Surgical Treatment of Atherosclerotic Occlusion of the Internal Carotid Artery *

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ALVAREZ [1] has recently defined a syndrome of "little strokes" wherein transient episodes of cerebral ischemia are a prelude to encephalomalacia or thrombotic infarction of the brain. This concept assumes that atherosclerosis of the intracranial vessels is the important underlying factor. Although it can be easily demonstrated that atheromatous plaques and anomalies of the circle of Willis occur intracranially,[3] recent studies [3, 19, 24, 26, 27, 38] identify the greater importance of segmental plaques in the innominate, common carotid, internal carotid, or vertebral arteries in an *extracranial* segment. As early as 1914, Hunt [25] recognized that atheromatous obstruction of the internal carotid *in the neck* might be complicated by a collateral flow from the circle of Willis so reduced as to be a menace to the affected hemisphere and to produce "intermittent cerebral claudication" during periods of transient hypotension.

Thrombosis of the internal carotid artery *in the neck* has emerged as a clinical entity within the past five years.[2, 4, 5, 7, 8, 9, 10, 11, 18, 20, 21, 22, 23, 27, 28, 33, 34, 35, 39, 40, 41, 42, 44, 45, 46, 47, 48] The neurologic manifestations are those of a frontal lobe syndrome. Initially, there is no cerebral thrombosis or infarction, but symptoms result from the ischemia due to inadequacy of collateral blood flow into the middle cerebral artery. Men are affected four times as frequently as women, and the age group between 30 and 60 years is especially vulnerable. About one-fourth of the patients show significant spontaneous recovery.[23, 35, 41] The site of predilection for the thrombogenic or obstructing plaque is in the region of the carotid bifurcation at or above the carotid sinus, but one-seventh of the occluded internal carotids show a primary plaque in that extra-dural, intracranial segment known as the siphon with retrograde thrombosis.[19, 24, 46] The increasing frequency of diagnosis stems from more routine study by carotid arteriography as introduced by Moniz.[32]

Significant cerebral infarction may complicate internal carotid occlusion if there is prolonged hypotension or faulty collateral. The most frequent cause of prolonged hypotension is a surgical operation or complicating myocardial infarction.[19, 25, 38] Although plaques or anomalies in the circle of Willis have obvious importance, the collateral more frequently fails because of complete or incomplete obstruction in the contralateral internal carotid [10, 18, 19, 20, 23, 38] or in the vertebral arteries.[26, 27] The great importance of the vertebrals in the collateral system is emphasized by the high incidence of infarction when plaques are present in both the vertebrals and the carotids in the so-called "carotico-vertebral syndrome." [26, 27]

Earlier attempts in the treatment of internal carotid occlusion were not clearly successful.[18, 22, 23, 34, 41, 42, 44, 47, 48] These methods included novocain block of the cervical sympathetic ganglions,[34, 35, 47] cerv-

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ical sympathectomy,[5, 7, 33, 34, 35, 42, 45, 47] denervation of the contralateral carotid sinus,[7, 8] thrombo-endarterectomy,[42, 43, 47] carotid-jugular fistula,[34] and anti-coagulants.[5, 7, 33, 34, 42, 45] As a result of this experience, renewed effort was urged for direct attack upon the obstruction.[18, 42, 48]

