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THE IMPORTANCE OF COMPLETE ANGIOGRAPHY IN CEREBROVASCULAR INSUFFICIENCY SYNDROME*

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Atherosclerotic disease of the carotid vessels in the neck is being diagnosed with greater frequency since Sjöqvist (1936)[1] and Moniz (1947)[2] first reported on the angiographic findings in such cases. Neurosurgeons are finding carotid occlusion or segmental partial occlusion with increasing frequency as a result of more frequent and more complete angiographic studies of patients having symptoms and signs of cerebrovascular disease. More complete pathological studies at autopsy are confirming the presence of atherosclerotic or narrowings in the neck vessels and clarifying the clinical syndrome produced. Physicians are now aware that this disease has many neurological manifestations from vague transitory sensory symptoms, primarily in the face and hand, to profound permanent hemiplegia.

Gowers (1875)[3], Guthrie and Mayou (1908)[4] recorded cases of unilateral blindness associated with contralateral hemiplegia. Hunt (1914)[5] described the role of the carotid arteries in the causation of vascular lesions of the brain and stressed the importance of palpation of the carotid artery in the neck as a means of diagnosing disease of the carotid artery. Fisher (1951)[6] pointed out the frequency of carotid artery occlusion as a causation of cerebrovascular accident and raised the question of surgical therapy for this condition. Johnson and Walker (1951)[7] reviewed the literature and found 101 angiographically proven cases of thrombosis of the carotid artery and reported six cases of their own.

Diagnosis of internal carotid artery occlusion or stenosis cannot be made with absolute certainty on purely clinical evaluation. However, the following signs, or maneuvers, together with an accurate history of intermittent episodes, makes the diagnosis reasonably tenable.

PALPATION OF THE CAROTID ARTERY in the neck may reveal a complete absence of the pulse or decreased pulsation in the entire vessel on one side of the neck. However, palpation of the internal carotid artery at the bifurcation, or in the tonsillar fossa, is unreliable because it is not possible to separate the pulses of the internal and external carotid arteries.[8,9] In addition, a good pulse may be felt in the presence of internal carotid thrombosis due to the thrust of the common carotid artery with each pulsation.

With atherosclerotic disease at the bifurcation, a SYSTOLIC BRUIT may often be heard on auscultation. Frequently this is due to a segmental stenosis or actual occlusion of the internal carotid artery but it can also be due to diffuse disease with increased tortuosity and roughening of the vessel walls.

Recently the use of OPHTHALMODYNAMOMETRY[10,11] to measure the blood pressure of the retinal arteries, has been shown to be helpful. In cases of complete or partial occlusion of the carotid artery the retinal artery pressure on the involved

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side is usually lowered to approximately one-third or one-half of the pressure in the opposite eye. This procedure, while useful, is subject to some error of interpretation.

Another maneuver of considerable importance has recently been popularized by Gurdjian and Webster. [12,13,14] This consists of COMPRESSION OF THE COMMON CAROTOID ARTERIES below the bifurcations. In the presence of thrombosis of the anterior cerebral artery or the internal carotid artery on the contralateral side, or of the vertebral basilar system, syncope or convulsions may occur within 15 to 20 seconds. Compression in the presence of occlusion of the middle cerebral artery usually does not cause syncope. Compression is made well below the bifurcation to avoid the syncope associated with hypersensitivity of the carotid sinus. Slow wave changes in the electoencephalogram usually coincide with the onset of syncope.

At present, the exact incidence of carotid occlusion and stenosis is not known with certainty. Fisher's excellent work on this subject leaves little doubt that it is one of the common causes of cerebrovascular accidents. In 432 routine, unselected autopsies in adults, Fisher[6,15] found occlusion or severe stenosis of one or both internal carotid arteries at the bifurcation in the neck in approximately ten percent of cases. Furthermore, a clinical pathological study of 45 cases of occlusion of the internal carotid artery at the bifurcation revealed that in 85 percent severe neurological disturbances were produced. Lofstrom, Webster and Gurdjian[16] found internal carotid artery occlusion or stenosis to be present in 29 of 100 consecutive cases of hemiparesis or hemiplegia.

