Provokable Bilateral Vertebral Artery Compression Diagnosed With Transcranial Doppler

Nils Jakob Brautaset, MD

Background: Head and neck movements may cause vascular compression that produces a syndrome of vertebrobasilar insufficiency. Vertebrobasilar circulation was examined noninvasively in two patients who were able to provoke these symptoms repeatedly on demand.

Case Descriptions: Blood flow velocities in the basilar artery (case 1) and both posterior cerebral arteries (both cases) were measured continuously by transcranial Doppler sonography while the patients voluntarily performed the offending maneuvers and reproduced their symptoms. The provocative maneuvers evoked an immediate and precipitous drop in blood flow velocity, producing symptoms within seconds. Upon relief, the blood flow velocities showed a transient overshoot before returning to baseline values.

Conclusions: These observations indicate that the symptoms of vertebrobasilar insufficiency were due to real reductions in blood flow and demonstrate the usefulness of transcranial Doppler sonography to diagnose bilateral extracranial vertebral artery compression. (Stroke 1992;23:288-291)

Patients reporting symptoms provoked by head and neck movements compatible with the vaguely defined syndrome of vertebrobasilar insufficiency are common.1,2 However, angiography is seldom performed based on this indication alone, and only rarely has vascular compression been objectively demonstrated as the etiology of such symptoms.3,4

Transcranial Doppler ultrasonography (TCD) allows the measurement of blood flow velocity in human basal cerebral arteries through the intact skull, enabling the noninvasive evaluation of individual hemodynamic states.5 The high time resolution of TCD is particularly useful for investigating rapid and brief hemodynamic events.6-8 I describe two patients in whom TCD confirmed clinically suspected mechanical compression of the extracranial vertebral arteries.

Case Reports

Case 1

For 3 years a woman aged 77 years had noted that when she kept her neck rotated to the left she perceived increasing dizziness, light-headedness, and bilateral blurring of vision. She could sustain this for only about 10 seconds, and turning her head back relieved the symptoms within seconds. The symptoms appeared without preceding illness or trauma. She was able to provoke the symptoms repeatedly and on demand and had never lost consciousness or had drop attacks doing so.

Findings from a neurological examination and electroencephalography (EEG) were normal. Her blood pressure was 160/90 mm Hg bilaterally by the brachial cuff method. Computed tomography of the brain and occipitocervical junction was normal. Plain x-ray films of the cervical spine in the neutral, flexed, and extended positions and during rotation to either side revealed moderate degeneration without signs of dislocation. Angiography was not performed.

The TCD examination (TC 2000S, EME, Überlingen, FRG) was carried out with the patient sitting with a self-retaining 2-Mz probe strapped suboccipitally or temporally to assess blood flow velocity in the basilar artery (at depths of 94, 96, and 100 mm) and in both proximal posterior cerebral arteries (at depths of 54 and 58 mm on the right side and 60 mm on the left). The only temporal window was located posteriorly, and no reliable signals from the middle cerebral arteries were obtained using self-retaining probes. On maximal rotation of her head to the left, a marked drop toward 0 flow velocity at all depths examined in the basilar artery and posterior cerebral arteries was observed. Symptoms appeared 2–3 seconds after the flow velocity drop (Figure 1). When
FIGURE 1. Transcranial Doppler ultrasonographic recordings in woman aged 77 years with symptoms of vertebrobasilar insufficiency provoked by neck rotation to left. Recordings from basilar artery (top) and right posterior cerebral artery (PCA) (bottom) show drop in blood flow velocity (V) when she turned her neck to left and transient increase in velocity when neck was turned back to normal position. No symptoms and no change in blood flow velocity were observed in right PCA when neck was turned to right (bottom).

FIGURE 2. Transcranial Doppler ultrasonographic recordings in man aged 19 years who provoked symptoms of vertebrobasilar insufficiency by pressing palms simultaneously under both mastoid processes. Both posterior cerebral arteries (PCAs) show blood flow velocity (V) reductions during this maneuver followed by transient flow velocity increases after pressure release.
the patient turned her head back, flow velocities increased abruptly to values about 30% above baseline and then returned to pretest values. Rotation of her head to the right evoked no symptoms and no blood flow velocity changes. The velocities and flow directions were not influenced by ipsilateral arm hyperemia. Both extracranial vertebral arteries were identified with 4-MHz pulsed Doppler sonography in the normal position, but reliable signals were not obtained during neck movements. Tests for subclavian steal were negative. Carotid artery sonography showed normal findings. Carotid compression tests were not performed.