The first successful operation was reported by Eastcott, Pickering and Rob.[15] Utilizing hypothermia 28° C., the common carotid was occluded for 28 minutes while the bifurcation and its occlusive plaque were resected and continuity was restored by suture of the patent distal end of the internal carotid to the common carotid. The preoperatively noted daily attacks of transient blindness, aphasia and right hemiparesis were relieved throughout the 4½ month period of postoperative observation. Carrea, Molins and Murphy [6] encountered blindness, aphasia and hemiparesis after massive gastro-intestinal hemorrhage and anastomosed the external carotid end-to-end to the distally patent internal carotid and then resected the plaque. This patient recovered from his aphasia and paralysis, but remained blind. Hypothermia was not used. Denman, Ehni and Duty [13] have reported staged bilateral carotid resection with replacement by arterial homograft. No hypothermia was used and, although the patient survived, he was able to speak only single words four months after operation. Cooley [12] undertook thrombo-endarterectomy for an incomplete carotid obstruction producing head noises and dizziness on standing. The head was immersed in crushed ice and a polyvinyl shunt was needled into the common and internal carotids as a temporary shunt to permit arteriotomy in the region of the plaque. The patient was paralyzed postoperatively and Cooley concluded that generalized hypothermia and a larger temporary external shunt would have been useful. Lin, Jarvid and Doyle [30] used a saphenous autograft to restore continuity after resection of a segment of internal carotid with an occlusion time of 22 minutes. No hypothermia was used and neurologic deficits persisted after operation. Edwards and Rob [16] have now reported the second case of hypothermia and repair by excision and direct suture of the internal and common carotids with recovery from neurologic deficits.

All of the above experiences identify the importance of the ipsilateral external carotid as a source of collateral blood flow when the internal carotid is obstructed. This has been confirmed on arteriography by retrograde filling of the anterior branch of the middle cerebral artery by way of the ophthalmic artery from the facial and angular branches of the internal maxillary trunk of the external carotid.[9, 29, 31] The transient unilateral blindness occasionally reported as a symptom of internal carotid occlusion has been attributed to reversal of flow in the ophthalmic artery [9] and the monocular optic atrophy noted in patients with common carotid occlusion [21] further emphasizes the importance of this collateral pathway. The occipital branch of the external carotid may also augment the vertebral flow.[36]

Our initial interest in the problem of internal carotid occlusion was the challenge presented by a segmental obstruction in the arterial system. Although it was apparent that hypothermia would permit a direct attack upon this lesion, a way was sought to retain the external carotid flow and avoid the risk and necessity of hypothermia. A shunt from the subclavian to the distally patent internal carotid appeared as a desirable bypass and our experiences with this procedure are recorded here.

1. *The selection of patients:* The primary criteria for surgical consideration of these patients are the presence of symptoms without evidence of major infarction, absence of major electroencephalographic abnormalities, and a demonstrated occlusion of the internal carotid artery by carotid arteriogram. The usual radiologic picture is that of the obstruction of the internal carotid at, or within 2 cm., of its origin from the

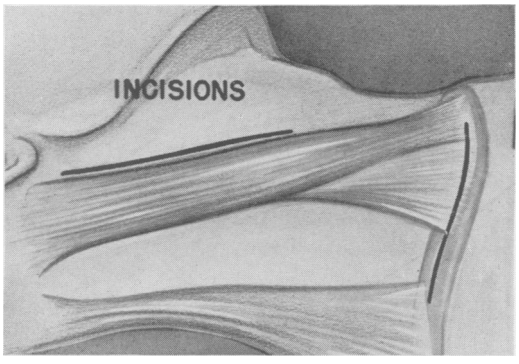

FIG. 1.

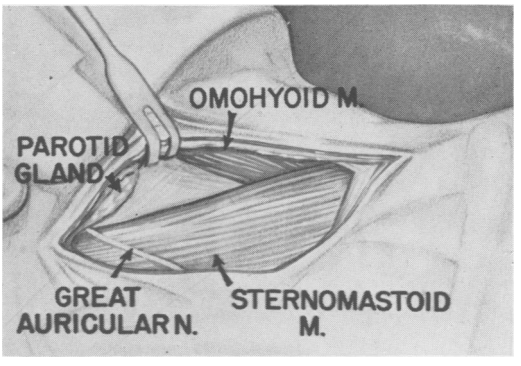

FIG. 2.

bifurcation.[23, 40] Retrograde thrombosis from a plaque in the siphon may progress to obstruct the carotid in the neck and give a picture identical to that of a plaque at the sinus.[22, 23] This could be clarified by contralateral carotid arteriography were it not for the risk of serious cerebral ischemia inherent in this manuever.[32] At the present time, it seems safer to expose the bifurcation in the neck to identify the presence or absence of a plaque in the region of the sinus.

