J Stroke Cerebrovasc Dis 1991;1:103–108

Reversible Basilar Artery Blood Flow in Subclavian Steal Syndrome

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Transcranial Doppler ultrasound examination of intracranial vertebral and basilar arteries was performed on a 61-year-old man who presented with symptoms of amaurosis fugax and subclavian steal syndrome. During arm exercise, flow in the basilar artery reversed direction from cephalad to caudal, coincident with symptoms of vertebrobasilar insufficiency. Following bilateral carotid endarterectomies, exercise-induced reverse flow velocities in the basilar artery were markedly increased, and the patient remained asymptomatic. These findings suggest that collateral flow from the anterior cerebral circulation may play an important role in the pathogenesis of brainstem ischemia in subclavian steal syndrome and that transcranial Doppler may identify a subgroup of patients who will benefit from procedures to augment cerebral blood flow. **Key Words:** Carotid endarterectomy—Subclavian steal syndrome—Transcranial Doppler—Vertebrobasilar ischemia.

The term *subclavian steal syndrome* was first used in 1961 to describe patients with a combination of angiographic and clinical findings suggesting a "steal" of blood from the brainstem circulation (16,18,27). The angiographic hallmark of this syndrome is high-grade stenosis or occlusion of the subclavian artery proximal to the origin of the vertebral artery, with reverse blood flow in the vertebral artery ipsilateral to the lesion (3,4,15,25,32). Blood is presumably shunted from the posterior circulation into the subclavian artery distal to the lesion, supplying collateral flow to the affected arm (7,11,17,23,27). Clinically, the syndrome is manifest by symptoms of vertebrobasilar insufficiency associated with upper extremity exercise or position, although reductions in posterior circulation blood flow have not been directly demonstrated. The presence of similar abnormal flow patterns by angiography or conventional Doppler ultrasound in asymptomatic patients (3,9,15,36) has raised questions regarding the significance of these flow abnormalities. A high correlation between symptomatic subclavian steal syndrome and concurrent anterior circulation stenoses (3,15) suggested the potential importance of collateral flow in determining ischemic symptoms. We present the case of a patient with subclavian steal syndrome and bilateral high-grade internal carotid artery stenoses, in whom basilar artery blood velocities were determined at rest and with exercise using transcranial Doppler (TCD) ultrasound.

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Case Report

A 61-year-old man presented to the Seattle VA Medical Center with a history of amaurosis fugax and symptoms of transient vertebrobasilar ischemia. For approximately 10 months he had noted intermittent

Figure 1. *Early arterial phase of preoperative subtraction angiogram (arch injection) shows web stenosis in proximal innominate artery (arrowhead), markedly diminished flow in left common carotid artery (small arrows), and occlusion of the proximal left subclavian artery (large arrow).*

episodic diplopia, occasionally associated with drop attack. These spells typically lasted 1–5 min, resolved entirely without residual effect, and were frequently precipitated by performing tasks with the left arm. One month prior to admission, the patient experienced a single episode of blindness in the left eye, which completely resolved after 10 min. In retrospect, he noted a progressive weakness and fatigability in both arms, especially the left, which was accentuated by working with his arms above his head.

The neurologic examination was entirely normal. Blood pressure measured in the right arm was 150/80 compared to 80/50 in the left. There was a diminished pulse over the left carotid and superficial temporal arteries and a bruit over the right carotid artery. Fundoscopic examination was unremarkable. The radial artery pulse on the left was decreased compared to the right and disappeared when the arm was moderately abducted above the head.

Sinus bradycardia was present on the electrocardiogram, and echocardiography demonstrated minimal mitral valve prolapse. Awake and sleep electroencephalography and computed cranial tomography (CCT) were normal. Conventional duplex Doppler studies revealed (a) 80–99% stenosis of right internal carotid artery, (b) apparent occlusion of the left common and internal carotid arteries with external carotid artery reconstitution, (c) bilateral subclavian stenosis, and (d) retrograde flow in the left extracranial vertebral artery.