More recently, studies by Crawford, DeBakey and Fields,[17], using percutaneous subclavian angiography, have shown that stenosing lesions may also be present at the origins of the carotid and vertebral vessels.

In our own experience this is by far less common than stenosis at the bifurcation in the neck and also is more difficult to correlate with the patient's clinical picture. In fifty consecutive subclavian arteriograms, we have found only three lesions at the origin of the vertebral artery which can be considered as having surgical significance. The remainder have shown no abnormality or diffuse sclerosis with no localized stenosis at the origin of the vessels. (FIG. 1).

In spite of all the clinical methods available for diagnosing vascular lesions in the major vessels leading to the brain, accurate diagnosis as to degree and site of occlusion cannot be made without complete angiographic studies. Indeed, symptomatology suggestive of vascular insufficiency due to occlusive disease, may occasionally be very misleading and if not verified by angiography result in improper treatment. We have personally had experience with cases of brain tumor, brain abscess, and subdural hematoma which clinically simulated episodes of vascular insufficiency as manifested by intermittent episodes of hemaparesis or other neurologic symptoms or signs. Since the use of anticoagulant medication is a popular method of treating the symptoms presumed to be due to vascular insufficiency, it is doubly important that this diagnosis be established by complete angiographic studies so that treatment may be carried out in a more intelligent manner. For instance, a short segmental stenosis of the internal carotid artery with essentially normal appearing distal branches of this vessel is best

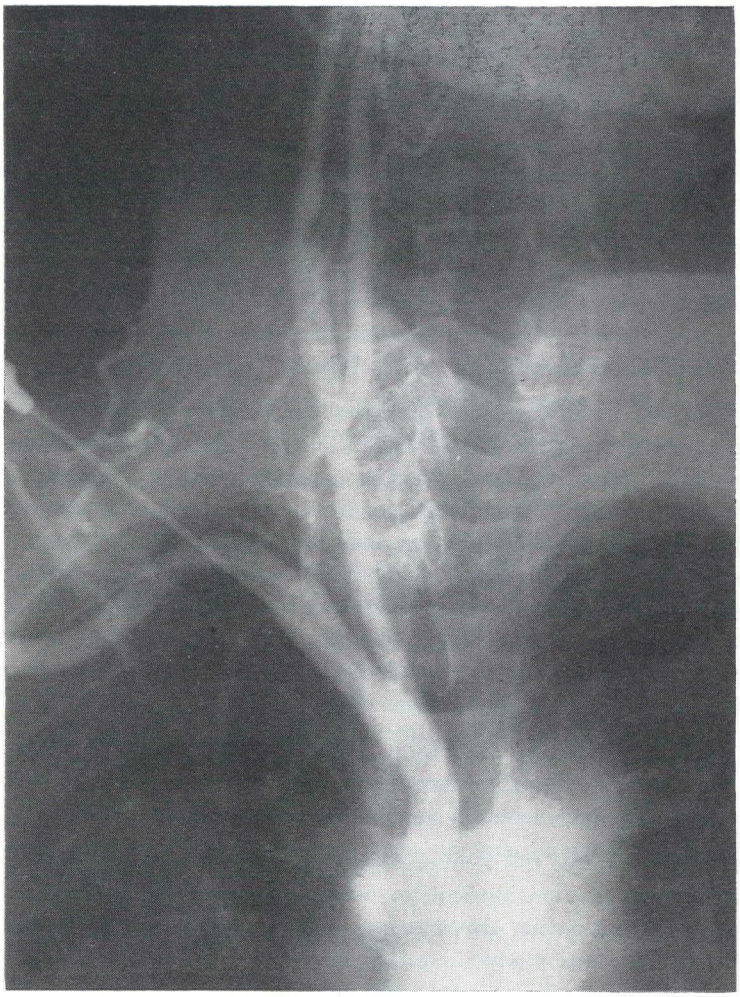


Figure 1

Subclavian arteriogram showing stenosing lesion at the origin of the right vertebral artery as well as stenosis of the internal carotid artery above the bifurcation.

treated by endarterectomy, whereas a more diffusely involved vessel producing the same symptomatology might better be suited to anticoagulant therapy.