2. *Technic of operation:* The technical details of the surgical approach are presented in Figures 1 to 6. The first incision is placed over the sternomastoid starting just below the mastoid process. The great auricular nerve is identified and preserved. Division of the common facial vein permits lateral retraction of the jugular vein and exposure of the carotid bifurcation. The area of the sinus is palpated and the presence of a plaque is regarded as evidence of local origin of the occlusive process. The dissection then follows superiorly to mobilize the internal carotid artery and to identify the hypoglossal nerve as it crosses superficially to the artery. No difficulties have resulted from rather forceful retraction of the nerve. The superior laryngeal nerve regularly courses behind the internal carotid and has not presented a problem. Occasionally, a collaterally enlarged occipital branch of the external artery crosses in front of the internal carotid and should be preserved. The

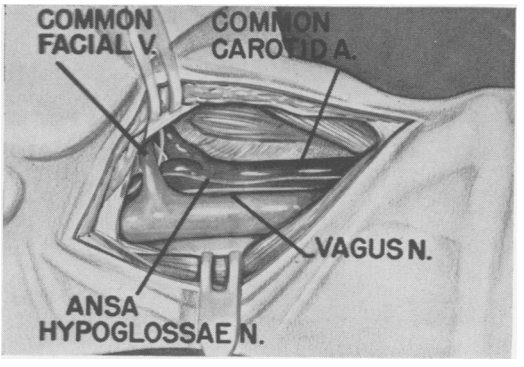

FIG. 3.

internal carotid may show a serpentine tortuosity, and considerable length, even in the non-tortuous vessel, may be obtained by traction with a tape through the carotid bifurcation. Subsequent handling of the internal carotid is facilitated by stripping of the adventitia with nerve hooks and fine scissors.

The next step in the operation is dictated by the nature of the obstructive process as shown by the preoperative carotid arteriogram. If the obstruction is complete, an arteriotomy is performed in the long axis of the vessel. Patency of the distal segment is evaluated in terms of back-bleeding, probing, saline-heparin infusion, or operative arteriogram. If distal obstruction is demonstrated, the artery is closed or ligated and

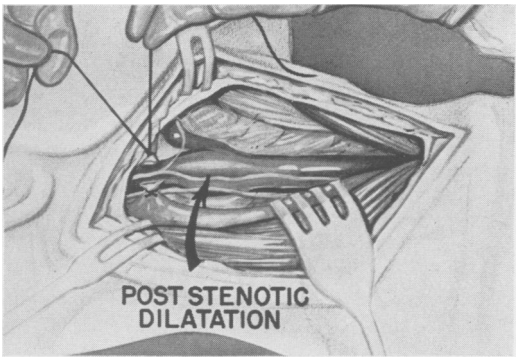

FIG. 4.

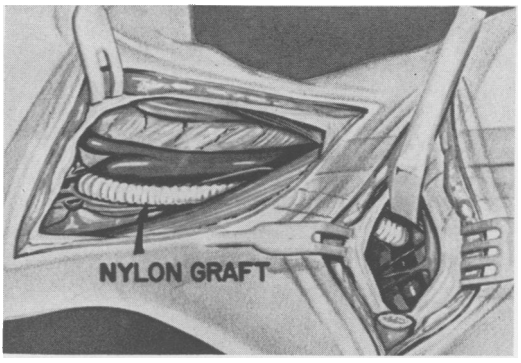

FIG. 6.

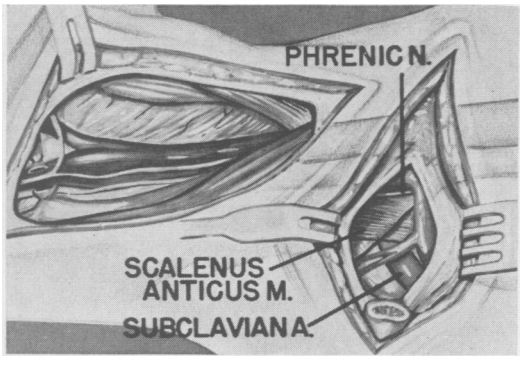

FIG. 5.

the operation is concluded. When the obstruction is incomplete in the region of the sinus, the arteriotomy has been delayed until the shunt from the subclavian is ready for insertion into the internal carotid.

