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.

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. The patient's 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. SI 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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the platelet count was 352,000/mm³; the prothrombin time was 10.3 s; the partial thromboplastin time was 22.8 s; the erythrocyte sedimentation rate (Westergren) was 20 mm/hr. The electrocardiogram and chest x-ray were normal.

The patient was submitted to cardiac catheterization for evaluation of what was considered to be atypical chest pain. A #7 French pigtail catheter was introduced percutaneously into the right femoral artery and was advanced retrograde across the aortic valve into the left ventricle. The aortic blood pressure was 170/80. Heparin 2000 U. was administered intravenously. Left ventriculography and coronary angiography were completely normal. Ergonovine maleate 0.05 mg was then administered intravenously. This initial dose did not produce symptoms, and 3 minutes later a second dose of 0.1 mg was administered. The blood pressure rose transiently to 200/95. The patient, who had been experiencing her usual headache throughout the procedure, suddenly complained of a more severe headache and then became unresponsive. She remained unresponsive for about 60 seconds, and during this period it was noted that her eyes were deviated to the right. When consciousness was restored, sublingual nitroglycerin was administered. The procedure was terminated, and upon returning to her room the patient was obeying commands, moving all four extremities and complaining of a severe headache and nausea.

About 5 hours after the catheterization the patient was found to be obtunded. She opened her eyes to loud verbal stimuli. Her speech was unintelligible, and she obeyed no commands. There was a right hemiparesis affecting the face and upper extremity more than the lower extremity. Right-sided hyperreflexia was noted, and the plantar response was extensor on the right. A computed tomographic (CT) scan of the brain without enhancement demonstrated a large frontoparietal intracerebral hematoma on the left with apparent rupture into the ipsilateral subdural space (fig. 1). The epicenter of the hematoma was in subcortical white matter, and it did not involve the basal ganglia. A left common carotid angiogram demonstrated only depression of the left Sylvian point and a round shift of the anterior cerebral artery from left to right; no abnormal vessels were seen, nor were there any intraluminal filling defects. Because of the patient's declining level of consciousness and because of the mass effect evident on the CT scan, an emergency craniotomy was performed for evacuation of the intraparenchymal and subdural hematomas. The hematoma cavity was carefully inspected for tumor or vascular malformation. No such lesion was seen, and biopsies were taken from the wall of the cavity and from the cortical surface where it had been breached by the hematoma. Upon awakening from anesthesia the patient obeyed commands, and her subsequent postoperative course was unremarkable.

Figure 1. A CT scan without enhancement through the level of the frontal horns showed a crescentic, high-density, extracerebral collection over the left hemisphere with swelling of the underlying brain and shift of midline structures (left). At a higher level was a globular, high-density mass abutting the cortical surface on the same side as the extracerebral collection (right). At operation a large intraparenchymal hematoma was found to have ruptured into the subdural space.
At the time of discharge her neurological examination showed an anomic dysphasia and a monoparesis of her right upper extremity. Microscopic examination of the biopsies showed no tumor. The blood vessels included in the specimen were normally formed. There was no amyloid or inflammatory infiltration of the vessels; Congo red staining was negative.

**Discussion**

Several different neurologic syndromes can complicate cardiac catheterization, and specific diagnosis is necessary for intelligent management. Angiographic contrast material can by itself cause alterations of consciousness, seizures and focal neurologic deficits such as cortical blindness.8,11 These reactions are often transient, and with modern contrast agents they are now very infrequent. Hypotension and dehydration can precipitate thrombosis of a major cerebral vessel.8,11 More commonly there is embolization of the cerebral circulation as a consequence of catheter-related mishaps: thrombus, atheroma and air have all been implicated in the generation of transient or permanent focal neurologic deficits in the course of cardiac catheterization.8-14 To this list of complications affecting the central nervous system, massive intracerebral hemorrhage must now be added.

In this case the intracerebral hemorrhage appeared to arise in subcortical white matter without involving the basal ganglia or thalamus. These characteristics define a "lobar" hemorrhage.15,16 It is generally thought that patients with lobar hemorrhage have a history of hypertension less frequently than patients with hemorrhage in other locations.15-18 One autopsy series reported that in less than 8% of patients with lobar hemorrhage was hypertension the sole predisposing condition; the comparable figure for ganglionic hemorrhage was 48%.19 Coagulopathy and blood dyscrasia, angioathy, tumor, angiitis, aneurysm and vascular malformation account for variable portions of clinical series of spontaneous lobar hemorrhages, but in the remaining 27-38% no etiology can be determined.15-17

There was no evidence for chronic hypertension in this case, but it seems reasonable to suspect that transient arterial hypertension caused by the discomfort of the procedure and by the infusion of ergonovine20-23 may have acted in conjunction with some other underlying condition to produce the consequent stroke.