At present, it is our opinion that with some exceptions, patients who have had a cerebrovascular accident, or symptoms suggesting cerebrovascular insufficiency, should have arteriographic studies as soon as the patient's condition will tolerate the procedure. Obvious contraindications would be those patients in preterminal state with massive cerebral hemorrhage or infarctions. Age and general health particularly cardiac status, diabetes, etc., are also factors to be considered. The intracranial circulation, the bifurcation of the carotid arteries in the neck, and the origins of the carotid and vertebral vessels, are studied by various combinations of aortic, subclavian and carotid arteriography. Stenosing or occlusive disease is found to be far more common

at the carotid bifurcations than it is at the origins of these vessels and is said to be more common than in any other vessel except the abdominal aorta.[18]

While there is certainly some risk to complete angiographic study, its relative safety is demonstrated by the accompanying table showing the complications occurring

Complications following arteriography in 1822 injections in 540 patients.

Case	Age	Diagnosis	Arteriogram	Complication
1	56	Metastatic brain tumor	Carotid	Became less responsive temporarily following procedure.
2	41	Metastatic brain tumor	Carotid	Generalized seizures.
3	48	Parasellar meningioma	Carotid	Aphasia and hemiparesis which cleared up in three days.
4	60	Diabetes mellitus internal carotid aneurysm	Carotid	Hemiparesis lasting thirty minutes.
5	54	Subarachnoid hemorrhage	Carotid	Seizures — Aphasia and hemiplegia lasting 10 minutes.
6	58	Arterial hypertension Cerebral arteriosclerosis	Carotid	Transitory hypertension.
7	60	Carotid sinus sensitivity Arterial Hypertension	Carotid	Syncope after injection and hemiparesis which lasted 3 days.
8	46	Convulsive disorder	Carotid	Seizures followed by hemiplegia which recovered completely after 30 minutes.
9	38	Hypertensive cardiovascular disease	Carotid	Transient episode of confusion.
10	47	Metastatic brain tumor	Carotid	Patient became less responsive and comatose and expired the following day. Metastasis and marked brain edema at the autopsy.
11	42	Subdural hematoma	Carotid	Transient dysarthria.
12	56	Internal carotid thrombosis	Carotid	Increased hemiparesis.
13	65	Subarachnoid hemorrhage Diabetes mellitus Arterial hypertension	Carotid	Previous right carotid arteriogram with no untoward effects. Following injection patient became semicomatose and developed right hemiplegia, returning to the prearteriogram status in 3 weeks.
14	51	Anterior cerebral aneurysm	Carotid	Transitory hypertension.
15	56	Metastatic brain tumor	Carotid	Hypotension and transient hemiplegia.
16	62	Metastatic brain tumor	Carotid	Transitory hypertension.
17	63	Diabetes mellitus Cerebrovascular insufficiency	Carotid	Transitory state of drowsiness and dysphasia.
18	63	Metastatic brain tumor	Carotid	Focal motor seizures.
19	58	Internal carotid thrombosis	Carotid	Temporary dysarthria and expressive aphasia.
20	43	Anterior cerebral aneurysm	Carotid	After the second injection the patient became unresponsive, had divergent strabismus and bilateral Babinski sign, recovering completely in approximately one hour.
21	54	Internal carotid aneurysm	Carotid	Hemiplegia which cleared up in two days.
22	56	Arterial hypertension Cerebrovascular insufficiency	Vertebral carotid	24 hours after arteriography patient became less responsive and expired two days after. Autopsy showed occlusion of the internal carotid and cerebellar hemorrhage.
23	60	Cerebral arteriosclerosis Diabetes mellitus	Carotid	Patient became comatose and hemiplegic following the third injection. Expired nine days after. No autopsy.
24	46	Brain tumor—Glioma	Carotid	Transitory hypotension.
25	60	Diabetes mellitus Cerebral arteriosclerosis	Carotid	Patient became confused, dysarthric and hemiplegic, downhill course expiring eight days after. No autopsy.
26	58	Cerebrovascular insufficiency	Subclavian	Pneumothorax