The approach to the subclavian artery has been through a separate incision with subperiosteal resection of the proximal two-thirds of the clavicle and transverse section of the scalenus anticus with gentle medial retraction of the phrenic nerve and jugular vein. At this time, the cephalic stump of the scalenus anticus may be retracted laterally to identify the vertebral artery lying medially and posteriorly. It is important to palpate this vessel and identify any atherosclerotic degeneration. Further exposure of the subclavian artery permits its delivery into the wound by traction with umbilical tapes. When these are occluded, a bloodless field permits arteriotomy and end-to-side suture of the shunt. We have preferred the five-sixteenths inch crimped nylon prosthesis of local design.[17] The prosthesis is delivered through a tunnel under the sternomastoid and sutured end-to-side into the internal carotid. An operative arteriogram may be done through the nylon tube if desired. The wounds have been drained for 24–48 hours.

3. *Results:* This report is based upon six cases with five successful shunts and four patients surviving without significant neurologic signs or symptoms.

CLINICAL ABSTRACTS

1. Mr. J. T. S., Hosp. No. B-78575, 67-years-old, retired tool-maker, was admitted August 1, 1956. For 7–8 months, he had noted a "swishing sound" in the right ear, an inability to sleep on his right side, and right-sided headaches. An angiogram demonstrated constriction and narrowing of the right internal caortid artery with filling of the intracranial portion. During the next week, the intensity of the bruit was noted to diminish by the patient and his examiners. At the same time he noted transient attacks of weakness of the left arm and leg on arising in the morning. The headache was more severe. These progressive signs of right internal carotid insufficiency were not associated with significant electro-encephalographic abnormality.

On August 9, 1956, the body temperature was reduced to 30° C. by external cooling. The bifurcation of the right carotid artery was exposed and a sclerotic plaque identified. The subclavian was exposed and a nylon prosthesis sutured end-to-side in

this vessel. The internal carotid was occluded for 14 minutes by a clamp at the level of the sinus. There was moderate bleeding when the proximal clamp on the carotid was released, but there was no back-bleeding from the intracranial portion of the internal carotid when the distal clamp was released. Opening of the shunt effected a vigorous pulsation in the graft and the internal carotid. The patient was re-warmed in the usual fashion and recovered from his operation without neurologic deficit. He sat up without symptoms on the second postoperative day.

One month after operation, the patient reported that his headache and the bruit were completely gone. There had been no further episodes of left-sided weakness and the patient and his wife felt that he showed considerable improvement in his ability to express his thoughts. The graft was pulsatile and there was a faint, systolic bruit audible over the graft. Contralateral carotid occlusion by Matas test caused only a slowing of the pulse. Seven months after operation he continued well.

Summary: A 67-year-old man developed progressive stenosis of the right internal carotid artery over a period of 8 months. Signs of arterial insufficiency developed as the intensity of the bruit diminished. Arteriogram demonstrated incomplete obstruction with complete filling of the intracranial portion of the carotid. At operation, no back-bleeding was demonstrated. A prosthetic nylon shunt between the subclavian and internal carotid artery restored arterial circulation with clinical improvement.

2. Mr. T. C. T., Hosp. No. B-79227, 66-year-old farmer, was admitted August 25, 1956. Five years previously, he first developed transient dizzy spells with episodic right hemiplegia and slurring of speech. During the year prior to entry, these intermittent seizures recurred with increasing frequency until they became of daily occurrence. At this time there was continuous left frontal headache and the speech was constantly slow and unclear. Within the past year, a staggering gait had developed in the pattern of a basilar insufficiency in addition to the frontal lobe symptoms. The electroencephalographic record was normal with low voltage. A left carotid arteriogram visualized only the left external carotid and its branches.

