13. HEART RATE (ARRHYTHMIAS) IN OBSTRUCTIVE SLEEP APNEA/HYPOPNEA SYNDROME

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Introduction. Normal sleep provides an advantageous physiological recovery period for the cardiovascular system (CVS). During non-rapid eye movement sleep (80%), sympathetic activity decreases and parasympathetic activity increases, resulting in a decrease in blood pressure (BP) and heart rate (HR). The study aims to determine the pathophysiological mechanisms of obstructive sleep apnea/hypopnea syndrome (OSAHS) that generate heart rate modifications and even arrhythmias.

Aim of study. OSAHS induces a cyclic variation in FCC with bradycardia during the episode of sleep apnea, followed by tachycardia caused by post-apneic hyperventilation, which presents an underestimated clinical problem with detrimental consequences.

Methods and materials. Performing literature review search from 2010-2023 using Google Scholar, PubMed and Medline databases using the keywords: “obstructive sleep apnea”, “sinoatrial node dysfunction”, “brady-tachy syndrome”.

Results. The pathophysiology of OSAHS is characterized by recurrent episodes of partial or complete collapse of the upper airways during sleep, with a reduction in airflow or its complete cessation. Inspiratory efforts at the beginning of obstructive apnea will generate negative intrathoracic pressure, which will cause an increase in the vagal tone. At the same time, due to the decrease in HR and the stroke volume of the left ventricle, cardiac output and systemic arterial pressure will decrease, respectively. The reduction of adequate alveolar ventilation leads to the development of hypoxemia and hypercapnia, which in turn will trigger respiratory arousal (post-apneic hyperventilation will occur) and autonomic arousal (the sympathetic tone will increase), which will cause the triggering of tachycardia and the increase of BP values. These changes in the cardiac autonomic nervous system activity during OSAHS cause hemodynamically and electrophysiologically increased susceptibility to developing sinus node dysfunction, complete AV block, or atrial fibrillation.

Conclusion. High-quality sleep is essential in maintaining cardiovascular health, providing a period of physiological recovery in which sympathetic activity decreases and parasympathetic activity increases, contributing to the optimal regulation of blood pressure and FCC. Pathophysiological mechanisms of OSAHS, characterized by repeated upper airway collapse and negative intrathoracic pressure, initially cause an increase in vagal tone. However, reduced alveolar ventilation develops hypoxia and hypercapnia, triggering autonomic and respiratory awakening by enhancing sympathetic tone. These changes in the autonomic activity of the cardiac nervous system in OSAHS predispose to the development of sinus node dysfunction, complete AV block, or atrial fibrillation.