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REVERSIBLE RIGHT VAGAL NEUROPATHY

Vagus nerve efferent fibers carry the parasympathetic innervation to the heart. The right and left vagal nerve innervation to the heart is believed to be asymmetric, with the right vagal nerve providing most of the parasympathetic innervation to the atria and sinoatrial (SA) node. The left vagal nerve carries parasympathetic innervation to the ventricles. This anatomic and physiologic asymmetry has been studied in animals¹ but not as extensively in humans. Cardiovascular parasympathetic function can be assessed noninvasively by measuring the heart rate variability in response to deep breathing (heart rate deep breathing range [HR_{DB}]) or heart rate changes during the Valsalva maneuver (Valsalva ratio [VR]).²

We describe a case of transient right vagal dysfunction and corresponding changes in the cardiovagal function in a patient with a right jugular foramen schwannoma. This report provides additional physiologic data to support the notion that cardiovagal innervation of the SA node comes from right vagus nerve.

Case report. A 37-year-old man presented with a history of 3 episodes of syncope over the past 10 years. Results of an extensive cardiac workup were normal. He also noticed occasional difficulty swallowing liquids for the past 2–3 years. Neurologic examination revealed right gaze nystagmus and slight tongue deviation to the right. Results of the rest of the cranial nerve, motor, and sensory examinations were normal. A MRI brain scan revealed an irregularly enhancing mass in the right cerebellopontine and cerebellomedullary cisterns, causing mass effect on the brainstem and a 5-mm shift and distortion of the fourth ventricle without hydrocephalus (figure 1, A and B).

As part of evaluation for unexplained syncope, an autonomic reflex screen³ was performed. Cardiovagal heart rate responses to deep breathing and the Valsalva maneuver were normal (figure 1, C and D). Beat-to-beat blood pressure changes during the Valsalva maneuver and head-up tilt were normal, indicating normal sympathetic vasomotor reflexes. Preoperative laryngoscopy revealed normal vocal

cord movement. The patient went on to have a suboccipital craniotomy and resection of the jugular foramen tumor, which proved to be a schwannoma (figure e-1 on the *Neurology*[®] Web site at www.neurology.org).

Immediately after surgery, the patient frequently aspirated when swallowing thicker liquids and required a temporary gastrostomy. His voice became hypophonic and hoarse. Repeat laryngoscopy revealed complete paralysis of the right vocal cord. He also developed unexplained persistent tachycardia with a resting heart rate in the range of 110–120 beats/min. Repeat autonomic testing revealed a profoundly impaired heart rate response to deep breathing and the Valsalva maneuver (figure 1, E and F) with normal sympathetic vasomotor function. Over the next 4 months, speech and swallowing function gradually improved and tachycardia resolved. Autonomic testing showed normalization of cardiovagal function (figure 1, G and H).

Discussion. The preganglionic parasympathetic outflow from the ventrolateral portion of the nucleus ambiguus is carried by the vagus nerve,⁴ which innervates the heart, lung, and upper gastrointestinal system. The right vagus nerve predominantly supplies the atria and SA node and provides cardioinhibitory tone. In contrast, stimulation of the left vagal nerve, which innervates the ventricles, has not been shown to cause any significant change in cardiac rhythm or rate.⁵ This observation is the rationale for implanting vagal nerve stimulator leads in proximity to the left vagus nerve⁶ because stimulation of the right vagal nerve might lead to bradycardia.⁷

In our patient, the preoperative HR_{DB} and VR, markers of cardiovagal function, were normal. After a surgical procedure involving a tumor that was intimately associated with the right vagus nerve, he developed sinus tachycardia, loss of baroreflex control of heart rate, and right vocal cord paralysis. A direct compressive effect of the schwannoma on the brainstem is unlikely to explain these findings because preoperative cardiovagal function was normal, and there was no postoperative brain stem compression. Serial autonomic testing in our patient, before and after surgery revealed that the right vagus nerve was prin-

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Figure 1 MRI brain and serial cardiovascular responses

MRI T1 axial images (A) without contrast and (B) with contrast show enhancement of the tumor in the right cerebellopontine and cerebellomedullary cisterns. (C) Systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) changes with the Valsalva maneuver. The maximum heart rate during the Valsalva maneuver over minimum heart rate after the maneuver (Valsalva ratio [VR]) was 1.94 (normal for age: >1.5). (D) Changes in RR interval with deep breathing. The average of the change in heart rate during the breathing cycle (HR_{DB}) preoperatively was 29.0 (normal for age: >13). (E, F) Blunted VR (1.49) and HR_{DB} (7.0) 1 month after surgery. Also note the baseline tachycardia compared with preoperative studies. (G, H) Improvement of VR (2.35) and HR_{DB} (25.0) 3 months after surgery. Amplitudes of both VR and HR_{DB} decreased after surgery and improved 3 months after surgery, indicating a transient impairment of the vagal control of heart rate.

cipally responsible for parasympathetic control of heart rate. The intact left vagus nerve could not compensate for this lack of the parasympathetic drive and unopposed sympathetic input to the heart led to tachycardia. Fortunately for this patient, both the control of heart rate and right vocal cord function improved over a few months, suggesting a reversible injury to the right vagal nerve rather than transection.

This case provides strong evidence to support the presence of highly asymmetric vagal innervation of the heart and highlights the use of simple autonomic reflex tests in diagnosis of right vagal nerve dysfunction. More importantly, vagal dysfunction should be considered in patients who have unexplained tachycardia after right-sided skull base or neck surgery.

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Author contributions: Dr. Muppidi prepared the manuscript, collected data, and prepared the figures. Dr. Gupta prepared the manuscript and figures. Dr. Vernino helped with collecting data and edited the manuscript and figures.

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Historical Abstract: October 1, 1978

EPIDEMIOLOGIC ASSESSMENT OF CHRONIC ATRIAL FIBRILLATION AND RISK OF STROKE: THE FRAMINGHAM STUDY

Philip A. Wolf, Thomas R. Dawber, H. Emerson Thomas, Jr., and William B. Kannel

Neurology 1978;28:973-977

Chronic atrial fibrillation (AF) as a precursor of stroke was assessed over 24 years of follow-up of the general population sample at Framingham, Massachusetts. Persons with chronic established AF, with or without rheumatic heart disease (RHD), are at greatly increased risk of stroke, and the stroke is probably due to embolism. Chronic AF in the absence of RHD is associated with more than a fivefold increase in stroke incidence, while AF with RHD has a 17-fold increase. Stroke occurrence increased as duration of AF increased, with no evidence of a particularly vulnerable period. Chronic idiopathic AF is an important precursor of cerebral embolism. Controlled trials of anticoagulants or antiarrhythmic agents in persons with chronic AF may demonstrate if strokes can be prevented in this highly susceptible group.

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Comment from Judith Harrer, MD, Editorial Board Member: *Wolf et al. demonstrate for the first time the importance of chronic idiopathic atrial fibrillation as an independent risk factor for stroke in a large epidemiologic study.*