in 1,822 injections of 50 percent Hypaque in 540 patients. It is interesting to observe that eight out of the twenty-six patients in whom complications occurred were harboring brain tumors and six of these were metastatic. The hypertensive, atherosclerotic, diabetic patient likewise seems to be a poor risk for angiography. These figures were accumulated from a series of 708 arteriograms on 540 patients between July 1956 and July 1959. 144 of these patients were suspected of having atherosclerotic vascular disease on the basis of clinical observation. In this group, only 80 showed positive angiographic evidence of this condition, the remaining being either negative or revealing other conditions. Of the 80 patients whose angiograms were positive, 26 had occlusion of one or both internal carotid arteries, and 14 had segmental partial occlusions. the remaining 40 positive showed either diffuse disease or occlusion of intracranial vessels. This corresponds closely with a series of 174 patients recently reported by Murphy and Miller[19] in which 43 showed either partial or complete occlusion.

While many cases of vascular insufficiency have been improved symptomatically by long term anticoagulant therapy, it seems more reasonable to attempt restoration of the circulation in those cases where angiographic studies show this to be feasible. The following two cases illustrate different problems where surgical treatment has seemed justified from the results obtained.

CASE SUMMARY

CASE 1-826291

49 year old white man admitted to Henry Ford Hospital April 7, 1956, with a four year history of failing vision in the left eye and progressive mental deterioration. In the year prior to his admission he had repeated episodes of confusion, in which he lost contact with his surroundings for ten to fifteen minutes. One week prior to his admission he had a sudden onset of temporary aphasia and confusion.

General physical examination was essentially normal. Chest clear. Heart sounds regular; no murmurs. Blood pressure 150/90. On neurological examination the patient, who was left-handed was oriented but somewhat obtunded. He had moderate expressive aphasia and right facial weakness. Systolic bruits were heard over both carotid arteries. The balance of the neurological examination was essentially normal. Laboratory tests showed blood count, urinalysis, fasting blood sugar, and NPN, within normal limits. Lumbar puncture showed normal pressure and clear, colorless spinal fluid, containing 86 mgms.% of total protein. The electroencephalogram was essentially normal. Chest and skull films were negative.

Bilateral carotid arteriograms performed three days after admission revealed thrombosis of both internal carotid arteries. On the left side there was no definite collateral circulation to the brain via the ophthalmic artery. The right side showed marked narrowing of the proximal portion of the external carotid artery, although the internal cerebral circulation filled via the ophthalmic artery on this side and the right vertebral artery.

On April 23, 1956 the right carotid artery was explored and a by-pass homograft was placed from the common carotid artery to the external carotid, by-passing the marked narrowing of the proximal portion of the external carotid artery. The patient

had a satisfactory postoperative course and was discharged on April 28, 1956.

He was admitted to the hospital one year later for progress carotid arteriograms which showed patency of the by-pass homograft and good intracranial filling through the ophthalmic artery. At that time his condition had improved definitely. He had only occasional episodes of loss of memory, lasting a few minutes, no visual difficulties, and his neurological examination was essentially negative except for the right facial weakness noted initially.

He was started on anticoagulants in September 1957 in spite of the fact that he was asymptomatic. He was last seen in June 1959 at which time his examination was essentially normal and he stated that he had been working a 12-hour day shift as a bulldozer operator. His memory was good and he had no significant symptoms. (FIGS. 2, 3, 4).

