Sleep Architecture and Blood Pressure

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KEYWORDS

• Sleep physiology • Cardiovascular physiology • Cardiorespiratory coupling • Arousal • REM • Non-REM

KEY POINTS

- Heart rate and blood pressure have a circadian rhythm characterized by a significant reduction during nighttime hours.
- During non–rapid eye movement sleep, there is an increase in parasympathetic drive and a reduction in cardiac sympathetic activity.
- By contrast, rapid eye movement sleep is a state of autonomic instability, dominated by remarkable fluctuations between parasympathetic and sympathetic influences.
- Any changes in sleep quality, associated with persistence of high sympathetic activity and reduction in physiologic nocturnal blood pressure dipping, results in increased blood pressure during the following days.
- Multiple cardiovascular conditions appear to have sleep-related disturbed autonomic regulation as their basis.

INTRODUCTION

Physiologic regulation during sleep varies with the state of the brain and is influenced by different stages of sleep. On the other hand, control of circulation during sleep requires coordination of the respiratory and the cardiovascular system. All these physiologic systems need to continuously interact with each other and at the same time each one has its own regulatory mechanisms, which adds more complexity to the sleep physiology. In normal circumstances, the heart rate (HR) is constantly being adjusted within each respiratory cycle and depends on the breathing frequency, known as respiratory sinus arrhythmia.[1] To date, little is known about the neural mechanisms of sleep-specific central commands. It is known that different sleep stages modulate regional blood flow at all levels of the nervous system. The strong relation between blood flow and metabolism in the central nervous system indicates that sleep modulates the activity of multiple pathways potentially involved in central autonomic control.[2] As sleep typically involves disengagement from the environment, blood pressure (BP) decreases at night. However, a variety of changes in brain activity during sleep have an effect on physiologic regulation beyond what occurs during wakefulness,[3] so these changes are not just because of disengagement from external environment.

The neural circulatory regulation appears to be coupled with the circadian rhythm; the sleep-wake cycle, including rapid eye movement (REM) and non–rapid eye movement sleep (NREMS) processes, is implicated in long-term BP regulation. The transition from wakefulness to NREMS is characterized by a relative autonomic stability

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and increase in parasympathetic drive and a reduction in sympathetic activity. As a consequence, HR, arterial BP, cardiac output, and stroke volume decrease, resulting in an overall decreased cardiac workload. Compared with a subject lying supine during wakefulness,[4–6] BP and cardiac output decrease during sleep. During REM sleep, electroencephalographic (EEG) patterns resemble that of wakefulness, but there is marked muscle atonia and intermittent REM. Not only is the EEG pattern similar to quiet wakefulness but also HR, BP, and sympathetic activity increase to levels that are present during relaxed wakefulness.[4,6,7] This phase is being repeated at 90-minute intervals and exhibits a more irregular pattern, with periodic surges in rather and arterial BP. These changes during REM bring more challenges to homeostatic regulation, especially in cardiovascular or pulmonary diseases. REM sleep also diminishes forebrain influence on brainstem, which has an effect on respiratory compensatory mechanisms that help BP management.

In this article, the author reviews the physiologic changes during different stages of sleep and discusses how these systems interact under normal circumstances.

SLEEP AND CARDIOVASCULAR INTERACTION

Changes in Circulatory Control

Under normal circumstances, HR and BP decrease during nighttime. The decrease in BP is about 10% or greater compared with daytime arterial BP, which is commonly known as "dipping." In addition, supine position and inactivity also contribute to a reduction in the double product noted above.[8]

Previous studies showed that measures of parasympathetic activity, such as RR interval (the time elapsed between 2 successive R waves of the QRS signal on the electrocardiogram), change as early as 2 hours before sleep onset,[9] whereas measures of sympathetic function such as catecholamines and pre-ejection period (the time elapsed between the electrical depolarization of the left ventricle and the beginning of ventricular ejection) decrease with the progression of sleep.[6,9,10]

Awakening has its own fascinating process. It induces a step-by-step activation of the sympathetic and adrenal system, which results in increased HR, BP, and plasma catecholamines. These changes during awakening, of course, will be perpetuated by postural changes and physical activity.[9,11]

On the other hand, the circadian rhythm may also play an important role in cardiovascular activity. Studies investigating the role of circadian rhythm showed subjects who were sleep deprived for 24 hours while in the supine position still had the nocturnal decrease in HR, whereas the decrease in nocturnal BP was blunted.[9,12] These changes as mentioned above suggest that parasympathetic activity (eg, HR) is mostly under circadian influences, whereas sympathetic mechanisms are largely related to the wake-sleep cycle. There is growing evidence showing that nocturnal BP is a key predictor of cardiovascular mortality regardless of the daytime BP levels.[12] Therefore, any changes in sleep can result in nocturnal BP increase and development of hypertension.

Cardiovascular Response During Non-Rapid Eye Movement Sleep

NREMS is characterized by autonomic stability due to a high degree of parasympathetic activity and a decrease in sympathetic tone, which results in a decrease in cardiac workload.[6] It has been shown that during relaxed wakefulness BP decreases progressively, but is interrupted during stage N1 of NREMS.[13] Further reductions in BP after sleep onset have been observed during stage N2[13] and deeper stages of NREMS.[14,15] The lowest levels of arterial BP are reached in stage N3 (formerly known as stages III and IV). This decrease in arterial pressure is primarily related to reduction in HR and sympathetic vasomotor tone.[16] However, the reported differences in BP between the early and late stages of NREMS have often been negligible in human subjects.[6,8,17] During NREMS, arousals increase BP variability,[18,19] which is comparable to average nightly decline in systolic pressure in normotensive individuals.[20] The K-complexes, which are spontaneous or stimulus-evoked phasic bursts activity, are also followed by slight increases in BP during NREMS.[6,14,15] It is of note that, despite these changes, the total variability of BP is less in NREMS than in wakefulness,[8,17] which is an expected event, as the variability of local metabolic needs is decreased, given that behavioral engagement with the environment in NREMS compared with the wakefulness is limited. The decrease in BP during NREMS may also be due to a reduction in HR and, hence, in cardiac output, without significant changes in stroke volume or total peripheral conductance.[21] Change in cardiac output or vascular conductance does not follow the operating logic of the arterial baroreflex during wakefulness. Some studies have shown that during NREMS the arterial baroreflex undergoes either a change in sensitivity or a resetting with respect to wakefulness,[6] and NREMS does not show a