

Arteriography of Cerebrovascular Disease*

FREDERICK S. VINES, M.D.

Associate Professor of Radiology and Neurology; Chief, Section of Neuroradiology, Medical College of Virginia, Health Sciences Division of Virginia Commonwealth University, Richmond

Advances in reconstructive vascular surgery have increased the importance of accurate angiographic demonstration of the extracranial vascular tree. The limitations of the clinical examination are well recognized and make it essential that complete angiography be obtained prior to contemplated surgery. The locus of cerebral involvement may be determined clinically, but the site and extent of vascular disease in a surgically accessible location must be confirmed. The role of the angiographer is, therefore, twofold: Firstly, he must be able to safely perform complete angiography with adequate evaluation of the multiple sites of possible involvement; secondly, he must be aware of the pathophysiological changes which may occur and recognize the techniques and pitfalls he may encounter in delineating these changes. The present communication will describe the more common findings observed during angiography, along with some pitfalls the angiographer and clinician may encounter.

The example of carotid stenosis in Figure 1A certainly would be considered by most to be a surgical candidate with a remaining internal carotid lumen of less than 1 mm; however, the patient shown in Figure 1B, with smooth carotid artery stenosis narrowing the lumen by approximately 60%, may not be a candidate for operative intervention. Extracranial surgery was originally performed based on a theory of decreased cerebral blood flow, relative to an arbitrary degree of narrowing of the proximal arterial lumen. Thus, factors such as hypotension or cardiac arrhythmias which would result in decreased flow were considered to produce cerebral

symptoms in the territory supplied by the stenotic vessel. This "stenotic theory" is now considered to play a minor role in the production of symptoms of cerebral ischemia (1). A significant change has occurred in recent years, so that now the patient with an "ulcerated plaque" and recurrent symptoms distal to this lesion is considered to be the prime candidate for extracranial carotid artery surgery. These ulcerated plaques are considered to be a source of cerebral emboli from clumps of blood elements or atheromatous materials which have developed from an area of irregularity and ulceration in a preexisting plaque. This explains the recurrent episodes of cerebral ischemia and the symptoms that may be present with a minor plaque or stenosis. Several examples of ulcerated plaques are seen in Figure 2. It is imperative that the carotid artery be evaluated carefully in multiple angulations, since these ulcers may at times be quite small and seen in only one projection. It is true, however, that most of the atherosclerotic disease and subsequent ulcer formation is on the posterior wall and the critical view is a lateral projection.

Another common problem in evaluating the extracranial carotid artery is the presence of an "irregular" plaque. This is a somewhat arbitrary designation and indicates only that there is no single focal area of ulceration (Fig. 3). The angiographer must be completely aware that the ulceration itself, however, may be filled with debris at the time of the contrast injection and should not be surprised if the pathological sample shows a much more irregular and ulcerated lesion. Additionally, a perfectly smooth carotid stenosis angiographically may at the time of surgery have a focal area of ulceration. Some authors consider that an irregular plaque cannot be differentiated from an ulcerated plaque

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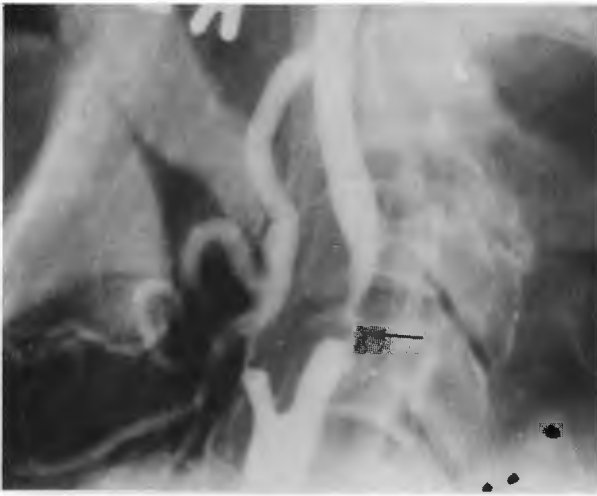


Fig. 1A—Severe internal carotid artery stenosis.



Fig. 1B—Moderate internal carotid artery stenosis with smooth borders.



Fig. 2A, B—"Ulcerated" internal carotid artery plaques. The atherosclerotic plaque projects into the arterial lumen with the collection of contrast in the "ulcer" crater (arrow).