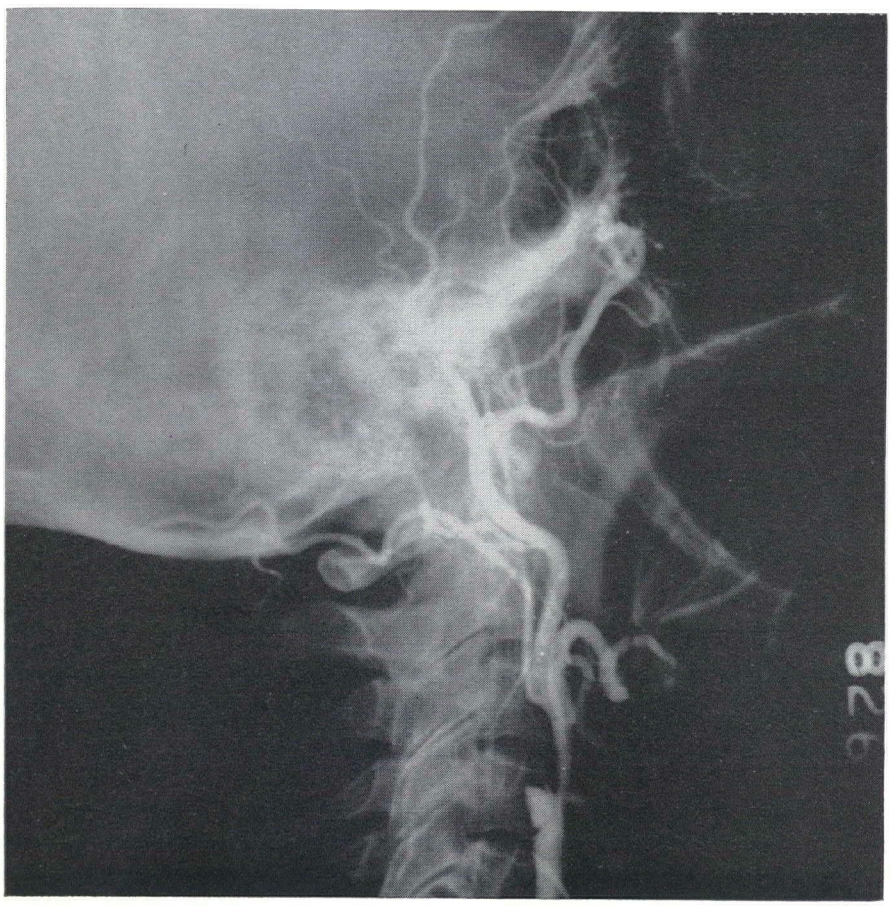


Figure 2

Case I — Right carotid arteriogram showing occlusion of the internal carotid artery above the bifurcation in the neck and narrowing of the proximal portion of the external carotid.

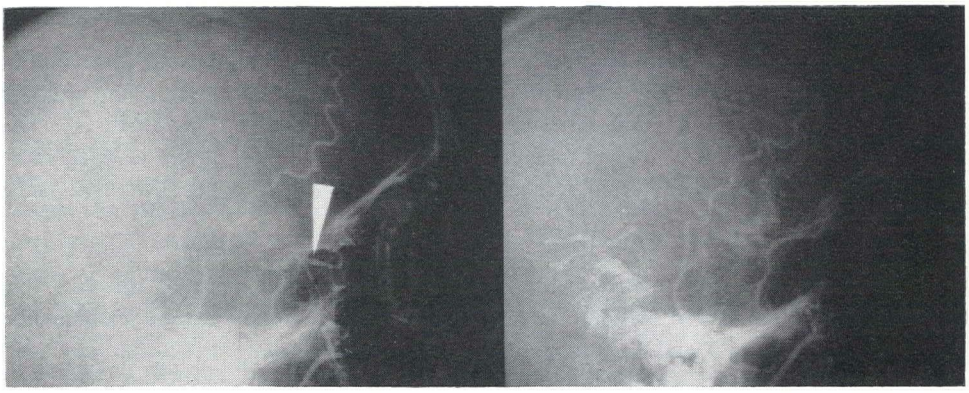


Figure 3

Same case showing filling of the intracranial circulation through the right ophthalmic artery (arrow).

