


Elimination of Subjective Bruit With Compression of Temporal Artery: New Physical Sign Indicative of Internal Carotid Artery Occlusion
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SUMMARY A patient is presented with a right central retinal artery occlusion and subjective bruit over right ear. Digital compression of the right temporal artery eliminated the bruit and release reproduced the bruit. Carotid arteriogram showed bilateral internal carotid occlusion with a prominent right temporal artery.

This sign reflects compensatory hyper-perfusion of the external carotid artery due to occlusion of ipsilateral internal carotid artery.

MANY CLINICAL SIGNS have been described in which internal carotid artery occlusion causes augmented blood flow through the external carotid system. Recognition of a dilation of conjunctival and episcleral blood vessels, neovascular glaucoma, newly formed circular vessels at the base of each iris indicate perfusion of intracranial vessels via the ophthalmic artery in a retrograde fashion from branches of the external carotid artery. Auscultation and palpation may reveal a carotid bruit which diminishes with pressure over the ipsilateral facial and temporal arteries. Ocular bruises over either eye, pulsation of the angular artery, or the brow artery, sudden disappearance of a transmitted cardiac sound, all may suggest occlusion of the ipsilateral internal carotid artery.

Case Report
The following case illustrates another physical sign indicative of occlusion of the internal carotid artery with augmented external carotid perfusion.

A 53-year-old man was seen with a history of the sudden onset of right eye blindness accompanied by minimal retro-orbital pain of 2 months duration. He became aware of a pulsating "swishing" sound over the right ear, most prominent at night. An episode of transient left hemiparesis had occurred 4 years earlier. Examination showed dilated right-sided episcleral arteries, the right pupil was 4 mm and minimally reactive, the left pupil 3 mm and reactive. An afferent pupillary defect was present on the right. There was marked attenuation of the retinal vasculature with a pale disc and a minimal retinal fiber layer consistent with prior central retinal artery occlusion. The bruit was accentuated when the examiner’s finger was inserted into the right external ear canal, and firm pressure over the right temporal artery anterior to the ear eliminated the subjective bruit. Release of this pressure led to a return of the bruit. With a modification of the stethoscope enabling the examiner and the patient to hear simultaneously, confirmed a bruit which disappeared with digital pressure over the artery. Carotid arteriograms detected bilateral internal carotid obstruction prominent right temporal artery perfusion and retrograde filling from the external carotid artery of the intracranial circulation to the homolateral cerebral cortex.

Discussion
A clinical distinction between a carotid ulcerative lesion and carotid occlusion causing cerebral or ocular symptoms is difficult. Management of an ulcerative lesion without obstruction is considered by some to require carotid endarterectomy. In the presence of complete occlusion, either medical therapy or extra-intracranial bypass may be under consideration. Although arteriography may be required in either case, recognition of an internal carotid artery occlusion on physical examination may be very helpful.
This new physical sign, the elimination of a subjective bruit with occlusion of ipsilateral temporal artery, indicates augmented flow due to ipsilateral internal carotid obstruction. If a subjective bruit is not present, insertion of the finger into the ear canal may bring out the bruit.

It is not certain how often this sign will be present without any other physical evidence of augmented flow through the external carotid system. The absence of this sign does not rule out significant stenosis or occlusion of the internal carotid artery. Caution should be exercised in eliciting this sign: marginal blood supply through the external carotid artery may be present and the appearance of ocular or cerebral symptoms with pressure over the temporal artery calls for immediate termination of the maneuver.

References

Massive Intracerebral Hemorrhage Complicating Cardiac Catheterization with Ergonovine Administration

JOSEPH H. PIATT, JR., M.D.

SUMMARY Massive intracerebral hemorrhage is reported as a complication of cardiac catheterization with ergonovine administration. Possible mechanisms relating stroke to cardiac catheterization are reviewed. Patients who suffer neurologic deterioration after this procedure require prompt evaluation including computed tomography.

STROKE is a recognized but infrequent complication of cardiac catheterization. In large series and surveys of catheterizations published in the last decade, the incidence of new, lasting neurologic deficits has been reported between 0.02% and 0.2%, and several series have been entirely free of this complication. The mechanism has been thought to be embolization of thrombus stripped from the catheter tip or guidewire or dislodged from the endocardial surface, while some reports have stressed atheroembolism from plaques in the aortic arch. In only a few cases has the pathology been defined by radiographic studies, operative intervention or autopsy. These cases have been characterized by embolic occlusion of a major cerebral artery with infarction, often hemorrhagic, in the territory of the occluded vessel. In contradistinction to hemorrhagic infarction, I was unable to discover a previous report of massive intracerebral hemorrhage (ICH) complicating cardiac catheterization.

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Case Report

The patient is a 51 year old right-handed white woman who was admitted for evaluation of chest pain. About one month earlier she had developed a dull, substernal pain radiating to the back and to both arms. Her pain was precipitated by exertion and was associated with dyspnea. It lasted 5 to 10 minutes, and it was ameliorated by leaning forward, by rest and by nitroglycerin. A treadmill test had been positive. There was no history of diabetes or hypertension. She described a chronic, bifrontal and bitemporal headache exacerbated by anxiety and at the time of her menses. The headache was throbbing and infrequently associated with nausea and vomiting. It typically lasted several hours at a time.

The brachial blood pressure was 125/85, and prior to cardiac catheterization it ranged no higher than 160 torr systolic or 85 torr diastolic. Her weight was 52 kg. The fundi were normal. There was a soft systolic bruit at the angle of the mandible on the left; the carotid pulses were normal. The cardiac rhythm was regular. S1 and S2 were normal, and there were no gallops. There was a grade 2/6 systolic murmur best heard over the pulmonic area with radiation to the left sternal border and to the apex. Peripheral pulses were normal. The neurological examination was normal.

Routine laboratory work was normal. Specifically,
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