Fig. 3—Irregular internal carotid artery plaque. There is mild stenosis with irregular borders of the contrast posteriorly.

cation in at least three views—the frontal, lateral, and oblique. The angiographer should also have a low threshold for performing additional views if necessary for adequate evaluation.

Unfortunately, the presence of an ulcerated plaque does not indicate that this is the cause for the patient's symptoms. We are certainly observing "asymptomatic" ulcerated plaques in a large number of cases studied for other clinical problems. Conversely, there may be distal vascular lesions that are equal in importance to the bifurcation lesion. The second most common site of carotid atherosclerotic disease is its cavernous portion. Severe stenosis and ulcerations at this site may preclude surgical repair of the cervical carotid artery. One may also see stenosis of the middle cerebral artery or proximal anterior cerebral artery that would limit surgical therapy. Careful subtraction may be necessary when the vessel is not well delineated and free from the dense bony structures at the base of the skull. The small size of these ulcers and overlapping of vessels, which occurs with

arch aortography, also severely limit its use alone to evaluate patients suspected of having vascular lesions.

Others have commented on the frequency of other pathological findings in the intracranial circulation of a patient studied for clinically suspected extracranial vascular disease. With more sophisticated brain scanning techniques and ancillary tools of clinical examination in recent years, however, the incidence of this observation is certainly lower than the previous reports of 5%. We do suggest routine angiography of the intracranial as well as the extracranial circulation, therefore, but for evaluating the distal vascular bed rather than for primary evaluation of an unsuspected subdural hematoma or neoplasm.

Complete occlusion of the internal carotid artery is now considered by most surgeons to be a nonoperable condition (3). The incidence of intracranial hemorrhage following reestablishment of circulation after an acute occlusion has made this procedure hazardous, and the poor success rate in reestablishing circulation in a chronically occluded lesion has made most surgeons unwilling to attack this lesion. There are, however, cautions of which

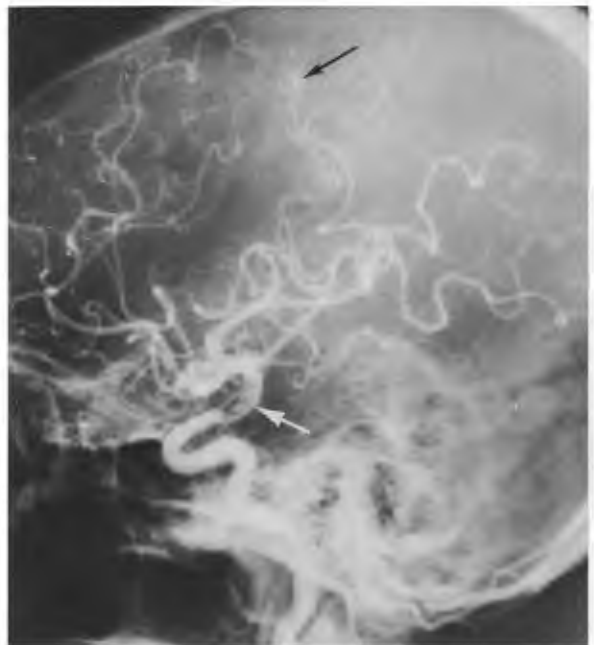


Fig. 4—Distal internal carotid embolus (white arrow). An area of infarction with arteriovenous shunting is present (black arrow). Avascular areas of branch occlusions are present in front of and behind the area of luxury perfusion.