COMMENT:

In this case with bilateral internal carotid occlusion, the intracranial circulation was maintained by the collateral circulation of the right external carotid into the ophthalmic artery as shown in Fig. 3. The proximal portion of the right external carotid showed marked narrowing. It was considered that in order to maintain adequate flow through the collateral system, by-pass of the stenosed area by means of a graft was indicated. The result obtained with this procedure has been satisfactory as evidenced by the patient's clinical course.

CASE SUMMARY

CASE 2-475548

58 year old white man admitted to Henry Ford Hospital on May 6, 1959. Two years before he had a "stroke" with paralysis involving the right side of the body, more so in the face and arm. The paralysis gradually cleared up entirely. Since then he has had recurrent episodes of "a curtain" dropping in front of his eyes, either one, or occasionally both at the same time. Two months prior to his admission he had an episode of complete blindness in the left eye for approximately 20 minutes and his vision gradually returned from the upper field down in 2 or 3 minutes. Since this latter episode he has had repeated transient episodes of blindness in the left eye.

General physical examination revealed a malnourished, chronically ill appearing man. Grade II arteriosclerotic changes were noted in the eye grounds. Lung and heart examination were normal. Blood pressure was 140/80. On neurological examination there was minimal central facial weakness on the right side, and slight, mild residual weakness in the right arm. A harsh bruit was present over the left carotid artery in the upper part of the neck with a softer bruit at the base of it and the pulse in the left carotid artery seemed to be diminished as compared with the right. Visual field examination was normal. Ophthalmodynometer measurements revealed a systolic pressure in the right eye of 90 and a diastolic pressure in the right eye of 45. In the left eye the systolic pressure was 60 and the diastolic 25.

Blood count, urinalysis, fasting blood sugar, urea nitrogen and total cholesterol within normal limits. STS non-reactive. Chest and skull films were negative.