There was no evidence in the admission laboratory data to suggest that our patient had a preexisting coagulopathy or blood dyscrasia, but she did receive heparin 2000 U. at the start of the procedure. Walker and associates studied the effects of comparable doses of heparin in this setting and found prolongation of the Lee-White clotting time 2 to 10 times normal for the duration of the catheterization.24 Nevertheless, the use of heparin to inhibit thrombus formation on the catheter and guidewire has become a standard technique and has been associated with infrequent, clinically significant hemorrhage.

The remaining recognized causes of lobar intracerebral hemorrhage were investigated appropriately and can with reasonable assurance be eliminated from consideration. Microscopic examination of the biopsy of the wall of the hematoma cavity provided no support for diagnoses of amyloid angiopathy, angiitis or neoplasia. An underlying vascular malformation was sought by angiography, by careful inspection of the hematoma cavity and by biopsy; none was found.

It is clear that the information available does not permit assigning our patient’s intracerebral hemorrhage to a certain cause, and logic compels consideration of the possibility that this complication was a coincidence, a "pseudocomplication".1 Against this consideration is the patient’s utter lack of predisposing conditions and the conjunction of two iatrogenic stresses: transient hypertension and anticoagulation. These stresses are common concomitants of cardiac catheterization with ergonovine administration. The frequent performance of this procedure and the apparent absence from the literature of previous reports of massive ICH complicating it testify to its relative safety. However, our experiences with this case indicate that patients who suffer neurological changes after cardiac catheterization require prompt evaluation including a CT scan. Not all such patients are the victims of cerebral embolism; some have suffered massive intracerebral hemorrhage and may require immediate neurosurgical attention.

**Acknowledgments**

I wish to thank Dr. Wesley A. Cook, Jr. for permitting me to report his patient and Dr. Peter C. Burger for performing special stains of the biopsy specimens.

**References**


The Terminology of Transient Visual Loss Due to Vascular Insufficiency

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SUMMARY Transient visual loss due to cerebro-ocular vascular disease is a common symptom. The purpose of this paper is to present a unified terminology of the monocular vs. binocular or homonymous types. Lack of proper identification may lead to mis-diagnosis and improper management of these entities.

Monocular blurred vision must be investigated since its origin is so commonly due to atherosclerosis of the carotid system. Binocular blurred vision due to vertebral-basilar insufficiency is managed conservatively in almost all instances.

IT IS UNIVERSALLY acknowledged that the most reliable indicator of impending ischemic stroke is a transient ischemic attack.1 The semantics of the ocular symptomatology have become confusing and are often merely referred to as transient visual obscurations. It is the purpose of this communication to attempt to clarify this matter.

The first distinction one must make in describing the ocular symptoms is whether they are monocular or binocular. The monocular attacks are designated as amaurosis fugax and transient monocular blindness and are due to vascular insufficiency in the optic nerve and retina, whereas transient binocular blindness indicates involvement of the posterior visual pathways. Secondly, one should ascertain the duration of the attacks. Accompanying neurologic signs or symptoms provide additional evidence as to the localization of the ischemic process in the visual system.

Amaurosis fugax is a monocular fleeting attack of partial to total (rare) blindness lasting from seconds to a few minutes. It is usually considered to be due to emboli but may be due to a perfusion deficit at the nerve head as in papilledema or incipient anterior ischemic neuropathy. The word obscurations means very fleeting loss of vision (few seconds). Transient visual obscurations may be most aptly applied to this phenomenon seen with papilledema; thus, as in pseudo-tumor cerebri especially and rarely in brain tumor, these attacks can be characteristic and associated with headache and other neurologic signs or symptoms.

Transient Monocular Blindness is an episode of transient visual obscurations longer in duration than amaurosis fugax which may be partial or total. Partial blindness may take the form of a shadow coming from below up, from above down, or from the side (usually temporal). It may progress towards fixation and then regress. A "shutter" effect may constrict the field and then disappear. These attacks may last many minutes, hours and rarely several days. These episodes are probably related to hemodynamic changes such as in hypertension or some hematologic cause. Migraine is an uncommon cause of monocular visual loss. Patients with bilateral carotid disease causing orbital hypoxia either due to retinal or optic nerve hypoperfusion can get transient visual loss but this is not simultaneous or binocular.

Transient Binocular Blindness is usually due to cerebrovascular ischemia affecting the calcarine cortex. It is bilateral and involves either the homonymous field, the inferior or superior altitudinal fields or the central fields. These episodes usually last 5 to 30 minutes and occasionally as long as several hours. They may exact-
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Stroke. 1984;15:904-907
doi: 10.1161/01.STR.15.5.904

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