Ocular Pneumoplethysmography and Ophthalmodynamometry in the Diagnosis of Central Retinal Artery Occlusion

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SUMMARY Ocular pneumoplethysmography and ophthalmodynamometry measure ophthalmic arterial system pressures to assess noninvasively the hemodynamics of the carotid system. A previously unreported circumstance in which these tests complement one another is central retinal artery occlusion. Typically, the ipsilateral retinal artery pressure, measured by ophthalmodynamometry, is greatly decreased or is zero, whereas the ophthalmic systolic pressure, measured by ocular pneumoplethysmography, is normal.

CENTRAL RETINAL ARTERY OCCLUSION usually presents as an acute, painless, unilateral loss of vision. Ophthalmoscopically, the optic disk and the retina become pale, the retinal vessels narrow, and a macular cherry red spot is often visualized. Central retinal artery occlusion may be confused with venous occlusions, branch artery occlusions, and ischemic optic neuropathy, despite the usual differences in temporal profile, clinical course, and ophthalmoscopic manifestations. Furthermore, any of these phenomena may occur in various combinations. Ocular pneumoplethysmography and ophthalmodynamometry, in combination, provide a safe and rapid means of confirming the diagnosis of central retinal artery occlusion and may provide clues as to the cause of the condition.

Report of Cases

Case 1
A 59-year-old man with a long history of hypertension experienced an episode of left amaurosis fugax 1 month before admission. The episode lasted 4½ minutes and resolved completely. Two more such episodes occurred the same day, prompting hospitalization and treatment with intravenously administered heparin. Results of four-vessel cerebral angiography were normal. The patient was dismissed on warfarin therapy.

Two weeks before admission, he had another episode of sudden onset of complete visual loss of the left eye, without improvement. An ophthalmologist in his home community diagnosed ischemic optic neuropathy. Treatment with inhalation of 95% oxygen and prednisone was given, but no improvement occurred. A left temporal artery biopsy specimen was normal, as was the sedimentation rate. He was referred to the Mayo Clinic for further evaluation.

Ophthalmologic examination showed a visual acuity of 14/21 on the right and light perception only on the left. Intraocular pressures were 16 mm Hg bilaterally. There was a left-sided afferent pupillary reflex defect. Ophthalmoscopic examination of the left eye showed a pale retina with thready vessels and edema of the macular area with an associated cherry red spot. Ophthalmodynamometry revealed retinal artery pressures of 130/54 mm Hg on the right and 12/0 mm Hg on the left. Ocular pneumoplethysmography revealed ophthalmic systolic pressures of 110 mm Hg bilaterally, with brachial systolic pressures of 154 mm Hg on the right and 148 mm Hg on the left. Results of a periorbital Doppler study were normal. General examination revealed a blood pressure of 190/110 mm Hg but was otherwise normal, as was a cardiac sector scan.

Case 2
A 71-year-old man reported the sudden onset of decreased vision in his right eye 2 weeks before admission. He could only see gross hand movements. By the following day, he was blind in the right eye. An ophthalmologist in his home community made a diagnosis of central retinal artery occlusion and referred the patient to the Mayo Clinic for further evaluation. The patient’s medical history had been unremarkable, with the exception of long-standing mild hypertension treated with a diuretic (Dyazide).

On admission, visual acuity was nonexistent on the right and 14/21 on the left. Ocular tensions were 11 mm Hg on the right and 15 mm Hg on the left. There was a right afferent pupillary reflex defect. Ophthal-
mosplethysmography showed ophthalmic systolic pressures of 123 mm Hg on the right and 120 mm Hg on the left. Brachial systolic pressures were 136 mm Hg on the right and 142 mm Hg on the left.

Case 3
While driving a car 2 days before her admission, a 70-year-old woman suddenly lost vision in her right eye. Two hours later, she was seen by an ophthalmologist in her home community, and a tentative diagnosis of central retinal artery occlusion was made. Treatment was attempted with inhalation of carbon dioxide and ocular massage for 3 hours. The patient regained some right temporal vision after this treatment, but central vision did not return.

Her examination 2 days later at the Mayo Clinic revealed only vague light perception in the right temporal field of the right eye. The left visual acuity was 14/21. Ocular tensions were 18 mm Hg on the right and 23 mm Hg on the left. Ophthalmoscopic examination of the right eye revealed a pale gray retina with extremely narrowed arterioles and a macular cherry red spot. The left eye was normal ophthalmoscopically. Ophthalmodynamometry revealed retinal artery pressures of 150 mm Hg on the right and 142 mm Hg on the left. Ocular pneumoplethysmography was normal. Patient 3, the presumed mechanism of the central retinal artery occlusion likely involved local hypertensive or atherosclerotic changes (or combinations of these), as well as occlusion of the vessel from embolism or aortic insufficiency, a calcified mitral valve anulus, and mild left ventricular hypertrophy. The patient was treated with aspirin and dipyridamole. No further symptoms have occurred during the past 9 months.

Discussion
All four patients demonstrated typical clinical symptoms and classic ophthalmologic signs of central retinal artery occlusion. In each patient, the ipsilateral retinal artery pressure, as measured by ophthalmodynamometry, was greatly reduced whereas the ophthalmic systolic pressure, as measured by ocular pneumoplethysmography, was normal.