On August 27, 1956, under general anesthesia, the carotid bifurcation was exposed and an atherosclerotic plaque identified. Four cm. above the bifurcation, the internal carotid was soft and contracted. It was possible to inject 2 ml. of saline containing 10 mg. of heparin into the vessel at this point. The internal carotid was then opened, demonstrating a patent vessel without back-bleeding. A dilated common carotid was partially obstructed with a C-clamp for attachment of the prosthesis. The vessel was friable and suture was complicated by intimal tearing. The nylon prosthesis was then sutured into the internal carotid at the site of arteriotomy with a second end-to-side anastomosis and prompt restoration of arterial flow.

The next morning the patient got out of bed without assistance to go to the bathroom and then ordered his breakfast without speech difficulty. The gait was improved but not completely normal. There were no recurrent seizures, the speech was clear, and thinking was effectively integrated. On his tenth postoperative day, he rode home by auto for 200 miles. That evening he had a headache and momentary dizziness without hemiplegia or aphasia. Two months after operation, there was a gastrointestinal upset with recurrence of severe headache for two days.

Four months after operation, the patient returned with excellent pulsation in the prosthesis, but with the clinical picture of basilar insufficiency. The course was rapidly down-hill and autopsy was not permitted.

Summary: A 66-year-old white male developed, over the course of five years, daily episodes of frontal lobe ischemia with persistent headache and partial aphasia. For one year, there had been a progressive ataxia suggesting basilar ischemia as well. Revascularization of the left internal carotid relieved the frontal lobe syndrome for four months. At that time there was apparently a fatal cerebral infarction incident to basilar insufficiency.

3. Mr. D. McC., Hosp. No. B-79580, 64-years-old, retired business man, was admitted August 29, 1956. Early in May, 1956, he noted, over a three day period, the progressive development of left hemiparesis. There was slow improvement with residual weakness of the left side, dizziness, fatigue, bifrontal headache and slowing of his mental processes. The electroencephalogram was borderline with slight changes in both temporal regions. Angina pectoris was present. Right carotid compression produced no symptoms, but left carotid compression induced syncope and muscular twitchings of the face in five seconds. The right carotid arteriogram showed occlusion of the internal carotid at the bifurcation.

On September 5, 1956, under general anaesthesia, the right carotid bifurcation was exposed. There was no evidence of atherosclerosis, but the internal carotid was contracted and there was the bluish discoloration of an intra-luminal thrombus. The distal segment of the internal carotid was opened, but the organized and adherent thrombus could not

be extracted. It was concluded that the lesion was one of retrograde thrombosis associated with an obstructive plaque in the region of the siphon and the wounds were closed.

Six months postoperatively, the patient's family reports continuing deterioration.

Summary: A 64-year-old man developed partial left hemiplegia four months prior to entry. He was found to have occlusion of the internal carotid on the right without evidence of plaque formation in the bifurcation. No treatment was attempted and it was concluded that the thrombosis had occurred in retrograde fashion from an intracranial plaque, presumably in the region of the carotid siphon. There has been progressive deterioration of the patient.

4. Mrs. L. L. S., Hospital No. B-80219, 67-year-old housewife, was admitted September 19, 1956. One month previously she had noted headache and "heaviness" of the right arm. The day before admission she had a sudden onset of dizziness, aphasia and right facial paralysis. The Babinski was positive on the right. The electroencephalogram was borderline with slowing in the transtemporal leads. The important neurologic residuals of this episode were moderate aphasia, right facial weakness, and weakness of the right arm. A left carotid arteriogram showed obstruction of the internal carotid at the bifurcation with intracranial collateral from the external carotid via the ophthalmic artery.