Angiography through a right femoral approach demonstrated the following (Figs. 1 and 2): (a) a 60% web stenosis in the proximal innominate artery; (b) occlusion of the left subclavian artery at its origin; (c) filling of the distal left subclavian artery via retrograde flow down the left vertebral artery; (d) irregular 80% stenosis of the right internal carotid artery that filled both left and right hemispheres; and (e) 95% stenosis

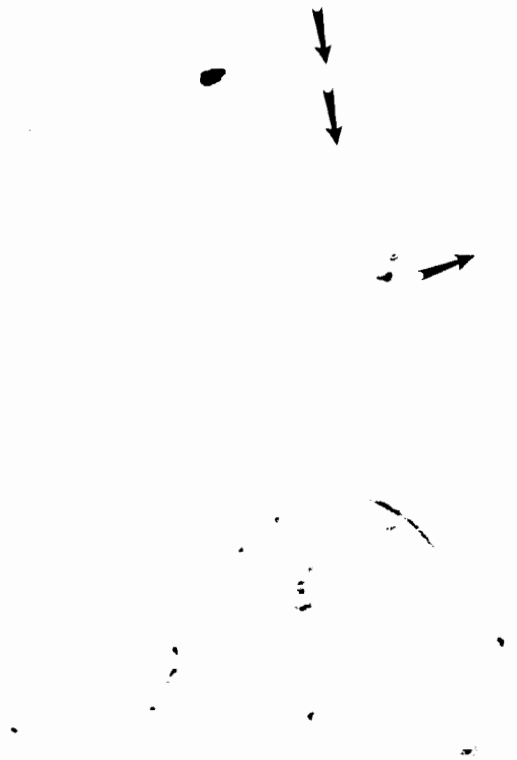

Figure 2. *Late arterial phase of preoperative subtraction angiogram demonstrates filling of left subclavian artery via retrograde flow through left vertebral artery (arrows show direction of flow).*

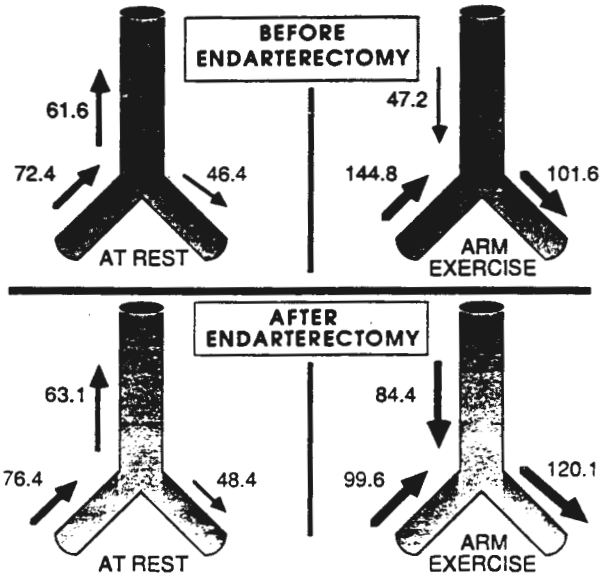

Figure 3. *Schematic diagram demonstrates intracranial right and left vertebral and basilar artery TCD velocities at rest and after left arm exercise. Prior to surgery, exercise produced increased cephalad velocity flow in the right vertebral artery, increased caudal velocity in the left vertebral artery, and reversal of flow from cephalad to caudal in the basilar artery, during which the patient had symptoms of vertebrobasilar ischemia. Following bilateral carotid endarterectomies, similar alterations in flow occurred after arm exercise, but the caudal flow velocity was nearly doubled in the basilar artery, and the patient was asymptomatic.*

of the left internal carotid artery and moderate collateral filling from left external carotid artery branches.

Preoperative TCD examination was performed as described by Aaslid et al. (1,2) using range-gated Doppler with an ultrasonic frequency of 2 MHz. Examinations were performed by a qualified technician with extensive experience in this technique. The patient was examined in the sitting position with the head slightly flexed, and blood velocities in the left and right intracranial vertebral and basilar arteries were determined at rest at depths of 70 mm and 85 mm, respectively. Mean velocities averaged for five measurements in each vessel before exercise were as follows (Fig. 3): right vertebral artery, 72.4 cm/s cephalad direction; left vertebral artery, 46.4 cm/s caudal (reversed) direction; and basilar artery, 61.6 cm/s cephalad direction. Concurrent anterior circulation TCD determinations could not be obtained at any time from the patient, presumably due to anatomical limitations of the temporal bone or marked reductions in blood velocity (1,2).

