Role of the Carotid Body in Obesity-Related Sympathoactivation

To the Editor:

Paton et al. reviewed the experimental and clinical data reporting that stimulation of the carotid body drives sympathetic tone, and that increased chemosensitivity may be one of the factors underlying autonomic imbalance in hypertension, heart failure, insulin resistance, sleep disorders, and chronic kidney disorders. The authors proposed bilateral surgical removal of the carotid body for therapeutic reduction of systemic sympathetic hyperactivity, with potential improvement of morbidity and mortality.

The proposal is intriguing and stimulates further considerations about the potential role of the carotid body in the increased sympathetic tone of obesity. This condition is frequently associated with obstructive sleep apnea syndrome, characterized by chronic intermittent hypoxia caused by frequent collapses of the pharyngeal musculature during sleep. Chronic intermittent hypoxia increases the chemosensory response of the carotid body, stimulates sympathetic tone, and produces hypertension.1,2

A direct role of leptin also may be hypothesized, on the basis of recent findings. Leptin is synthesized in proportion to body fat mass and stimulates sympathetic nerve activity by means of various mechanisms. Many central structures regulating sympathetic tone (solitary tract nucleus, central amygdala, hypothalamic nuclei, parabranchial nucleus) express leptin receptors and may be activated by this hormone. However, leptin and leptin receptors also have recently been identified in human and rat glomus cells,3 and intravenous injections of leptin have been reported to increase Fos, Fra-1/2, and extracellular signal–regulated kinase 1/2 immunoreactivity in glomus cells, indicating cell activation.4,5 Immunoreactivity for the long form of the leptin receptor has also been found in vagal nerve and carotid sinus nerve fibers, together with afferent neurons of the petrosal and nodose ganglia. Increased expression of Fos and Fra-1/2 has also been observed in these neurons in response to leptin infusion.4 Thus, in patients who are obese with high plasma levels of leptin, increased activation of the peripheral chemoreceptor reflex pathway may be hypothesized, together with a contributive role in mediating sympathoactivation. Hyperleptinemia and chronic intermittent hypoxia may be inter-related mechanisms of sympathoactivation through peripheral chemoreceptors because chronic intermittent hypoxia increases plasma leptin levels and leptin immunoreactivity in the carotid body.4,5

In conclusion, the carotid body may potentially participate in sympathoactivation of obesity through increased chemosensitivity because of chronic intermittent hypoxia or the direct action of leptin. Thus, the carotid body also may be viewed as a therapeutic target for obesity-related sympathoactivation, although further experimental and clinical data are certainly needed.

Disclosures

None.

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