On September 28, 1956, under general anesthesia, the carotid bifurcation was exposed and found to be sclerotic. A purple thrombus was externally recognized in the proximal 2 cm. of the internal carotid. Above this, the internal carotid was contracted but a feeble pulse was identified. A nylon prosthesis was attached to the subclavian and ultimately sewn into the internal carotid. Opening of the internal carotid permitted thrombectomy but no flow from the common carotid occurred. Back-bleeding from the distal end was present but small in amount. With opening of the shunt, a strong pulse was restored in the distal segment.

There was marked improvement neurologically in the immediate postoperative period. The aphasia cleared completely and there was only weakness of the right face without asymmetry.

Three months later there was no residual neurological deficit and the patient continues well 7 months after operation.

Summary: A 67-year-old housewife had prodromal headache and "heaviness" of the right arm for one month prior to the sudden onset of asphasia, right facial paralysis and weakness of the right arm. There was limited immediate recovery from this episode and arteriogram identified left carotid occlusion. At operation, a thrombus was removed from the carotid and a shunt was established. Postoperatively, the aphasia cleared immediately and at three months there were no neurologic deficits. She continues well.

5. Dr. R. B., Hosp. No. B-81926, 59-year-old surgeon, was admitted December 8, 1956, with a two year history of intermittent dizziness and syncope, occasionally associated with transient, monocular blindness. His surgical activities had accordingly been modified. On November 21, 1956, there was an episode of left facial paralysis, numbness of the left arm, and partial aphasia. This cleared with residuals of left facial weakness, thick speech, and scotomata. Electroencephalographic studies demonstrated initially a suppression of activity on the right side. This improved preoperatively and was essentially normal postoperatively.

On December 10, 1956, the arteriographic demonstration of carotid obstruction was confirmed with the finding of a plaque in the region of the sinus. Arteriotomy above the point of occlusion demonstrated a patent vessel and back-bleeding. A nylon prosthesis between the subclavian and the carotid restored pulsations. The speech was clear and distinct upon recovery from anesthesia. The facial expression was assuredly normal on the day following operation and there were no scotomata.

Three months after operation the patient continued well and resumed active surgical practice.

Summary: A 59-year-old surgeon, well known to us, developed episodic seizures of frontal lobe ischemia with resultant limitation of clinical activities. A "little stroke" was associated with residual deficits of facial weakness and partial aphasia. Restoration of carotid blood flow reversed these deficits completely and permitted return to active practice three months later.

6. Mr. D. L. H., V.A. Reg. 22033, 48-years-old, was admitted November 15, 1956, because of lower leg claudication of two years' duration. No pulses were palpable in the legs below the femorals. Bilateral femoral arteriograms demonstrated a complete block of the left superficial femoral and a segmental block of the right superficial femoral. A left lumbar sympathectomy and right superficial femoral bypass operation were done with the anticipated result.

Immediately after the operative procedure for arterial insufficiency of the lower legs, attempts at ambulation produced episodes of dizziness and numbness and weakness of the left face, arm and hand. Closer questioning revealed that minor episodes had occurred previously over several years

with more significant symptoms in the six weeks prior to hospitalization. Physical examination demonstrated muscle atrophy of the left arm without weakness, but minor sensory changes were definite. Carotid arteriography demonstrated a plaque at the sinus on the right with characteristic poststenotic dilatation. A nylon shunt satisfactorily bypassed the area of stenosis. Postoperatively, the face and hand were subjectively and objectively improved within 24 hours. Eight weeks after operation, he had returned to useful employment and had no neurologic deficits.

Summary: A 48-year-old male entered for treatment of arteriosclerotic peripheral vascular disease. Operative treatment of the leg lesions was complicated by worsening of previously unrecognized right carotid insufficiency. After a carotid bypass, the patient was sufficiently rehabilitated to return to useful activity.

DISCUSSION

In spite of much interest and many operative attempts, only two successes have been previously recorded in the treatment of internal carotid obstruction. These are the patients of Eastcott, Pickering and Rob [15] and of Edwards and Rob [16] in England. They utilized hypothermia, resection of the obstructed segment, and restoration of continuity by direct anastomosis.

