Role of the carotid body and arterial chemoreflex in the pathophysiology of cardiovascular diseases – review of the current research

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The arterial chemoreflex originates from the carotid and aortic bodies and is triggered by hypoxia, increase in carbon dioxide concentration, acidity and hypoperfusion. Its activation leads to sympathoexcitation, as well as blood pressure and ventilation increase. Several lines of evidence indicate that the arterial chemoreflex is augmented and tonically active in hypertension, heart failure (HF) and cardiac arrhythmia. In the present review, we discuss results of recent clinical and preclinical studies, which point to the role of arterial chemoreflex in the cardiovascular pathophysiology. Specifically, a growing body of evidence shows that hyperactivity of the chemoreflex is involved in inducing central sympathetic drive in hypertensive animals and that resection or denervation of the carotid bodies (CBs) results in a decrease in blood pressure.

Furthermore, the reflex is also augmented in hypertensive humans and resection or inhibition of CBs normalizes their arterial blood pressure. Thus, the enhanced arterial chemoreflex is one of the mechanisms responsible for the elevated blood pressure and increase in peripheral resistance. Moreover, in animals with HF, tonic activation of the arterial chemoreflex is caused by hypoperfusion of the CBs due to decreased cardiac output.

This leads to the increase in systemic and renal sympathetic nerves activity. Chemoreflex-induced sympathoexcitation may further contribute to even faster progression of HF.

In light of these findings, limited number of studies show that inhibition or resection of the CBs in humans with HF normalizes sympatho-vagal balance. Finally, some recent findings show that stimulation of the arterial chemoreflex may contribute to cardiac arrhythmia and that inhibition of the reflex may limit the episodes of ventricular tachycardia.

In summary, recent research and progress in understanding the role of the arterial chemoreflex in the pathophysiology of cardiovascular diseases may result in new therapeutic options available in the future both as pharmacological and surgical treatments.