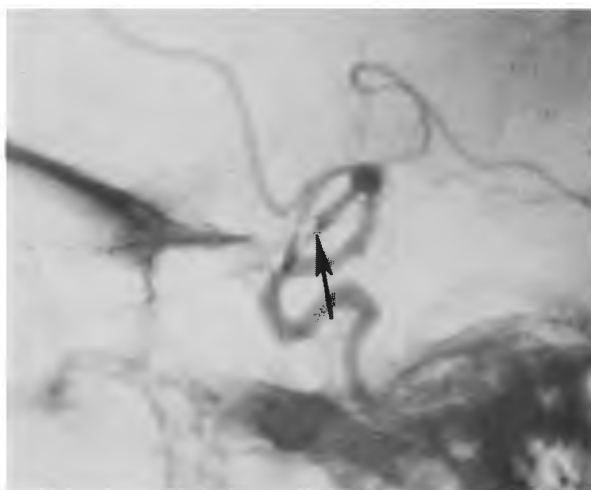
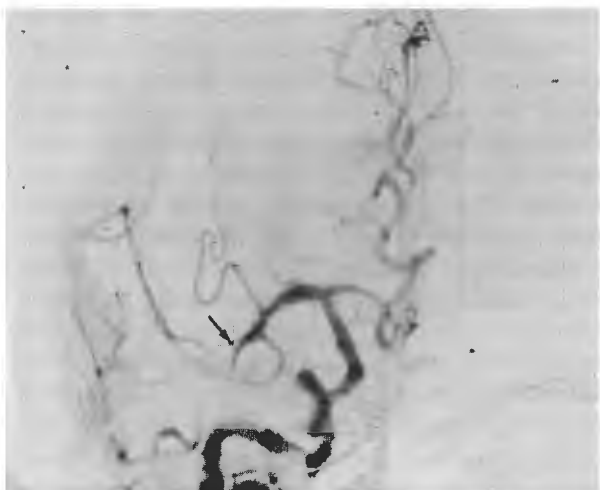


Fig. 5A, B—Frontal and lateral views of major branch occlusion of the middle cerebral artery (arrow).

one must be aware when evaluating what appears to be complete occlusion of the cervical portion of the internal carotid artery. The typical occluded internal carotid artery will obtain its intracranial filling via the opposite anterior cerebral artery or by the supraorbital branches of the ipsilateral external carotid, filling the ophthalmic artery and cavernous carotid in a retrograde manner. "Pseudo-occlusion" of the internal carotid artery may occur, however, when the stenosis is so severe that only a trickle of contrast extends beyond the obstruction, even though the internal carotid lumen immediately distal to the stenosis is normal. This faint linear accumulation of contrast may be obscured by over-

lying external carotid branches and may not reach the supraclinoid carotid for several seconds. Therefore, every apparent internal carotid "occlusion" should have a series prolonged for 10–15 seconds with a careful search for any patency into the skull.

The significance of cerebral emboli from other sources is also assuming increased clinical importance. Certainly most of the emboli will arise from a cardiac valve or an endocardial surface but increasing interest is developing regarding the possibility of emboli from the pulmonary veins. Large emboli may obstruct the carotid artery itself and this possibility must be carefully evaluated whenever there is obstruction other than at the bifurcation

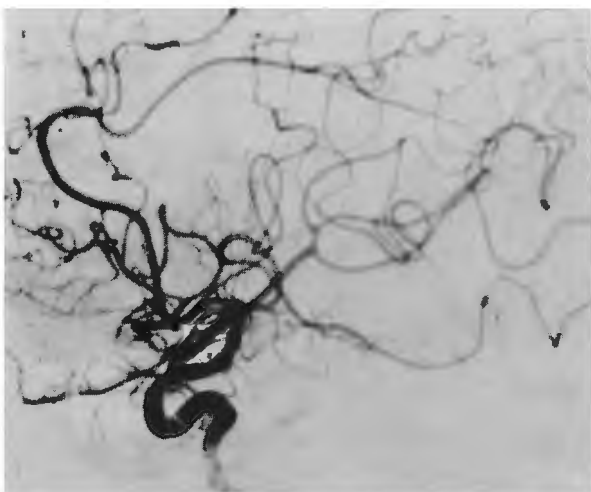
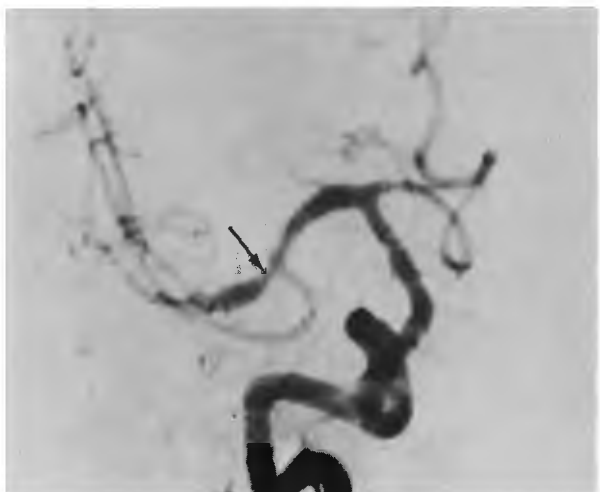


Fig. 5C, D—Frontal and lateral view of recanalization of this occlusion. Minor narrowing of the lumen is still present (arrow). An avascular area of a peripheral branch occlusion persists in frontal operculum.



