Surgical Treatment of Occlusive Disease of the Carotid Artery *

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IN TREATING 22 patients with rather liberal indications for surgical exploration of the carotid arteries for occlusive disease, our criteria for selection of patients for operation have become more clearly defined. In this report we wish to describe the condition as we have met it clinically, to present a follow up on the first patient treated in 1953, and to discuss our current ideas on the handling of this syndrome.

It is now well recognized that disease of the extracranial carotid arterial system can produce significant cerebral symptoms. Although Chiari in 1905 [2] emphasized the frequency of atherosclerosis in the region of the carotid bifurcation, he thought that cerebral symptoms and lesions were due to emboli. Hunt in 1914 [12] recognized the condition almost as clearly as we do today, pointing out the importance of collateral circulation in prevention or development of lesions and comparing the cerebral dysfunction to intermittent claudication of the legs. The introduction of cerebral angiography by Moniz in 1927 [20] allowed the certain detection of carotid arterial disease during life, and by 1951, 107 angiographically proved cases of carotid arterial obstruction were reported by Johnson and Walker.[14] In 40 per cent of their cases there were transient symptoms that preceded a catastrophic episode of severe neurologic injury, and in 1955 Millikan and Siekert [19] described the characteristics of the partial obstruction and defined a syndrome which they termed "intermittent insufficiency of the carotid arterial system," a term and condition not dissimilar to "cerebral intermittent claudication" described by Hunt 40 years before. In 1954 Fisher [10] reported that severe disease is usually localized to the extracranial carotid artery and that the intracranial vessels are relatively free of disease. Reports of successful surgical therapy,[9, 22, 23] which had antedated Fisher's report, confirmed the localized, accessible, and remediable nature of many of these lesions.

Symptoms in this group of patients have included unilateral impairment of motor or sensory function, sometimes with both modalities involved, syncope, headache, speech disorders and impairment of vision. Unilateral blindness and contralateral hemiplegia, described formerly as a hallmark of the condition, are uncommonly seen. When partial obstruction is present these symptoms may be transient and episodic, as Millikan and Siekert emphasized, and probably are related to the state of the collateral circulation and changes in blood pressure. Meyer [18] and associates demonstrated the effects of a fall of blood pressure, induced by carotid compression or a change of position on a tilt table, in producing symptoms and electroencephalographic

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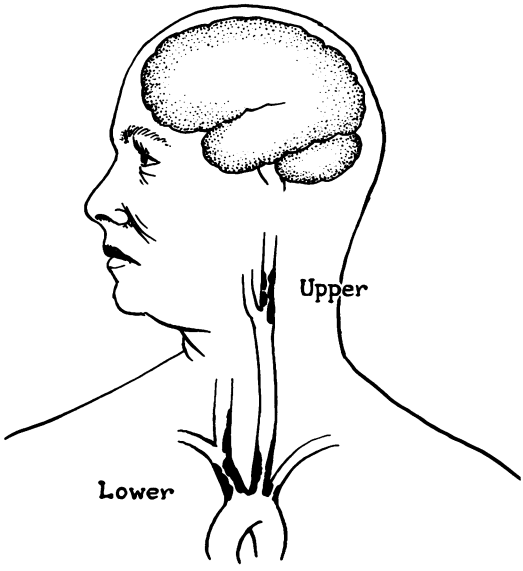

FIGURE 1

alterations in patients with carotid obstruction. Since surgical treatment is more effective before complete occlusion and thrombosis of the internal carotid occur, recognition of the premonitory symptoms and the early stage of the condition is of considerable importance.

Occlusive lesions in the carotid system are usually found in two areas (Fig. 1). The more common site is near the bifurcation of the common carotid artery; this was involved in 20 of our 22 cases and similarly accounted for 90 per cent of the 41 cases reported by Fisher from 432 unselected autopsies. The etiology of obstruction in the upper area is atherosclerosis, the prominence of this disease in areas of bifurcation accounting for the localization in this region. The lesion probably begins as a plaque in the carotid sinus or occasionally in the common carotid artery which later ulcerates, is involved with hemorrhage in the plaque, or enlarges to produce stenosis. Involvement may be bilateral but usually symptoms are predominantly one-sided, probably related to the adequacy of the collateral circulation through the circle of Willis from the other carotid and vertebral arteries. Changes in posture, myocardial infarction, or a sensitive carotid sinus may be specific causes for a drop in blood pressure that precipitates an attack. Symptoms may be slowly progressive from a few to many transient episodes or they may appear catastrophically, as in Case 6 with abrupt occlusion of the artery.

