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PHYSIOLOGICAL MECHANISM OF RESPIRATORY SINUS ARRHYTHMIA

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Oscillation of heart rate (HR) during breathing cycle defined as respiratory sinus arrhythmia (RSA) which is in normal physiological conditions serves as an index of healthy heart. Respiratory sinus arrhythmia determines cardiac vagal tone and characterizes patterns of autonomic modulation of cardiovascular control.

Modulation of vagal tone is caused by both central and peripheral mechanisms. It is known that during inspiration there is an increase in HR as a result of inhibitory effect to vagal activity caused by respiratory center of brainstem. Recently some researchers showed the role of subparabrachial nucleus, also called Kolliker-Fuse nucleus, which is located between midbrain and pons that is active during postinspiration and critical for phasic activity of cardiac vagal neurons.

The peripheral mechanism is initiated by pulmonary stretch receptors during inspiration as volume of lungs increases causing systemic venous return to right atrium to increase that activates atrial stretch receptors which results in tachycardia and vasodilation according to Bainbridge reflex. Simultaneously due to increased right atrial venous return, the venous return to left atrium decreases thus reducing stroke volume from left ventricle. As a result carotid sinus and aortic arch baroreceptors activity is decreased leading to tachycardia during inspiration. Additionally to central and peripheral control there is intrinsic mechanism caused by direct stretching of the sinoatrial node during filling of the heart that leads to increase in HR.

Currently several mechanisms are known generating respiratory sinus arrhythmia but which of them is more important is still debated question. However, the understanding the mechanism of HR oscillations caused by breathing could help with prevention of diseases development, diagnosis, treatment and prognosis of cardiovascular and respiratory pathology.