Fig. 6A—Basilar artery occlusion (black arrow). Collateral filling of superior cerebellar arteries has occurred via cortical anastomosis with the posterior inferior cerebellar arteries.

or in the cavernous carotid, for example, the common carotid or the midcervical carotid. These emboli may also lodge in the intracranial vascular tree, creating a flow deformity (Fig. 4) with re-



Fig. 6B—Embolic basilar artery occlusion (arrow).

current symptomatology as fragments of the embolus migrate distally. An area of "luxury perfusion" is seen peripheral to the obstructed frontal opercular branch with a poorly defined stain and early venous filling. This patient developed further left hemisphere symptoms, and pathological confirmation was obtained of large hemorrhagic infarction involving most of the left hemisphere. Not all emboli result in this course, however. The patient in Figure 5 developed acute occlusion of the middle cerebral artery, two of the redundant trifurcation branches being occluded. Subsequent repeat angiography in several days, however, shows that this middle cerebral artery has recanalized and only a single frontal branch is now occluded. This illustrates a major reason for the frequent normal angiographic findings in the stroke syndrome. Emboli may be rapidly lysed and may migrate distally, allowing a positive angiographic diagnosis to be obtained only in the first few hours after the onset of clinical symptoms.

Basilar artery occlusive disease is a more serious problem, usually with a fatal outcome. Recent clinical data indicate, however, that some of these people survive with a reasonably minor neurological deficit. The patient in Figure 6A developed signs of severe upper midbrain dysfunction, with angiographic findings of complete occlusion of the basilar artery just below the origin of the superior cerebellar arteries and retrograde filling via the posterior inferior cerebellar arteries. A recent case illustrates a surgically altered course with a large basilar artery embolus (Fig. 6B). There is a filling defect ob-

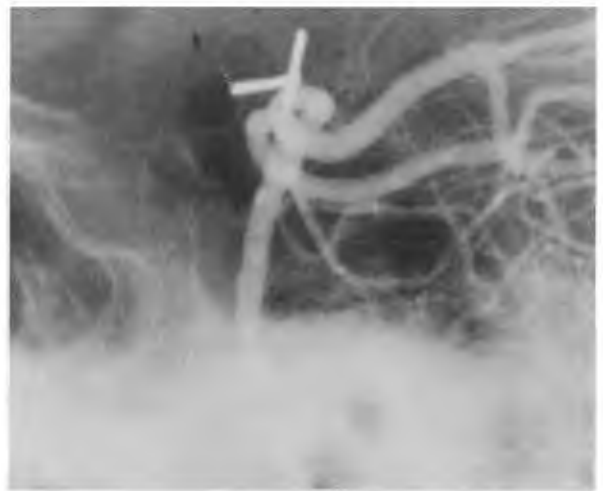


Fig. 6C—Postoperative patency of the basilar artery and its branches.

structing the distal basilar artery with markedly delayed flow. This view was obtained nine seconds following the injection of contrast. Collateral filling of the superior cerebellar arteries occurred via the posterior inferior cerebellar arteries. There was no filling of the posterior cerebral arteries or the dome of the basilar artery from either carotid injection or from collateral vessels. The patient underwent surgical removal of a large basilar artery embolus that was extending into both posterior cerebral arteries. The postoperative arteriogram (Fig. 6C) shows restoration of patency of the basilar artery and posterior cerebral arteries. This procedure did not alter the ultimate outcome of the patient but does indicate the development of surgical techniques and procedures which allow the performance of this embolectomy.

Summary. The importance of complete angiographic studies in patients suspected of extracranial vascular disease has been emphasized. Examples of a variety of lesions of the intracranial

and extracranial circulation have been shown. It is important that the angiographer obtain adequate visualization of the circulation cephalad to the aortic arch with awareness of the type of lesion and technical problems that may be encountered. It is only in this way that further understanding of the "stroke syndrome" can be attained with appropriate therapy.

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