The diagnosis should be considered in the presence of episodic neurological symptoms, especially in association with abnormalities of the carotid pulse or with murmurs or thrills over the carotid bifurcation; it can be confirmed by arteriography. In spite of the known hazards of arteriography [3] we have seen no evidence of significant difficulty with the procedure in this group of patients. Crevasse and associates [5] have attached considerable importance to murmurs and their variation with occlusion of the contralateral carotid artery. They have been of little aid in our experience.

Surgical treatment was first described by Webster, Dolgoff and Gurdjian in 1950 [26] in the form of excision of the internal carotid artery but a direct reparative attack was first advocated by Eastcott, Pickering and Rob,[9] who performed excision of the carotid bifurcation and anastomosis of the common and internal carotid arteries. Successful surgical procedures subsequently used have included excision and replacement with prosthesis or vein graft, bypass, and endarterectomy.[4, 8, 11, 13, 16, 17, 21, 25] The last procedure has been used exclusively in our experience.

A prominent problem in surgical technic concerns minimizing the cerebral ischemia when the carotid is temporarily occluded. We have done this in one of three ways. Hypothermia at 28–30° C. was used in one case. A bypass shunt with a 3/16 inch I. D. cannula connected to a No. 15 needle by plastic tubing was used in two cases, and in the remaining cases the occlusion has

TABLE 1. *Upper Carotid Obstruction*

		Total	Improved	Course Unchanged
Partial Obstruction	Episodic Symptoms	10	8*	2
	Persistent Symptoms	3	0	3
	Total	13	9	4
Complete Obstruction		7	0	7†

* Three died later with contralateral disease.
† Two died later.

simply been kept as short as possible by rapid performance of the endarterectomy. Endarterectomy has usually been completed within ten minutes, often in less than half of this time. In facilitating and expediting this procedure it is important to obtain adequate exposure of the bifurcation of the carotid artery with an incision along the anterior edge of the sternocleidomastoid muscle, careful and complete mobilization of the common carotid artery and its branches, and careful preparation of the plan and means of attack before the vessel is occluded. If the atherosclerosis is extensive a temporary bypass should be used, especially if the neurological state was altered preoperatively by carotid compression; if not, a rapidly executed endarterectomy is well tolerated in these patients.

Our experience is summarized in Tables 1 and 2. Improvement has been most striking in the patients with episodes of transient cerebral dysfunction, in all of whom a partial obstruction was present. Eight of the ten appeared to improve *as a result of operation,* improvement in the other two being consistent with the expected progress of the disease. Three of these patients, after initial improvement for two weeks, and three and four months respectively, subsequently died of contralateral vascular disease. The three patients with partial carotid obstruction and significant neurological deficit before operation but without episodic attacks did not improve remarkably and one later died. Perhaps the endarterectomy has helped prevent extension of preexisting disease in the other two.

In none of the seven patients with complete occlusion has significant improvement occurred. In two instances back-bleeding was obtained, but neither patient obtained a good result. Unfortunately complete obstruction cannot always be determined before operation as the internal carotid may not be visualized by arteriography when there is nearly complete but remediable obstruction without thrombosis. Case 6 is a good example of this; he obtained an excellent result.

The second area of the carotid system which is commonly affected involves the origin of the innominate or left common carotid artery from the aorta and the adjacent portions of the carotid or innominate arteries or the aorta. This accounted for two of our 22 cases, an incidence similar to the four of 41 cases reported by Fisher. Involvement of this portion of the carotid system is part of a syndrome originally described by Broadbent[1] (Fig. 2), often ascribed to Takayasu, imaginatively called "pulseless disease" by Shimizu and Sano[23] and perhaps best termed obliterative disease of the branches of the aortic arch.[15] Etiology of the condition is not yet known

