CAROTID INSUFFICIENCY—DIAGNOSIS AND SURGICAL TREATMENT
A REPORT OF TWENTY-ONE CASES*
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(Received for publication June 2, 1958)

CAROTID insufficiency is one of the most important problems that neurosurgeons have ever faced. As a cause of disability, invalidism and loss of human dignity, it has few equals. The solution of this pressing problem is a challenge to neurosurgeons everywhere.

At present the exact incidence of this condition is not known with certainty. However, Fisher's$^{10-12}$ magnificent work on this subject as well as mounting evidence from other sources$^1,9,14,16,22,24,26,27,31$ leaves little doubt that it is one of the common causes of cerebrovascular accidents. In 432 routine unselected autopsies in adults, Fisher$^{12}$ found occlusion of one or both internal carotid arteries or severe stenosis of one or both of the internal carotid arteries at the bifurcation in the neck in approximately 10 per cent of cases. Furthermore, a clinical pathological study of 45 cases of occlusion of the internal carotid at the bifurcation in the neck revealed that in 85 per cent severe neurologic disturbances were produced. Lofstrom, Webster and Gurdjian$^{19}$ in 100 consecutive cases of hemiparesis or hemiplegia found internal carotid occlusion or severe stenosis to be present in 29 cases.

In a six-month period, March to September 1957, after certain changes had been made in our arteriographic apparatus, the most important of which was the use of a flexible nonmetallic cassette in which the film comes within one-eighth of an inch of the patient's shoulder, we have been able to show the bifurcation of the carotid artery in the neck in the great majority of all carotid arteriograms without additional injections. In 249 carotid arteriograms in 174 patients, 43 patients and 51 arteries showed some degree of stenosis or occlusion in the internal carotid at the bifurcation in the neck which was thought to be the cause of symptoms of which 24 of the patients complained (Table 1).

By all odds the most common cause of occlusion or stenosis is an arteriosclerotic plaque which begins in the carotid sinus at the bifurcation of the artery in the neck and slowly encircles the intima and occludes the artery.$^{12}$ In addition, a superimposed thrombus on an ulcer in the plaque may be the final occluding factor, or hemorrhage into the plaque may suddenly occlude

* Presented at the meeting of the American Academy of Neurological Surgeons, Sea Island, Georgia, November 13, 1957.
the vessel. It is worth noting that an arteriosclerotic plaque at this segment of the vessel is more common than in any other artery of the body except the abdominal aorta. Fisher states that if there is a segmental stenosis or occlusion in the neck, there is much less likely to be arteriosclerotic disease in the intracranial branches of the carotid tree and our observations confirm this finding. DeBakey has reported a similar situation in the iliacs and femorals distal to occlusions of the abdominal aorta. Other causes of occlusion or stenosis of the carotid in the neck are trauma, dissecting aneurysm from the aorta, arteriosclerotic occlusion of the carotid or innominate as they arise from the arch of the aorta, saddle emboli from the heart, arteritis, and pressure by tumors or infections in the neck.

The sequence of events that follow occlusion or severe stenosis of the internal carotid artery is finally coming to light. At the present time these events may be generally classified as ischemic, embolic or thrombotic.

### TABLE I

*Incidence of carotid artery disease in a six-month period, March to September 1957, in 249 consecutive carotid arteriograms on 174 patients*

<table>
<thead>
<tr>
<th>Detected carotid artery disease</th>
<th>43 patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total occlusion</td>
<td>5</td>
</tr>
<tr>
<td>Severe stenosis (60% or more)</td>
<td>12</td>
</tr>
<tr>
<td>Moderate stenosis (30% or more)</td>
<td>14</td>
</tr>
<tr>
<td>Mild stenosis</td>
<td>20</td>
</tr>
<tr>
<td>Bilateral disease</td>
<td>8</td>
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Whether or not occlusion or stenosis of one internal carotid artery without resulting embolus or propagating thrombus will produce symptoms depends upon the adequacy of the collateral circulation in the head as well as the patency of the other carotid and vertebals and the stability of the blood pressure. If the blood pressure drops in the presence of inadequate collateral circulation, transitory symptoms may develop. Should further stenosis of the carotids and/or vertebals develop, a state of chronic ischemia may lead to gradually progressive senility and/or hemiparesis.

Following occlusion of the internal carotid in the neck, the most common finding is a thrombus which forms distal to the plaque and which may propagate upward to occlude the middle or anterior cerebral arteries or a portion of the thrombus may break off from movement of or pressure on the neck and float upward as an embolus, occluding major or minor vessels in the head. There is also good reason to believe that mural thrombi form just distal to the point of severe stenosis and break off as emboli which may occlude large or small vessels in the head.

Fisher states that prior to angiography there had grown up a classical concept of thrombosis of the internal carotid artery, namely, monocular blindness with contralateral hemiplegia. We agree with him that this is
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much too narrow a concept and that the disease can produce a rich and varied symptomatology which ranges from mental deterioration through strokes to mimic brain tumor, hemorrhage, multiple sclerosis and a whole gamut of so-called degenerative diseases. Strangely enough, convulsive seizures have been quite rare in our cases. Fortunately, the onset is usually heralded by prodromata, a fact of greatest significance. It is of utmost importance to recognize the condition at this stage before occlusion of a major vessel in the head has taken place with resultant permanent brain damage. Less commonly the onset may be suddenly catastrophic. Johnson and Walker\(^{16}\) reported that in 35 per cent of their cases there was such an onset. It is our impression that this percentage is too great because only one of the patients (Case 1) in this report had such an onset. The onset may also be gradual with mental changes or progressive hemiplegia, not unlike an expanding lesion. The frequency of personality changes and so-called psychotic reactions in the middle and older age group lead us to believe that psychiatric evaluation of such patients will in the future of necessity include study of the carotid circulation.

