Comment

In physiology, autonomic nervous system (ANS) is instrumental mainly in short-term regulation of cardiovascular homeostasis. Its parasympathetic and sympathetic (SNS) components are regarded as two opposing forces with the parasympathetic nervous system endorsing "rest and digest" while the SNS "flight and fight". For instance, common stressors like sudden postural change, physical exercise or hemorrhage result in momentary drop in cardiac output and arterial pressure resulting in reflex activation of SNS (within seconds), and the renin-angiotensin-aldosteron system and the vasopresinergic system (within minutes). This acute neurohormonal activation is aimed at maintaining cardiovascular homeostasis (in particular of central arterial pressure and brain perfusion) by mediating functional changes in the heart, vessels, respiratory system and kidneys. These acute regulatory mechanisms result in an increase in mechanical and metabolic load to the heart and in a partial loss of its regulatory reserve. Nevertheless they are usually short-lasting and, as such, remain without major distant functional consequences. In contrast, chronic neurohormonal activation results in several adverse consequences, including systemic oxidative stress, inflammation, and altered gene expression resulting in various structural alterations. These occurring in cardiovascular system are seen as myocardial and vascular hypertrophy and remodeling.

Evidence indicates that chronic activation of SNS (and usually also of the renin-angiotensin-aldosteron system) is a common pathogenetic denominator of various cardiovascular diseases (e.g. heart failure, essential hypertension, metabolic syndrome). This is evidenced by an increased production of the neurohormons and/or the increased sympathetic nerves activity observed those states, and also by the fact that β-blockers are medicine of choice in many cardiological diseases.

Articles presented in the current issue of the "Progress in Medicine" review the mechanisms of the chronic SNS overactivity in the cardiovascular diseases, clinically useful methods of the ANS assessment, nonpharmacological methods of the therapeutic modulation of the ANS imbalance, and also the role of ANS disregulation in the mechanism of heart failure and cardiac arrythmias. In addition, presented are reviews on several "hot topics" in cardiovascular medicine, including physiologic cardiomyocyte necrosis and regeneration, gut bacteria origin of obesity and atherosclerosis and others.

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