In our series, we have had four completely successful results utilizing a bypass of crimped nylon from the subclavian to the internal carotid. The obstructing segment was not excised and the internal carotid was not permanently occluded above the site of obstruction. Hypothermia was used only in the first case. The total series is summarized in Table I.

One patient (Case 3) proved to have a plaque intracranially with retrograde thrombosis of the carotid in the neck. No shunt was possible and the patient was unchanged after the exploratory procedure.

Another patient (Case 2) entered with carotid and basilar insufficiency. He was considerably relieved of frontal lobe symptoms after a carotid shunt but died of an acute cerebral infarction due to basilar insufficiency four months later.

PATIENT	DURATION	SYMPTOMS	BACK-BLEEDING	DATE OF OPERATION	RESULT
1. ♂ 67	8 MONTHS	HEADACHE AUDIBLE BRUIT EPISODIC PARESIS	0	AUGUST 1956	RELIEVED
2. ♂ 66	5 YEARS	HEADACHE EPISODIC APHASIA EPISODIC PARESIS	0	AUGUST 1956	RELIEVED INITIALLY D. 4 MO. BASILAR ARTERY THROMBOSIS
3. ♂ 64	4 MONTHS	HEADACHE PERSISTENT PARESIS	THROMBOSED	SEPT. 1956 EXPLORATION ONLY	NO IMPROVEMENT SIPHON OCCLUDED
4. ♀ 67	1 MONTH	APHASIA FACIAL PARESIS	+	SEPT. 1956	RELIEVED
5. ♂ 59	2 YEARS	EPISODIC BLINDNESS EPISODIC APHASIA EPISODIC PARESIS	+	DEC. 1956	RELIEVED
6. ♂ 48	6 WEEKS	EPISODIC PARESIS	+	JAN. 1957	RELIEVED

TABLE I-SUMMARY OF PATIENTS

Two patients (Cases 1 and 6) had incomplete occlusions. The first was done with hypothermia and the second with normal body temperature. Both recovered without neurologic deficit.

Two other patients (Cases 4 and 5) had complete occlusions with deficits of partial aphasia. Both recovered without residual neurologic signs.

We believe that critical selection of candidates for this operation is the reason for the successes recorded here. From conversation and correspondence with others interested in this problem, it is apparent that many have explored the carotid in the neck only to find it completely occluded with an organized thrombus. The important difference in this latter group is the presence of an established infarct with permanent and major neurologic deficits at the time of operation. The earlier cases in our series were referred primarily because the episodic frontal lobe symptoms suggested an expanding intracranial lesion, and routine carotid arteriography incidentally revealed obstruction of the internal carotid in the neck. It is apparent that no method of palpation of the pulses in the neck suffices to identify internal carotid occlusion.[14, 37] Prominent pulsation of the temporal artery on the affected side, suggesting an increased flow through the external carotid, has been noted in some cases.[29] It has also been noted that digital compression of the affected carotid produces, at the most, only slight slowing of the heart, whereas compression of

the patent carotid may occasionally, through interference with collateral, produce convulsive twitchings and syncope.[8, 34] A bruit is audible only when the occlusion is incomplete. Since there are no completely reliable clinical signs for the recognition of internal carotid occlusion, adequately early diagnosis must rest upon more frequent use of carotid arteriography in patients with the "little stroke" syndrome. There is reason to believe that the situation is analagous to the pattern of peripheral vascular disease in the extremities where femoral bypass relieves claudication but is rarely possible or helpful when gangrene is present. In other words, carotid bypass should be considered for the patient with "cerebral claudication" before major infarction has occurred.

The experience to date is too limited to answer many questions that naturally arise. It is apparent that coexistent vertebral stenosis requires further study and that the identification of the point of origin of the vertebral as a dominant site for plaque formation [27] suggests further operative bypass procedures. It may well be that hypothermia shall prove necessary in this latter instance, but this series establishes the fact that hypothermia is not necessary for internal carotid occlusion when the flow of blood through the external carotid is maintained during the operative formation of a shunt from the subclavian to the distal portion of the internal carotid in the neck.