Exercise testing was performed using a 12-pound weight. The patient was instructed to lift the weight over his head 15 times with his left arm, then hold the weight in his lap while squeezing it continuously with his left hand. TCD measurements of the previously studied vessels, using the same range settings, were obtained immediately after the arm exercise. Averages of mean velocities (five readings per vessel) after exercise were as follows (Fig. 3): right vertebral artery, 144.8 cm/s cephalad direction; left vertebral artery, 101.6 cm/s caudal (reversed) direction; and basilar artery, 47.2 cm/s caudal (reversed) direction. During exercise testing, the patient complained of dizziness, blurred vision, and diplopia. Left-hand squeezing was discontinued, and symptoms resolved within 2 min. Repeat TCD examination after resolution of symptoms showed that basilar artery blood velocity had returned to pre-exercise flows in a cephalad direction.

Carotid endarterectomy was initially performed on the left (symptomatic) side. At surgery, an ulcer with fibrin thrombus was located just proximal to a focal atherosclerotic web producing a residual lumen of 1.5 mm. Because of the apparent anterior-to-posterior circulation collateral flow during subclavian steal, the 80% internal carotid stenosis on the right (asymptomatic) side was operated at 2 weeks after the initial surgery. At surgery, a nonulcerated atherosclerotic plaque reduced the residual lumen of the origin of the internal carotid artery to 2 mm. The patient tolerated both procedures well and was subsequently studied by TCD examination at 6 weeks following the second procedure.

Postoperative TCD recordings were performed in a manner identical to preoperative measurements. After surgery, the magnitude and direction of velocities at rest corresponded closely to those obtained preoperatively (Fig. 3). Similarly, left-arm exercise increased cephalad flow in the right vertebral artery and increased caudal flow in the left vertebral artery, compared to measurements at rest. In contrast to preoperative exercise determinations, however, basilar artery caudal (reverse) velocity was nearly doubled (84.4 cm/s versus 47.2 cm/s). The patient did not develop any neurologic symptoms during prolonged exercise.

Discussion

This report documents TCD evidence of reversed basilar artery flow during symptomatic subclavian steal. Moreover, bilateral carotid endarterectomies

effectively increased the basilar artery caudal (reversed) flow velocity during exercise, corresponding to a resolution of symptoms of brainstem ischemia. In this patient, TCD measurements at rest showed reversal of flow in the left intracranial vertebral artery and normal flow direction in the basilar artery (Fig. 3). Exercise of the affected arm caused an increase in cephalad flow in the right vertebral and caudal flow in the left vertebral, as previously observed in extracranial vessels by conventional Doppler (3). During exercise, there was reversal of basilar artery blood flow, which was temporally related to the onset of symptoms of vertebrobasilar insufficiency. Basilar artery blood velocity returned to the normal direction soon after cessation of exercise and the symptoms resolved. Following bilateral carotid endarterectomies, the reverse (caudal) basilar artery flow during exercise was increased, and symptoms of brainstem ischemia did not occur. These results suggest that subclavian steal syndrome may reflect a true "steal" of flow from the vertebrobasilar circulation and demonstrate the importance of collateral flow from the anterior cerebral circulation in the pathogenesis of this disorder.

TCD makes use of low-frequency, range-gated Doppler ultrasound to penetrate the bone and soft tissue, which effectively block conventional Doppler signals (1,2), enabling accurate determination of blood velocities in large intracranial arteries. When large vessels at the base of the brain maintain a constant diameter during changes in blood flow, TCD velocity measurements directly reflect changes in cerebral blood flow (CBF) (22). In subclavian steal syndrome, brainstem ischemia is presumably not accompanied by significant alterations in basilar artery diameter; therefore, the observed changes in blood velocity detected by TCD probably corresponded to changes in CBF.