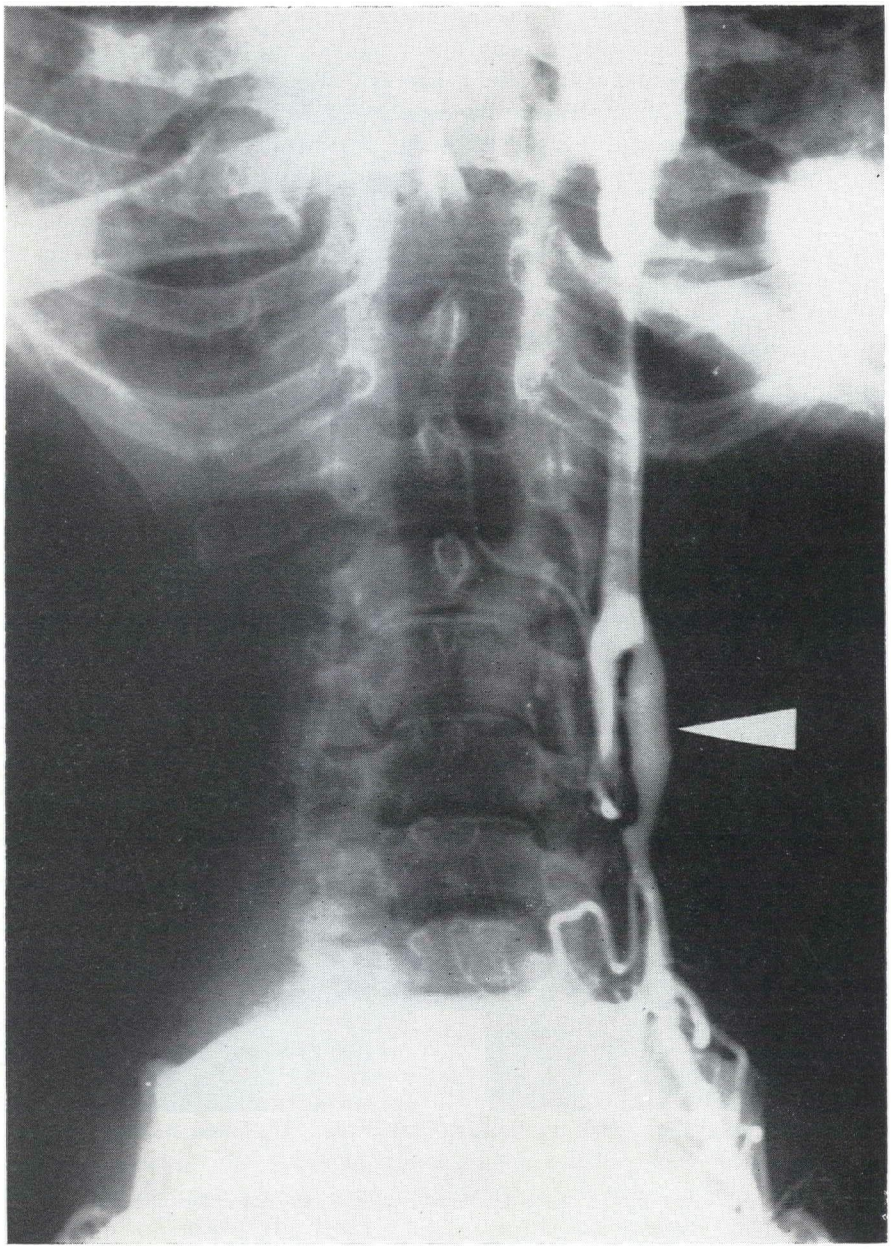


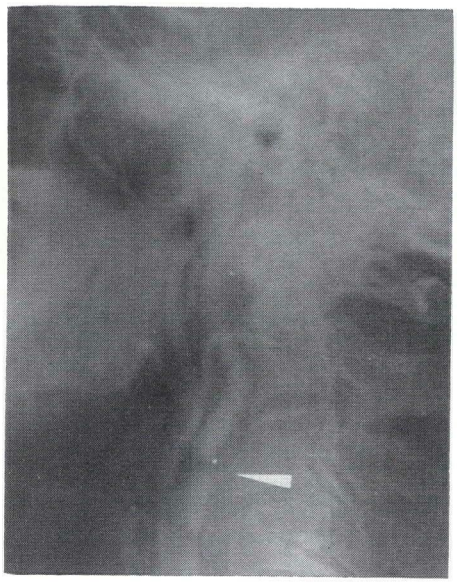
Figure 4

Carotid arteriogram showing patency of by-pass homograft one year postoperative.

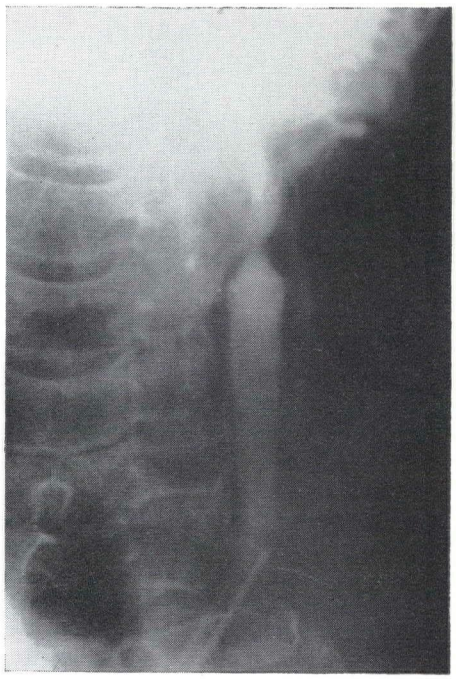
Patient was seen by the Neurosurgical Service on May 12, 1959 and the following day a left carotid arteriogram and right subclavian arteriogram were performed which revealed a segmental partial occlusion of the left internal carotid artery. This lesion was considered for surgical removal and on May 15, 1959, under

local anesthesia, endarterectomy of the left internal carotid artery and carotid bifurcation was performed. Following surgery a left carotid arteriogram showed complete filling of the internal carotid artery. The patient had a satisfactory postoperative course and four days later, on dynamometry, the right eye showed a systolic pressure of 120 and diastolic of 50, and the left eye a systolci pressure of 110 and diastolic of 60. He was discharged on May 20, 1959.