CONCLUSIONS

1. Cerebral circulatory insufficiency, usually in the distribution of the middle cerebral artery, may result from segmental occlusion of the internal carotid artery in the neck.

2. Cerebral ischemia associated with segmental carotid occlusion may be relieved by a bypass of the obstruction.

3. Subjective and objective relief of neurologic deficits has been observed after restoration of carotid blood flow by shunt.

4. Symptoms of episodic arterial insufficiency before major infarction has occurred affords maximum opportunity for complete relief.

5. A shunt from the subclavian to the patent carotid distal to the point of obstruction affords effective restoration of carotid flow, and in no way impairs existing or potential collateral blood flow to the brain.

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DR. GALBRAITH: As Dr. Lyons has emphasized, the recognition of this lesion is on a clinical basis, but arteriography has been a great help in the elucidation of the pathology involved, and in planning the therapy.

(Slide) This represents our first case with a plaque at the origin of the internal carotid and with poststenotic dilatation. This patient presented the classical syndrome of internal carotid insufficiency (without thrombosis). As you see, the internal carotid is patent, with good intracranial vessels. This patient had episodes of vertigo, and there was transient motor and sensory deficiency of the contralateral side, and episodes of monocular visual impairment on the ipsilateral side.

(Slide) A second similar case, with the bruit at the site of the plaque, and with slight poststenotic dilatation, and with a good vascular tree beyond the site.

(Slide) This represents a comparable situation in a patient who had a previous asymptomatic thrombosis of the contralateral internal carotid. Here the circulation through the remaining carotid became jeopardized, this carotid supplying anterior and middle cerebral complexes bilaterally.

(Slide) This is the usual picture of a complete thrombosis of the internal carotid. Here the clinical picture is of the same pattern, except that the symptoms are perhaps more accentuated, and there is no bruit. Instead, there is a diminished carotid pulse in the neck on this side. All we see is the external carotid with somewhat dilated common carotid trunk.

(Slide) This represents a common form of collateral circulation where there has been complete occlusion of the internal carotid, with anastomosis through the external carotid and ophthalmic artery into the intracranial portion of the internal carotid. This highlights 2 points we feel are important in devising a bypass.

First of all, as Dr. Lyons has pointed out, the bypass can be carried out here without having to interrupt the cerebral circulation, obviating the need for hypothermia. Secondly, it does not in any way jeopardize the external carotid flow, either temporarily or permanently, and we feel that it is important that the external carotid be preserved in order to maintain a collateral flow that is present, or to have a potential collateral flow present, particularly as regards the retina.

(Slide) This is a case that, at operation, was found to have a thrombus which we considered to be retrograde, from a plaque in the syphon down into the neck. There was no patent distal segment. Here we see the initial segment of the carotid is perfectly normal. There was no plaque in the neck.

We think that possibly this type of arteriographic depiction might indicate a retrograde thrombus, but we would still consider exploration of this type of case warranted, in order to bypass it, possibly, if the thrombus has not extended all the way up.

(Slide) This is a thrombus, intracranially, that we think is probably the underlying pathology of the previous slide, where there was retrograde thrombus in the neck. This type of case, of course, is not amenable to any shunt that we have at present. [Applause]

DR. DEBAKEY: Mr. Chairman, members and guests of the Association, I think that Dr. Lyons and his associates deserve commendation for bringing to our attention this important form of occlusive disease, and the methods by which it can be corrected. Experience now with this problem is still limited, and ours, like others, is not sufficiently great to generalize. However, this experience does suggest a certain pattern to this form of occlusive disease which is not entirely dissimilar from the patterns assumed in the more common forms of occlusive disease, namely those involving the lower extremities.

Our experience with this form of segmental occlusive disease (and I think that this concept of the segmental or localized nature of the occlusion is of considerable significance in terms of therapy) in the lower extremities based upon more than 500 cases suggests that there are 2 broad groups, namely the aorta-iliac type and the femoral type.