Subclavian steal syndrome was first described in patients with clinical symptoms of vertebrobasilar insufficiency and angiographic evidence of abnormal retrograde flow in the vertebral artery ipsilateral to a stenotic subclavian artery (15,16,18,27). Typical clinical presentation of subclavian steal syndrome may include various symptoms of intermittent vertebrobasilar ischemia, usually precipitated by exercise or position of the affected arm (3,10,15,16,27). Common presenting neurologic symptoms include dizziness or vertigo (50%), visual blurring (30%), and diplopia (15%) (16). Although atheromatous disease is by far the most common etiology for subclavian steal syndrome, congenital lesions (e.g., coarctation or atresia), trauma, and arteritis have been reported to produce the syndrome (3,4,10). Lower subclavian artery pressure (11,17,23,27) promotes the development of segmental intervertebral anastomoses (5) or direct vertebro-vertebral steal (7,25,33). Exercise of the arm ipsilateral to the subclavian stenosis accentuates the abnormal flow pattern (24,31) and presumably leads to the development of vertebrobasilar ischemia (3,10,15,16). The relationship of subclavian steal to lesions affecting the anterior circulation or circle of Willis has been well documented (7,35), although the hemodynamic significance of these lesions is not known. The concept of abnormal flow associated with subclavian stenosis led to the development of a variety of surgical techniques to restore anterograde flow in the vertebral artery (10,19). These procedures were often associated with high morbidity and have not been demonstrated to be more effective than medical management in preventing strokes (15).

Abnormal conventional Doppler flow patterns in subclavian steal syndrome are manifest as asymmetric brachial artery signals and reversed flow direction in the extracranial vertebral artery (3,8,9,13,19,23,29,36). Although conventional Doppler correlates well with angiographic evidence for subclavian stenosis (7,12), especially when combined with cuff occlusion and release of the brachial artery (3), this technique cannot differentiate latent flow abnormalities from symptomatic lesions (28). The incidence of significant subclavian stenosis with ipsilateral reversal of vertebral flow varies considerably according to the criteria, techniques, and population studied. From 6,534 angiograms, Fields and Lemak (15) noted 168 (2.4%) patients with subclavian steal flow pattern. By conventional Doppler, the incidence of abnormal flow patterns consistent with subclavian steal has varied from 0.65% (3) to 2.5% (36).

The relationship of abnormal Doppler flow patterns to symptomatic vertebrobasilar ischemia varies in the literature. Early reports estimated the incidence of symptoms at 80% or higher (15,30), although a more recent study described symptoms in only 37% of patients with this lesion (3). Nevertheless, in all studies, the presence of either abnormal flow patterns or symptomatic subclavian steal was not correlated with an increased risk of vertebrobasilar stroke. In two large retrospective reviews (3,10), no patients with subclavian steal developed brainstem strokes, although transient ischemia was common. In a prospective analysis of asymptomatic patients with abnormal conventional Doppler flow patterns, only 3 of 45 developed symptoms, and no infarcts occurred (9). These data suggest that subclavian stenosis with ipsilateral reversal of extracranial vertebral flow is infrequently associated with progression of symptoms and has a relatively benign natural history.

There is a high incidence of other hemodynamically significant lesions elsewhere in the craniocerebral circulation in patients with subclavian steal syndrome, particularly lesions involving the anterior circulation (3,15). It has been suggested that compromised collateral flow from the anterior circulation is a necessary prerequisite for the development of symptoms in patients with subclavian stenosis (15,20,23, 25), although means to document intracranial blood flow dynamics during exercise have not been previously available. Clinical investigation and animal models of subclavian steal syndrome have demonstrated the importance of intracranial collateral flow in the vertebrobasilar circulation. Acute subclavian occlusion in primates produced reversed flow in the ipsilateral vertebral artery, with associated anterograde increases in contralateral vertebral and both carotid artery flows (27). Although subclavian occlusion in primates produced variable reductions in cortical and brainstem regional cerebral blood flow (rCBF) (26), other investigators (14,16) showed no change in rCBF, electroencephalogram, or cortical oxygen tension, even in the setting of arm exercise. Bentivoglio et al. (6) demonstrated the importance of anterior-to-posterior circulation collateral flow during relative hypotension in a primate model of basilar artery occlusion. In humans, variable decreases in both brainstem and cortical rCBF and metabolism ($CMRO_2$) have been demonstrated with subclavian steal (21,34), although similar changes were observed in patients with generalized cerebrovascular atheromatous disease (34). In patients with reversed vertebral artery flow by angiography, individuals with symptoms of vertebrobasilar insufficiency were nearly twice as likely to demonstrate a discontinuity of the circle of Willis between anterior and posterior circulations (20). These findings suggest that posterior communicating arteries provide an important source of intracranial collateral flow in the setting of vertebrobasilar insufficiency and that development of ischemic symptoms may depend on the adequacy of this collateral flow.

Acknowledgment: Dr. Mayberg is a recipient of the American Association of Neurological Surgeons Young Clinician-Investigator Award and NINCDS Clinical Investigator Award (NS01191).

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