased blood pressure [4,5,6].Blood pressure is physiologically regulated via several mechanisms. Renal fluid filtration and reabsorption regulate blood volume and are under hormonal control, most importantly the renin-angiotensin system. Cardiac contractility (the force of cardiac ejection of blood into the systemic circulation), cardiac output (blood volume pumped in liters per minute) and peripheral vascular resistance are the other major determinants of measured blood pressure. These are under autonomic nervous control and linked to blood pressure via a feedback loop termed the baroreflex. The baroreflex involves a series of receptors, located in the heart itself as well as in the carotid artery and aortic arch, which sense blood pressure and relay information to the nucleus tractus solitarius in the medulla. If blood pressure needs to be adjusted, sympathetic or parasympathetic output then can influence cardiac contractility, heart rate and peripheral vascular resistance.BP could be increased during sleep deprivation due to increased sympathetic outflow to the heart or periphery, due to changes in baroreflex sensitivity, due to baroreflex resetting to a higher level, or a combination of these factors.

Endocrine/metabolic changes associated with sleep loss (Diabetes, Obesity):

During sleep deprivation, the sleep-associated Growth hormone pulse is substantially dampened or abolished and cortisol levels increase.Glucose metabolism is slowed during sleep deprivation [7]. Thus, insufficient sleep may be a potentially important contributing mechanism in the clinical development of insulin resistance, increased accrual of adipocytes and resulting elevated inflammatory mediators. TSH, T_3 and T_4 levels also increase during sleep deprivation[8,9].The small TSH changes described during sleep deprivation, if chronic, may contribute to the development of metabolic changes.Recent studies have shown that dysregulation/decrease of melatonin secretion in sleep deprivation is associated with increased sympathetic tone and insulin resistance leading to HTN and DM respectively.[10]

Sleep loss, inflammation/Oxidative stress and cardiovascular disease:

Monocytes and neutrophils, phagocytic cells in peripheral circulation, are elevated during acute sleep deprivation [11]. IL-6 is also produced by adipocytes and by endothelial cells. IL-6 and CRP have both been found to increase during acute total sleep deprivation [12]. IL-6 is a potent stimulator of CRP production.CRP is an independent predictor of a first cardiovascular event in asymptomatic individuals and is furthermore associated with adversity of that event [13].One of the explanations for why inflammatory mediators are elevated in cardiovascular disease is that the increased blood pressure increases endothelial shear stresses, resulting in endothelial production of inflammatory mediators. As well, autonomic system activation may contribute to elevated inflammation via multiple pathways. Catecholamine elevation is also associated with increased inflammatory mediators, and in the in vivo model, norepinephrine can stimulate production of inflammatory mediators including IL-6 and TNF-alpha.Hypoxia is a hallmark of the sleep apnea syndrome and is strongly associated with elevations in inflammatory mediators.

So how much sleep is adequate?

Both the quantity and quality of sleep matter. For 'optimal health' the American Academy of Sleep

Medicine recommends seven hours of sleep per night for adults. While the American Heart Association suggests seven or eight is ideal. Meanwhile a study presented at the 2018 European Society of Cardiology conference suggested that between six and eight hours of sleep a night is the ideal for heart health.

So, seven to eight hours sleep is likely a good target.

CONCLUSION:

The recent studies confirm that sleep deprivation is associated with HTN, IHD, and DM. Increased sympathetic nervous system activity and endothelial dysfunction are considered to serve as a common pathophysiology in sleep deprivation's relationships with these diseases. Especially, the relationship between sleep time and incidence of CHD or DM is U-shaped. Sleep periods that are neither too short nor too long may be important to keep us healthy.

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