TABLE 2. *Partial Obstruction*

Case	Age Sex	History	Physical Exam.	Arteriogram	Operation	Result
1. B. N.	72 F	Transient lt. weakness for 6 weeks, brief aphasia	Thrill over both carotids. Dysarthria and weakness on carotid compression	None	Plaque removed from rt. carotid bifurcation with temporary bypass. Hemorrhage seen into plaque	Improved
2. J. W.	72 F	1 mo. of confusion, transient aphasia, rt. hemiparesis, syncope. Progressive increase in weakness rt. side	Rt. hemiparesis. Dysarthria	50% stenosis first portion of lt. internal carotid	Endarterectomy of lt. bifurcation and adjacent internal carotid	Improved
3. L. R.	62 F	Dizzy spells, blackout, scintillating scotoma in lt. eye 3–4 times a day. Transient lt. hemiparesis 18 mo. ago	Normal	Rt. normal. Plaque at origin of lt. int. carotid	Lt. carotid endarterectomy	Complete relief. Fully patent carotid arteriogram
4. W. R.	48 M	Rt. hemiplegia and slurred speech 2 mo. before	Partial rt. hemiplegia	Stenosis of lt. internal carotid	Endarterectomy with temporary shunt	Improved
5. W. S.	63 M	3 mo. lt. hemianopsia	Lt. homonymous hemianopsia	50% stenosis of rt. int. carotid	Rt. endarterectomy	Improved
6. J. B.	49 M	Occasional tingling rt. hand 2 mo. "Numb" rt. hand and aphasia 2 hours	Slight motor aphasia improving	No filling of lt. int. carotid	Lt. endarterectomy. No shunt. Hemorrhage into plaque at origin of internal carotid	Improved. No residuum
7. F. S.	66 M	3 episodes of headache, dysarthria, rt. hemiparesis during last 10 days	Hemiparesis and dysarthria	None	Endarterectomy of lt. carotid bifurcation with temporary bypass. Plaque about 50% occlusive	Improved and well 3 mo. Returned and died with basilar artery thrombosis
8. G. D.	54 M	Numerous small strokes on rt. side, one on lt. Rt. hemiparesis and aphasia 2 weeks before		Severe stenosis lt. internal carotid at its origin and suggestive narrowing just proximal to circle of Willis	Endarterectomy of plaque in lt. common int. and ext. carotid	2 wks. improved. Arteriogram showed wide cervical carotid with narrowing of siphon
		Massive lt. hemiparesis and coma 2 wks. after operation on lt. carotid	Unresponsive	Plaque at rt. carotid bifurcation and int. carotid	Endarterectomy origin of rt. int. carotid	Died next day. Extensive sclerosis interacerebral vessels occl. rt. vertebral art. Carotids patent in neck
9. F. H.	59 F	Partial aphasia and visual difficulty for 6 mo. Red halo in lt. eye during early morning hours	Pallor of lt. retina	Poor	Large plaque filling lt. carotid bifurcation and into internal carotid. Endarterectomy	Relief of all symptoms for 4 mo. Returned with lt. hemiplegia, bloody spinal fluid. Died. No autopsy
10. A. B.	68 M	Transient rt. stroke, several years aphasia, visual difficulty often since		Rt. unsuccessful. Lt. stenosis int. carotid at origin	Plaque in common and int. carotid on rt. Endarterectomy. On lt. large plaque almost occl. int. carotid	Postoperatively carotid arteriogram widely patent. No improvement. Vision and speaking poor
11. N. L.	68 M	Rt. hemiparesis 4 mo. ago, recurrent dizziness and gradual cerebral degeneration	Residual rt. hemiparesis	Stenosis of origin of lt. int. carotid	Lt. carotid endarterectomy	Carotid widely patent. No significant improvement