The pathogenesis of central retinal artery occlusion varies widely and includes vaso-obliteration due to local central retinal artery inflammatory, hypertensive, or atherosclerotic changes (or combinations of these), as well as occlusion of the vessel from emboli from cervical artery or cardiac sources. Patients 1 and 2 had long-standing hypertension historically, without identifiable embolic sources or inflammatory disease, and hence the mechanism of the central retinal artery occlusion likely involved local hypertensive or atherosclerotic (or both) changes. In patient 3, the presumed mechanism for central retinal artery occlusion was embolization from the ulcerated atherosclerotic lesion of the ipsilateral internal carotid-common carotid system. The stenosis of this lesion was not sufficient to reach pressure significance (greater than or equal to luminal area stenosis of 75%), and therefore the results of ocular pneumoplethysmography were normal. Patient 4 had a potential cardiac embolic source (aortic and mitral valvular abnormalities) as well as long-standing hypertension, and either could be implicated as the underlying mechanism.

The two most frequent sites of central retinal artery occlusion are where the artery enters the dura of the expected a "blockage of a blood vessel." Past medical history included long-standing diabetes melitus and hypertension but was otherwise unremarkable.

Examination at the Mayo Clinic revealed a right eye visual acuity of finger counting at 4 feet and a left eye acuity of 14/28. Ocular tensions were 20 mm Hg bilaterally, and an affenter pupillary reflex defect was noted on the right. Ophthalmoscopic examination of the right eye revealed a pale retina with a decreased caliber of the arterioles and no apparent blood flow. The left eye was normal ophthalmoscopically, without evidence of background diabetic or hypertensive retinopathy. Ophthalmodynamometry revealed retinal artery pressures of 0/0 mm Hg on the right and 101/30 mm Hg on the left. Ocular pneumoplethysmography showed ophthalmic systolic pressures of 125 mm Hg bilaterally, with brachial systolic pressures of 188 mm Hg on the right and 186 mm Hg on the left. Cardiac examination revealed a grade 3-4/6 rough systolic ejection murmur at the base, which extended to the apex and the carotid arteries bilaterally. There was a grade 2/6 diastolic murmur over the left lateral sternal border. A cardiac sector scan revealed evidence of mild to moderate aortic stenosis and aortic insufficiency, a calcified mitral valve anulus, and mild left ventricular hypertrophy. The patient was treated with aspirin and dipyridamole. No further symptoms have occurred during the past 9 months.
optic nerve sheath and at the lamina cribrosa. In both instances, the pressure in the distal retinal vessels becomes greatly reduced or unmeasurable, producing abnormality on ophthalmodynamometry. However, the medial and lateral posterior ciliary arteries, which supply approximately 90% of the total ocular blood, have origins in the ophthalmic artery that are separate from the central retinal artery. Consequently, these vessels remain intact in central retinal artery occlusion, allowing normal eye pulsation and normal ocular pneumoplethysmographic results. Thus, abnormal ophthalmodynamometric results (particularly when the values are 0) in the presence of ipsilateral normal ocular pneumoplethysmographic results are strongly suggestive of central retinal artery occlusive disease. Conversely, when both ophthalmodynamometric and ocular pneumoplethysmographic results are normal, the diagnosis of complete central retinal artery occlusion should be seriously questioned. If the clinical setting is typical for central retinal artery occlusion and both ocular pneumoplethysmographic and ophthalmodynamometric results are abnormal, a separate pressure-significant lesion of the internal carotid system probably exists proximal to the central retinal artery and may have served as an embolic source.

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**References**


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**Pattern Difference of Reversed Ophthalmic Blood Flow Between Occlusion and Stenosis of the Internal Carotid Artery**

An Ultrasonic Doppler Study

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**SUMMARY** Reversed ophthalmic blood flow in the occlusions (33 patients) and stenoses (11 patients) of the internal carotid artery (ICA) was examined using the ultrasonic Doppler technique. The Doppler shift frequencies of the blood flow signal were analyzed to obtain their sound spectrogram. In stenosis of the ICA, "presystolic notch" was more frequently observed and "d/S" value (S, d; maximum blood flow velocity at systolic and diastolic) was smaller than in occlusion.

These two characteristics of stenosis distinguish it from occlusion with 89% accuracy although this method is applicable only for the patients with reversed ophthalmic blood flow.

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REVERSED BLOOD FLOW in the ophthalmic artery is frequently detected by the ultrasonic Doppler flowmeter in patients with occlusion or stenosis of the internal carotid artery (ICA).

Reports suggest that obstructive lesions of the ICA may be diagnosed noninvasively by detection of the reversed ophthalmic collateral flow.

A few authors have reported the possibility of the differential diagnosis between occlusion and stenosis of the ICA with the use of the Doppler techniques. No author has discussed the differences in patterns blood flow in the ophthalmic artery itself as a distinguishing feature. The purpose of this report is to clarify the rheological differences of reversed blood flow in the ophthalmic artery per se between the occlusion and the stenosis using an ultrasonic Doppler method and then to describe the possibility of the differential diagnosis.

**Subjects and Methods**

Consecutive patients with 54 occlusions and 62 stenoses of the ICA admitted from June 1976 to October 1979 were all examined both by cerebral angiography and ultrasonic Doppler studies. The ultrasonic Doppler examination was performed after angiography except for 15 patients. Among them, 33 patients with carotid occlusion (21 were men, and 12 were women, the average age was 63.6 years old) and 11 patients with stenoses of the ICA (9 were men, and 2 were women, with an average age of 69.3 years old.) were

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