Carotid Body Ablation: a New Target to Address Central Autonomic Dysfunction

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Abstract

PURPOSE OF REVIEW:

An abnormal heightened carotid body (CB) chemoreflex, which produces autonomic dysfunction and sympathetic overactivation, is the common hallmark of obstructive sleep apnea (OSA), resistant hypertension, systolic heart failure (HF), and cardiometabolic diseases. Accordingly, it has been proposed that the elimination of the CB chemosensory input to the brainstem may reduce the autonomic and cardiorespiratory alterations in sympathetic-associated diseases in humans.

RECENT FINDINGS:

A growing body of evidence obtained in preclinical animal models support that an enhanced CB discharge produces sympathetic hyperactivity, baroreflex sensitivity and heart rate variability impairment, breathing instability, hypertension, and insulin resistance. The elimination CB chemosensory input reduces the sympathetic hyperactivity, the elevated arterial blood pressure in OSA and hypertensive models, abolishes breathing instability and improves animal survival in HF models, and restores insulin tolerance in metabolic models. These results highlight the role played by the enhanced CB drive in the progression of sympathetic-related diseases and support the proposal that the surgical ablation of the CB is useful to restore the autonomic balance and normal cardiorespiratory function in humans. Accordingly, the CB ablation has been used in pilot human studies as a therapeutic treatment for resistant hypertension and HF-induced sympathetic hyperactivity. In this review, I will discuss the supporting evidence for a crucial contribution of the CB in the central autonomic dysfunction and the pros and cons of the CB ablation as a therapy to revert autonomic overactivation. The CB ablation could be a useful method to reverse the enhanced chemoreflex in HF and severe hypertension, but caution is required before extensive use of bilateral CB ablation, which abolished ventilatory responses to hypoxia and may impair baroreceptor function.

KEYWORDS:

Autonomic dysfunction; Carotid body ablation; Heart failure; Intermittent hypoxia; Metabolic disease; Neurogenic hypertension; Obstructive sleep apnea