Positional Vertebrobasilar Ischemia: Pathogenesis and Diagnosis

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Purpose
Positional vertebrobasilar insufficiency, also known as Bow Hunter syndrome (1), is a rare condition in which the patient experiences posterior circulation ischemic symptoms with head turning (2, 3, 4). We are reporting the sonographic and angiographic findings in seven patients with this syndrome treated at our institution over 10 years. Based on our experience, we propose specific angiographic and sonographic criteria to diagnose this condition, as well as the use of ultrasound during angiography to aid in lesion identification.

Materials & Methods
All patients underwent ultrasound/transcranial Doppler (TCD) and angiographic evaluation. TCD was used to screen for posterior circulation flow arrest during duplication of symptoms by head turning. Angiography, often supplemented by TCD, was used to define the anatomical variations associated with this syndrome.

Results
All patients in our study demonstrated occlusion of a vertebral artery with head turning. Three patients had bilateral vertebral artery occlusion. Three patients had occlusion at C1-2, the remainder had occlusion more inferiorly in the cervical spine or neck soft tissues. All patients had limited collateral flow from anterior to posterior circulation (small or absent posterior communication arteries, fetal origins of the posterior cerebral arteries or some combination of both). One patient had a prior dens fracture and a second had a Klippel-Feil deformity. Demonstration of the anatomical obstruction to flow required duplication of symptoms during angiography, including angiography in an upright position in one patient. Angiographic demonstration of vascular obstruction was most reliable when flow arrest was confirmed by TCD during angiography.

Conclusion
Based on our experience, we believe that the following findings are present in this symptom complex: (1) poor collateral flow from anterior to posterior circulation; (2) unilateral or bilateral vertebral artery flow-arrest during symptomatic head turning; (3)
TCD demonstration of posterior circulation flow arrest during symptomatic head turning and hyperemia when the head is returned to a normal position; (4) level of obstruction most commonly at C1-2, but may occur at any cervical level. Additionally, based on our experience we believe that simultaneous TCD during angiography can confirm flow arrest prior to each angiographic run. This facilitates the diagnosis, increases angiographic sensitivity, and decreases the risk by reducing the number of angiographic runs required to demonstrate the pathology.

References