He was readmitted to the hospital on June 26, 1959, at which time he stated that his condition had been satisfactory and he was having no further visual difficulties. On examination, no bruits were audible in the neck and the previously noted right facial weakness and mild paresis of the right upper extremity was still present. On June 27, 1959 a left carotid arteriogram was performed which revealed good filling of the internal carotid artery with no evidence of the previous stenosis, which had been corrected by endarterectomy. (FIGS. 5, 6).



Lateral view



Antero-posterior view

Figure 5

Case II — Marked stenosis of the left internal carotid artery above the bifurcation.

COMMENT

The symptomatology in this patient was due to an obvious segmental occlusion with resulting ischemia. Simple removal of the obstruction relived the symptoms.

CONCLUSION

In order to make a definite diagnosis in patients with symptoms suggestive of cerebrovascular insufficiency, it is necessary to study both carotid and vertebro-basilar circulation by means of angiography. With this information at hand it is possible to

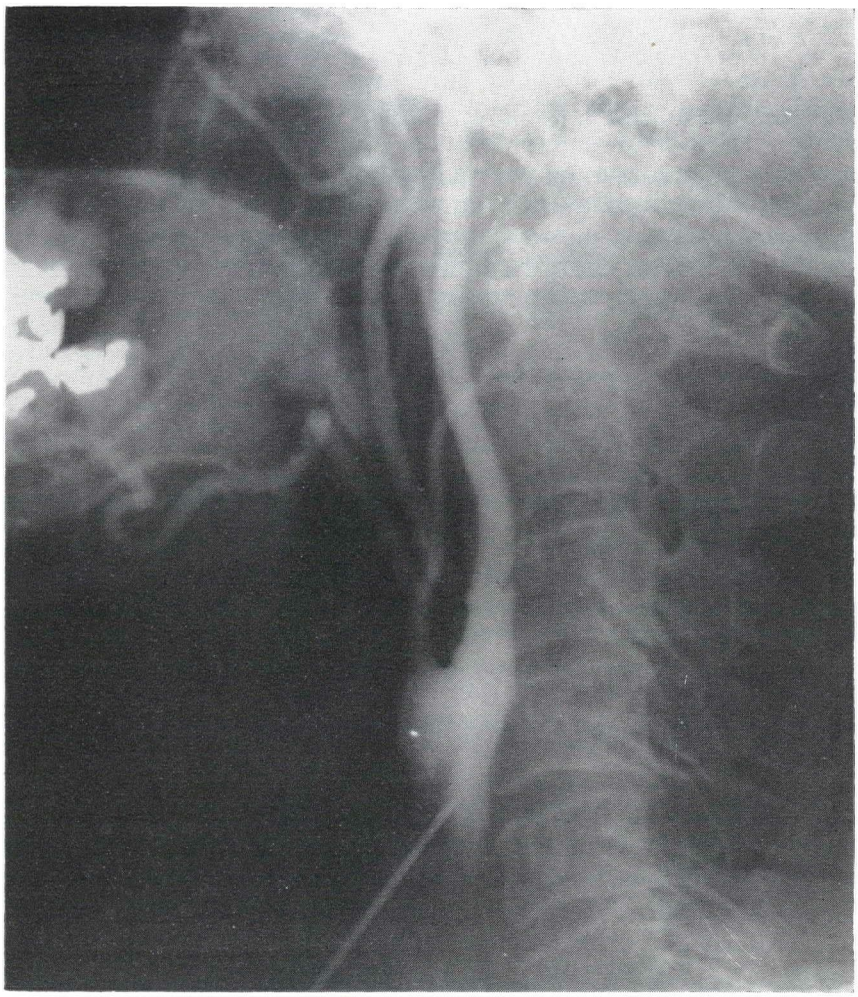


Figure 6

Left carotid arteriogram six week after endarterectomy.

be more objective about the future management of the patient. At the present time our studies indicate that stenosing lesions of the carotid artery may best be treated surgically, while insufficiency syndromes related to vertebro-basilar circulation are less likely to fall within this group.

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