TABLE 2.—*Continued*

Case	Age Sex	History	Physical Exam.	Argeriogram	Operation	Result
12. J. W.	78 F	4 days numbness of rt. arm, 3 days weakness of rt. arm. Progressed to rt. paralysis and slurred speech	Barely conscious	Marked narrowing lt. int. carotid, some narrowing on rt.	Endarterectomy of int. and ext. carotids. Good pulse restored under hypothermia	No improvement. Died week later
13. E. J. C.	54 F	Lt. hemiparesis 4 mo. before, considerable clearing	BP 210/120. Lt. hemiparesis and spasticity	Unsatisfactory	Endarterectomy of rt. bifurcation. Little loss of pulse before, or gain	Progressive clearing, not strikingly influenced by operation
			Complete Obstruction			
14. E. McE.	68 F	Rt. hemiparesis 2 years ago. Dizzy spells every 2 wks. or so since. 24 hrs. severe rt. hemiparesis and aphasia	Able to squeeze hand. Progressive worsening	Complete obst. lt. int. carotid	Plaque at lt. bifurcation; organized thrombus in int. carotid. Plaque in rt. int. carotid removed	Died in one week. Unchanged
15. F. M.	62 F	4 hrs. rt. hemiparesis and aphasia	Able to squeeze hand	Rt. side normal. Lt. int. carotid totally occl.	Organized thrombus in lt. int. carotid could not be removed	Unchanged
16. F. G.	64 M	5 days lt. hemiparesis and aphasia	Aphasia. Lt. hemiparesis	Rt. side normal. Occlusion origin lt. int. carotid	Plaque in common and int. carotid removed and thrombus sucked out of int. carotid, good back bleeding	Responsiveness improved slowly. Hemiparesis improved. Died 6 wks. later of aspiration pneumonia
17. A. S.	58 M	Rt. hemiparesis, dysphagia and dysarthria 9 mo. ago. Cleared. Reappearance 24 hours ago with syncope	Minimal hemiparesis. Syncope on carotid compression	No filling of lt. int. carotid	Lt. int. carotid firmly thrombosed; no back bleeding obtained. Endarterectomy of bifurcation. Temporary bypass to ext. carotid	Unchanged
18. C. H.*	50 M	Hemiplegia 3–4 weeks	Lt. hemiplegia	Complete occl. int. carotid	No intimal disease. Thrombectomy but no back bleeding	Unchanged
19. J. L.*	38 M	Lt. hemiplegia 3–4 weeks	Lt. hemiplegia	Complete occl. int. carotid	No intimal disease. Thrombectomy. No back bleeding	Unchanged
20. S. M.*	41 M	Aphasia, rt. hemiplegia 3–4 weeks	Rt. hemiplegia	Lt. common carotid occl.	No intimal disease. Thrombectomy. Good back bleeding	Unchanged
			Lower Carotic Obstruction			
21. A. L.	58 M	Almost monthly syncopal attacks, tinnitus, blurred vision, convulsive movements. Temporary relief with endarterectomy by G. Murray, 1951. Recurrence of symptoms and lt. hemiplegia 1952. Treated syphilis	No pulse in upper body BP in legs 170/60	Obstruction of branches of entire aortic arch	Aug. 1953. Bypass graft around occlusion of first portion of innominate artery. Lt. carotid a fibrous cord	Return of BP to normal in rt. arm and legs, carotid pulse on rt. Asymptomatic and well Nov. 1958
22. B. A.	22 F	5 yrs. ago no pulse at rt. wrist. 6 mo. ago "black curtain over eyes," rt. frontal headache, numb and weak on lt. Gradual improvement since	Weak pulses in rt. arm and carotid. Systolic murmur over upper rt. chest	None	Chronic arteritis from innominate to common carotid. Resected and replaced by Nylon from base of innominate to common carotid	Improved. Well 1 yr. later

* Treated at Walter Reed Army Hospital with Col. Arthur Cohen, MC.

XLII.—*Absence of Pulsation in both Radial Arteries, the Vessels being full of Blood.* By W. H. BROADBENT, M.D. *Read May* 14, 1875.

that the mouth of the former was exceedingly small. On further examination the innominate was found not only to be narrowed at its origin, but rigid. An atheromatous patch surrounded the mouth of the vessel involving the structures both of the aorta and the innominate; it was hard and brittle without being distinctly calcareous, and separated readily from the outer tunic. Immediately above its origin the innominate enlarged to its usual size, and possessed its normal elasticity. Here, then, was the explanation of the absence of pulse with a full vessel at the right wrist.

Tr. Clinical Society, London

FIGURE 2

and probably is not unique. The majority of pathologic descriptions have indicated a nonspecific arteritis. Syphilis, tuberculosis, collagen vascular disease, periarteritis nodosum, thromboangiitis obliterans, rheumatic fever, and trauma have all been implicated. The predilection for young women has suggested an endocrine connection. Our first patient, previously reported,[22] is representative of the syndrome except for his sex. He was 58 years old when treated and had noted the onset eight years previously of episodic attacks of syncope, occasionally with convulsions, tinnitus, and blurred vision. Two years before admission Dr. Gordon Murray had performed a bilateral cervical sympathectomy and thrombectomy of both common carotid arteries. He was improved for four and one-half months, but the attacks reappeared and after one of them a left hemiplegia and a speech defect persisted. On examination no pulse could be felt in either carotid, brachial, or radial artery. Angiocardiograms showed narrowing of the origin of the innominate and left carotid arteries. The serological test for syphilis was positive.

