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 nuclei) 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 sympahtoactivation. 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.

Andrea Porzionato
Veronica Macchi
Raffaele De Caro
Section of Anatomy
Department of Molecular Medicine
University of Padova, Italy

Role of the Carotid Body in Obesity-Related Sympathoactivation
Andrea Porzionato, Veronica Macchi and Raffaele De Caro

Hypertension. 2013;61:e57: originally published online May 6, 2013;
doi: 10.1161/HYPERTENSIONAHA.113.01248
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2013 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://hyper.ahajournals.org/content/61/6/e57

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published
in Hypertension can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial
Office. Once the online version of the published article for which permission is being requested is located,
click Request Permissions in the middle column of the Web page under Services. Further information about
this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Hypertension is online at:
http://hyper.ahajournals.org/subscriptions/