Case 2

During the last 2 years this man, aged 19 years, had experienced 10–15 episodes of light-headedness often but not always provoked by various neck movements. For 1 year he had noted that he could provoke bilateral blurring of vision and dizziness by folding his hands behind his neck and pressing simultaneously under both mastoids.

The neurological examination, EEG, plain x-ray films of the cervical spine, and 24-hour ambulatory electrocardiography were normal. His brachial blood pressure was 140/90 mm Hg bilaterally.

Various neck movements provoked no symptoms and did not change blood flow velocities recorded from the basilar artery or the posterior cerebral arteries. When carrying out retromastoid pressure the position of the patient’s hands precluded recordings from the basilar artery and extracranial vertebral arteries. However, in both posterior cerebral arteries (at depths of 70 mm on the right side and 68 mm on the left) a striking decrease in blood flow velocity was observed when he provoked the symptoms. The patient managed to maintain the pressure for about 10 seconds, and on releasing the pressure blood flow velocity increased abruptly and a transient overshoot was observed (Figure 2). No changes in middle cerebral artery blood flow velocities were seen during these maneuvers. Doppler investigation of the extracranial vertebral arteries showed no signs of subclavian steal. Carotid artery sonography showed normal findings.

Discussion

The findings in case 1 probably reflect compression of both vertebral arteries during neck rotation to the left. The location and cause of the vascular compression has not been delineated exactly. Angiography was considered as not indicated because it could hardly have had any therapeutic consequences. Case 2 inadvertently performed bilateral suboccipital vertebral artery compression on himself. The cause of his fits of light-headedness remains unclear, but since the TCD recording remained normal during neck movements, mechanical artery compression seemed unlikely. Thus, angiography was not performed.

Some patients give histories suggesting transient vertebrobasilar insufficiency provoked or aggravated by certain neck positions. Mechanical vertebral artery compression may be suspected in such patients, but angiographic documentation requires vertebral artery injections performed in the offending neck position. The risk of complications from angiography and the relatively small chance of unveiling a surgically correctable lesion often leaves the clinician with a diagnosis based on clinical grounds alone. In this situation TCD can be a valuable tool. Technical pitfalls and limitations must, however, be considered. Due to vessel tortuosity, unequivocal differentiation between the right and left intracranial vertebral arteries may be difficult. If during testing the ultrasonic probe becomes dislodged, the signal loss may be spuriously interpreted as indicating blood flow arrest. Insonation of the extracranial vertebral artery is difficult to maintain during neck movements, especially when a given neck position can be sustained for only a few seconds. In the present cases, strapping the probe to the head facilitated continuous TCD recordings from the intracranial vessels during provocation testing. The reproducible finding that, upon relief, blood flow velocities immediately rose and transiently increased to levels considerably above baseline probably reflects reactive hyperemia and strongly supports the hypothesis that the symptoms of vertebrobasilar insufficiency were due to real reductions in blood flow.

Test occlusions of one extracranial verteobasilar artery have been performed by von Reutern and Büdingen. When one vertebral artery is compressed briefly, a normal contralateral artery will probably be able to provide the extra blood flow required and no major changes in the basilar and posterior cerebral artery blood flow velocities are expected. During compression of both vertebral arteries, filling of the posterior cerebral arteries and the basilar artery depends on the capacity of the posterior communicating arteries. It remains unclear whether the symptoms of vertebrobasilar insufficiency and the blood flow velocity changes observed in the patients reported here unequivocally reflect poorly developed posterior communicating arteries or if compensation is inadequate under such circumstances even with a normal, textbooklike circle of Willis.

Sakai et al used single-photon emission computed tomography to demonstrate reductions in blood flow in the left cerebellum and right occipital region during an attack of vertebrobasilar insufficiency in a patient whose vertebral angiograms revealed a congenitally small right vertebral artery and, with the head turned to the left, severe extravascular compression of the left vertebral artery.

The two cases reported here illustrate that TCD is clinically useful to demonstrate the hemodynamic effects of bilateral extracranial vertebral artery compression. Thus, TCD may be the method of choice to detect rapid intracranial hemodynamic changes and to relate them to the symptoms reported by the patients.

Acknowledgment

The author wishes to thank Dr. Karl-Fredrik Lindegaard for scientific assistance and critical review of the manuscript.
References


KEY WORDS • blood flow velocity • ultrasonics • vertebrobasilar insufficiency
Provokable bilateral vertebral artery compression diagnosed with transcranial Doppler.

N J Brautaset

Stroke. 1992;23:288-291
doi: 10.1161/01.STR.23.2.288
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1992 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/23/2/288

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke 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 Stroke is online at:
http://stroke.ahajournals.org//subscriptions/