On August 10, 1953 surgical exploration * revealed the left common carotid artery to be fibrosed, narrowed, and completely obliterated throughout its intrathoracic and lower cervical course (Fig. 3). The base of the innominate artery at its origin from the aorta was narrowed and a thrill was palpable in this region. The left subclavian artery was not visualized; it was thought to be occluded as no pulse was palpable in the distribution of this artery. A frozen dried homograft, the aorta of a child, was sutured to the side of the ascending aorta and then to the end of the divided innominate artery, the latter vessel being interrupted for a total of 8 minutes. His convalescence was uneventful and he has had no further syncopal or other attacks. The buzzing in his ears was initially more prominent, probably related to the systolic bruit heard over the vigorously pulsating right carotid artery. In November 1958 he was well, retired to Florida, but working daily in his yard.

* Performed with the surgical resident, Dr. Dwight McGoon.

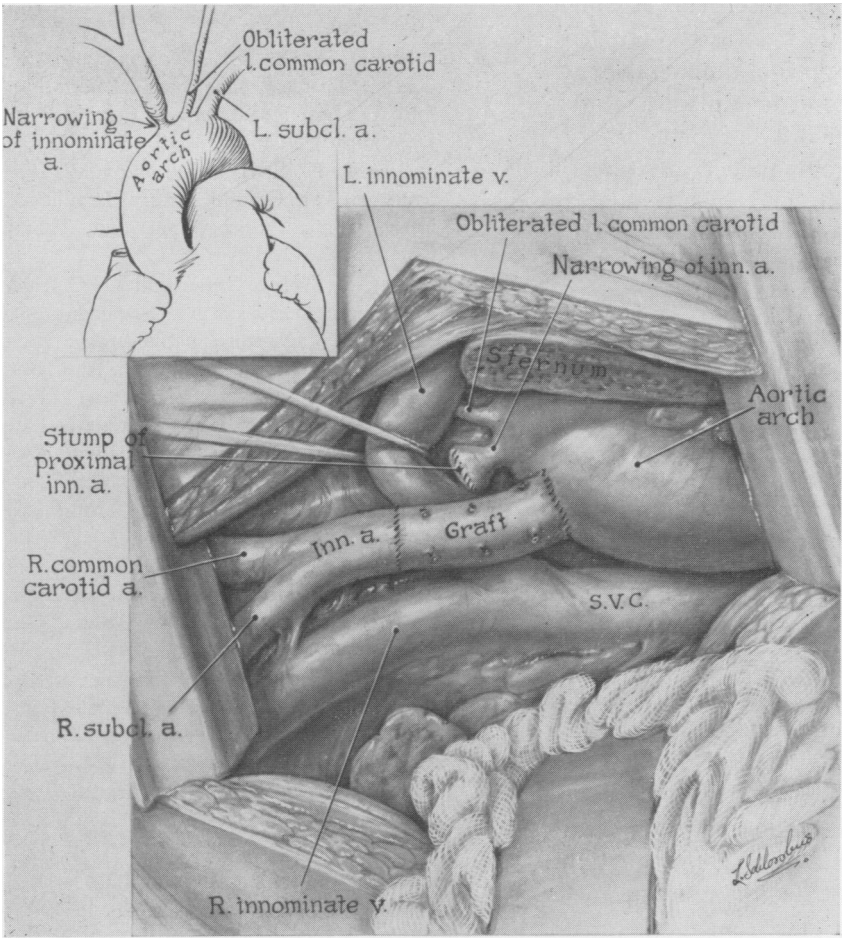

FIG. 3. Occlusive disease of the extracranial carotid system characteristically occurs either near the aortic arch or at the bifurcation of the common carotid artery, the other areas being relatively free of disease.

The second patient, a 22-year-old female, with a similar condition has obtained complete relief of symptoms following resection of the innominate artery and replacement with a nylon prosthesis between the innominate and the right common carotid artery.

Excision with bypass or direct replacement, also used by others,[6, 7] appears at the present time to be the surgically preferred treatment although endarterectomy should give a good result for a short obstruction due to arteriosclerosis. Spittel and Siekert,[24] however, have reported dramatic relief from symptoms in a somewhat similar case with long term anticoagulant therapy.

