The mechanisms of dysautonomia from carotid artery dissection were discussed recently in Circulation. A case was presented of a 49-year-old woman with a history of hypertension and vasovagal syncope who developed abrupt slurred speech and left hemiplegia. She had a right internal carotid artery dissection and right cerebral hemispheric infarction involving the insular cortex. During her hospitalization, the patient had episodes of severe bradycardia that Dulay et al1 attributed to “mechanical pressure to an already sensitive carotid sinus from thrombus secondary to a dissection of the internal carotid.” Sarikaya et al2 argued that disrupted descending autonomic pathways from the infarcted insular cortex were responsible. Right-sided insular lesions are known to cause bradyarrhythmias.3 We present a case of bilateral carotid artery dissection in which dysautonomia occurred in the absence of cortical damage.

Summary of Case
A 47-year-old right-handed woman with rheumatoid arthritis experienced symptoms of an apparent upper respiratory infection with a persistent cough after the syndrome resolved. After 2 weeks of coughing, she developed persistent headache and was found to have new hypertension, which persisted for the next month. Magnetic resonance imaging without angiography demonstrated a right internal carotid artery dissection without cerebral infarction (Figures 1 and 2). Follow-up computed tomographic angiography that day showed bilateral distal cervical internal carotid artery dissections, with marked luminal narrowing extending into the skull base (Figure 3). While being evaluated, she had nausea and diaphoresis, lost consciousness, and became unresponsive to noxious stimuli. Although a femoral pulse was present, blood pressure was unobtainable by sphygmomanometry. She was
on the verge of intubation and cardiopulmonary resuscitation, but awoke and had a normal neurological examination. The patient was admitted to the neurology intensive care unit, where her blood pressures (100 to 210/70 to 110 mm Hg) and heart rates (60 to 110 bpm) were labile, with marked fluctuations over minutes (Figure 4). Both enteral and parenteral antihypertensives were employed to maintain systolic blood pressures between 100 and 180 mm Hg.

During 1 episode of systolic hypotension to 80 mm Hg, she had transient left-sided weakness without residual symptoms or signs. Transthoracic echocardiogram showed a small or underfilled left ventricle with mild concentric hypertrophy and a normal ejection fraction of 65%. Magnetic resonance angiography of the renal arteries was normal. Results of 24-hour urine collection for metanephrines, normetanephrines, and vanillylmandelic acid were normal. Computed tomography 4 days after her initial magnetic resonance imaging showed no cerebral infarction, and the patient remained neurologically normal. Four months after the discovery of her dissections, her headaches had resolved, and she was symptom free with normal heart rate and blood pressure while on losartan and metoprolol.

**Discussion**

Limited data are available on autonomic disturbances after carotid artery dissection. Autonomic repercussions of manipulation or damage to the carotid artery in the absence of dissection are more commonly described. In a prospective series of 471 patients undergoing carotid stenting, there were 34 incidences of bradycardia, hypotension, or both. Presumably from manipulation of the carotid artery, sudden cardiac arrest may follow head and neck dissection. In this case, we hypothesize that edema of the vessel walls extended more inferiorly than the computed tomographic angiography–demonstrating luminal narrowing. Thus, we presume that pathology of at least 1 of the carotid arteries extended into a carotid bulb, producing dysautonomia.

Our patient acquired dysautonomia after bilateral carotid artery dissection with labile blood pressure characterized by (1) episodic hypertension; (2) episodic, severe, and symptomatic
hypotension; and (3) abnormal variability in heart rate. Although
dysautonomia may result from isolated lesions of the insular
cortex, our case illustrates that insular lesions are not required.
Thus, isolated lesions of the carotid arteries, without cerebral
infarction, are sufficient to cause dysautonomia.

Disclosures
Dr Ropper has accepted speaker’s honoraria from the American
Neurological Association, American Academy of Neurology, Cor-
nell Weill Medical College, Emory University School of Medicine,
and University of Kentucky College of Medicine.

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