Pearls & Oy-sters:

The orbital bruit

A poor man's angiogram

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CLINICAL PEARLS

1. An orbital bruit represents increased blood flow through the collateral arterial system and intracranial arterial supply. In the correct clinical context, the presence of an orbital bruit should make the examiner suspect either a severe stenosis or occlusion of either the ipsilateral or the contralateral internal carotid artery (ICA).
2. Orbital bruits can represent compensated perfusion to the contralateral hemisphere. This occurs as flow velocity increases through the ipsilateral ICA and through the anterior communicating artery. Thus, blood flow moves from the asymptomatic side to the occluded vascular territory.
3. When listening for an orbital bruit, auscultate by placing the bell of the stethoscope over the patient's closed eye. In an effort to eliminate the noise of rhythmic eyelid flutter, the patient should then be instructed to open both eyes and gaze at a point across the room. If necessary, the eyelid can be passively shut using the bell of the stethoscope.
4. Although not featured on many popular stethoscopes, a deep and narrow bell (i.e., the Ford-Bowles stethoscope) is ideal for the purpose of ocular and arterial auscultation.

CLINICAL OY-STERS Orbital bruits are also found in systemic illness (severe anemia, thyrotoxicosis) and vascular anomaly (carotid-cavernous fistula, arterial vascular malformation).

CASE REPORT An 83-year-old right-handed man presented with increasing episodes of right limb shaking over a 9-month period. Each episode lasted several minutes and was brought on by both exertion and orthostatic intolerance. Medical history was significant for metabolic syndrome complicated by coronary artery disease and dialysis-dependent end-stage renal disease. One month prior to presentation, these limb-shaking episodes increased in frequency and were also noted during dialysis. Over this same time

interval, routine blood pressure assessment revealed a decrease of 10 to 20 mm Hg in systolic pressure.

At presentation, he was diagnosed with probable limb-shaking transient ischemic attacks. While his neurologic examination was essentially unremarkable, cerebrovascular examination identified a high-pitched systolic left cervical bruit at the level of the carotid bifurcation, and a right orbital bruit. MRI did not demonstrate any acute or chronic infarcts. Carotid ultrasound revealed high-grade left internal carotid artery (ICA) stenosis. Cerebral angiogram demonstrated an estimated 95% focal, short-segment stenosis with calcifications at the left carotid bifurcation (figure, A). Collateralization to the left anterior and middle cerebral arteries was provided by flow through the anterior communicating artery, in addition to collateral flow from the left external carotid artery (ECA) (figure 1, B and C). The latter results were anticipated on the basis of the cerebrovascular examination.

DISCUSSION In 1928, Harvey Cushing referred to cephalic auscultation as a “forgotten practice,” commenting that it was “the one thing most likely to be neglected in a routine neurologic examination.”¹ Utilizing the bell of the stethoscope, proper auscultation of the skull involves listening to the orbits, frontal region, temporal region (including the mastoid process), and atlanto-occipital region. Generally speaking, bruits are a consequence of increased blood velocity or turbulence, and result from a spectrum of both benign and pathologic conditions. As described by C. Miller Fisher² in 1957, the physical finding of an orbital bruit can sometimes provide evidence for the presence of contralateral internal carotid occlusion or stenosis. In our patient, the presence of an orbital bruit provided valuable clinical information regarding collateralization, carotid occlusion, and a robust anterior cerebral circulation.

Collateralization is an important process in stroke physiology, and can influence both ischemic localization and size of infarct. Regarding collateralization

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Figure Cerebral angiogram findings in a patient with an orbital bruit

Left carotid angiogram (A) reveals high grade, short segment carotid stenosis (white arrow) at the bifurcation. Left carotid angiogram (B) demonstrates retrograde flow across the ophthalmic artery (white arrow) to the supraclinoid internal carotid artery from the left external carotid artery (black arrow). Cerebral angiogram performed from the right carotid artery (C) shows collateral filling of the left anterior and middle cerebral arteries (white arrow).

and intracerebral blood flow, primary collateralization results from an acute vascular occlusion, while secondary collateralization develops over longer periods of time. In primary collateralization, blood flow redistribution occurs primarily within the circle of Willis. Secondary collateralization can occur via both leptomeningeal collaterals and external carotid arterial flow. Compensatory ECA flow occurs in a retrograde manner via the ophthalmic artery.

Compensatory increased flow through the nonoccluded carotid may account for the bruit occurring over the eyeball contralateral to the stenosis.³ However, carotid siphon stenosis ipsilateral to the ocular bruit has also been demonstrated to be a very common occurrence.⁴ With this in mind, the finding of a unilateral carotid bruit or a lateralizing neurologic deficit can further aid in the interpretation of an ocular bruit. Importantly, the significance of a carotid bruit can be judged by the findings of increasing focality and duration, along with a higher pitch.

An additional clinical sign of increased ophthalmic arterial flow is the presence of dilated episcleral arteries, which have been reported as a manifestation of secondary collateralization from progressive ICA stenosis.⁵ Palpation of the branches of the facial ar-

tery can be useful in assessing a hyperdynamic external carotid system ipsilateral to a high-grade ICA lesion.⁶ However, common carotid disease will result in decreased pulsations ipsilateral to the lesion. Several branches may be palpated, including the angular arteries on the sides of the nose, and the superficial temporal arteries, anterior to the tragus. Retrograde ophthalmic artery flow may be inferred by a loss of the supraorbital pulsation upon compression of the superficial temporal artery, as ophthalmic arterial flow is dependent on ECA flow through the superficial temporal artery.⁷ Retrograde supraorbital arterial flow may also be demonstrated by ultrasound Doppler, which can be a useful extension of the bedside examination.

In this patient, the presence of orbital bruits informed the examiner of both vascular disease and compensatory collateralization. The patient's severe carotid stenosis manifested clinically as limb-shaking transient ischemic attacks.⁸ The history and neurovascular examination guided the diagnostic evaluation strongly away from a pursuit of focal motor seizures and toward a vascular etiology.

If multifocal cranial bruits are identified, the examiner should consider additional diagnoses. For instance, hyperdynamic states such as thyrotoxicosis and anemia, in addition to structural lesions, such as an arteriovenous fistula or carotid-cavernous sinus fistula, may also be identified by an ocular bruit. This again emphasizes the importance of a thorough history and physical examination.

DISCLOSURE

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