In a disease with a variable natural course as this evaluation of results is difficult. A good result has been described in a patient in whom subsequent arteriogram showed an occluded vessel.[13] Realizing this we have classed our patients as improved only when the course of the disease seemed significantly altered by the operative procedure, but we are cognizant that further evaluation of this procedure is necessary.

Conclusions

Based upon these experiences the following conclusions have been drawn. A remediable narrowing of the carotid arterial system may be present in patients who have sudden or episodic unilateral motor or sensory disturbances, syncope, ocular

symptoms, or speech disorders. A single symptom complex does not exist. The hallmark of carotid occlusion, unilateral blindness with contralateral hemiplegia, is present only in the minority of patients. The variations in symptoms reflect variations in collateral circulation and involvement of other vessels and emphasizes the need for carotid arteriography in evaluation of these patients. Such an obstruction must be sought at both the origin of the vessels from the aorta and at the bifurcation of the common carotid artery in the neck.

The most favorable cases are those in which there is a history of transient episodes of neurologic symptoms without significant continuing neurologic deficit and in which a point of localized obstruction can be demonstrated. When symptoms are continuous and neurologic deficit is marked, benefit from surgical treatment is less likely. Complete occlusion of the internal carotid will rarely be treated successfully unless the occlusion is of very brief duration.

Endarterectomy has been our procedure of choice in treating upper carotid obstruction and bypass or direct replacement the preferred treatment for obstruction near the origin of the vessels from the aorta. In the usual operation performed expeditiously no measure to support the distal circulation is necessary during the required temporary carotid occlusion. When the obstruction is unusual or extensive, a temporary bypass may be used to allow a longer period of safe operative occlusion.

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Discussion

Dr. J. K. Quattlebaum, Jr.: Dr. Lilly, members and guests, again let me thank you for the privilege of the floor.

I would like to take this opportunity to show a few slides which bring up a different cause for the stroke syndrome due to carotid occlusion and one which, I think, has not heretofore been recognized. That is to say, obstruction of the carotid arteries secondary to degenerative elongation of the vessel to the point of kinking.

We have treated surgically three patients who presented with transient hemiparesis of the opposite side which had cleared to a greater or lesser degree. I'll show you some of the material on these patients and then some other patients.

(Slide) Here is an arteriogram and I'll have to apologize for the fact that with the initial injection, I missed the carotid artery and extravasated dye in the soft tissues surrounding it. Percutaneous arteriography demonstrates the needle in the common carotid down here, and here is the superior thyroid and other branches of the external. The internal carotid artery ascends and then angulates sharply on itself before coming forward and then ascending again into the skull.

This patient had had a transient hemiparesis on the opposite side.

(Slide) This—if you will bear with me—is a picture taken at surgery. The common carotid and bifurcation are not shown, but here is the internal carotid artery coming up and kinking sharply on itself again before ascending out to the skull which is in this direction.

(Slide) After the resection of a two centimeter segment of the common carotid artery, the anastomosis being here (indicating), one can now see the bifurcation of the external carotid here and the internal carotid gradually ascending without kink to the skull. It had previously as you've seen, had kink in this region.

(Slide) Here's a postoperative arteriogram showing obliteration of the kinking.

(Slide) Here's another patient who presented with a transient hemiparesis and whether you can see it or not, I don't know. On his arteriogram, we saw an elongation of the vessel and at this point, there is a band-like affair across the vessel, a radiolucency.

Now, we didn't know the significance of this and suggested exploration of the vessel under local anesthesia. This was done.

(Slide) Here, I think, one can see the common carotid artery, the external carotid disappearing under the retractor and the kinked internal carotid artery.

(Slide) The carotid bifurcation was resected and end-to-end anastomosis of the common to the internal carotid artery.

(Slide) Here is a picture of the third case, the arteriograms of which I don't have on slides. The common carotid artery is here, the external goes up this way. Here is the internal carotid artery going up, back down, kinking on itself and then ascending to the skull which is in this direction.

This is the way we found it in the carotid sheath minimally dissected here.

(Slide) Our next slide shows, after dissection of the carotid, sheath the degree of tortuosity. The remarkable thing about all of these is the pliability of the vessels, the absolute absence of arteriosclerosis except for the last two cases which had small nonobstructing plaques in the carotid bifur-