So far as is known, there are no purely clinical neurologic findings upon which one could base a diagnosis of internal carotid occlusion with certainty. However, certain additional maneuvers together with the history and neurologic picture may allow positive diagnosis in some cases. Palpation of the artery may reveal a complete lack of pulse in the entire carotid on one side of the neck and one may even feel the pulseless artery reduced to a cord. However, palpation of the internal carotid at the bifurcation or in the tonsillar fossa\(^{1,29}\) is usually unreliable because it is difficult to separate the pulses of the internal and external carotids. Auscultation may be of value in a high-grade stenosis in which one may hear a systolic bruit over the bifurcation. Another maneuver of considerable importance, according to Gurdjian and Webster,\(^{14}\) is compression of the opposite common carotid artery. After 15 or 20 seconds syncope or convulsion may occur if there is carotid insufficiency on the other side. Care should be taken to compress the common carotid below the carotid sinus because hypersensitivity of the structure may confuse the issue. It has been suggested\(^{22,30}\) that the use of the ophthalmodynamometer with which the blood pressure in the eye is measured may be helpful in these cases. It is well known that if one carotid artery is occluded, pressure above the point of occlusion and in its branches is usually reduced 40 to 50 per cent. The finding of a significantly reduced blood pressure in the eye on the side under suspicion is suggestive of occlusion or severe stenosis of the carotid, but, of course, does not localize the site of obstruction in the artery.

At present arteriography is the \textit{sine qua non} in the diagnosis of carotid insufficiency. It is our conviction that with few exceptions every patient who has had a cerebrovascular accident which apparently involves the carotid circulation should have bilateral carotid arteriography as soon as compatible with the patient’s condition. If the lesion is not found in the
carotid circulation and reasonable doubt exists as to its location, vertebral arteriography should be carried out. There can no longer be any doubt that when carotid arteriography is carried out the bifurcation in the neck should be visualized in all cases when any arterial disease in the head or neck is suspected and when any surgery on any part of the carotid tree is anticipated.

The arteriogram of occlusion or stenosis of the internal carotid in the neck is usually clear-cut, but in cases in which the obstruction is in the intracranial portion of the carotid artery, one may be misled. In such cases the dye is unable to go up into the internal carotid in the neck or goes up very slowly if the occlusion is above the ophthalmic artery. Fortunately the arteriogram in such a case is fairly characteristic.*

As effective as the medical treatment of this condition may be in some cases, we are concerned here only with attempts at restoration of normal blood flow to the brain by surgical means. So far, five methods have been attempted: (1) attempt to by-pass the point of obstruction by anastomosing the external to the internal carotid above the obstruction in the internal carotid, (2) resection of the occluded segment with an end-to-end anastomosis, (3) resection of the occluded segment with insertion of a graft, (4) by-pass of the occluded segment by a prosthesis and (5) endarterectomy. We have been able to find a total of 24 cases in the literature\(^3,4,6-8,18,20,23,32,33\) in which the authors claim to have re-established the carotid blood flow. In only 5 of these\(^2,6,18,23,33\) however, were postoperative arteriograms carried out to prove the patency of the artery and in one of these\(^6\) the arteriogram was made on the operating table. Postoperative arteriography is extremely important in establishing the patency of the circulation because, (1), as Jackson\(^15\) has pointed out, patients may improve in the face of persistent occlusion and, (2), if re-occlusion has occurred, either re-operation or anti-coagulant therapy may be necessary.

So far, our efforts have been confined to endarterectomy because, (1), it is by all odds the simplest procedure, (2), one does not have to sacrifice the collateral circulation to the brain through the external carotid and ophthalmic and, (3), we have an aversion to the use of foreign materials in the body. Cases will probably be encountered in which the plaque has destroyed the muscularis and the remaining wall of the vessel will be so fragile that it would not stand suture or pressure. In such cases a prosthesis or graft might be necessary, but so far we have not encountered such a case. We would not be inclined to use a by-pass in a case of severe stenosis. It is our feeling that although this would undoubtedly relieve the ischemia, it would not remove the lesion responsible for the embolic phenomena which are so catastrophic in these cases.

It should be made clear that the primary objectives of this operation are two, to relieve ischemia and to prevent infarction from thrombosis and em-

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* See Murphey and Shillito this issue (p. 24).
Fig. 1A. Exposure and incision of internal carotid artery showing a clot distally and the plaque proximally inside the artery.

Fig. 1B. Method of removing the plaque.

Fig. 1C. Lumen of the artery after endarterectomy. Note suture through distal intima.

Fig. 1D. Closure of the artery.
bolism. To obtain these objectives, it is important that the diagnosis be made and surgery performed during the stage of transitory symptoms and before, of course, permanent brain damage has occurred. In the present stage of our knowledge, operation would be indicated (1) in cases of complete occlusion or severe stenosis with transitory symptoms, (2) complete occlusion or severe stenosis with emboli to smaller vessels from which the patient may be reasonably expected to recover, (3) bilateral disease as in (1) and (2), and (4) bilateral disease with early mental deterioration. It might also possibly be indicated in cases of unilateral occlusion or severe stenosis with early mental deterioration or progressive neurologic deficit, particularly if there is evidence of insufficiency of the circle of Willis. It probably would be futile to restore circulation in the neck after occlusion of the major vessel in the head, such as the middle cerebral, but one may well ask how, in the presence of complete occlusion in the neck, can one be sure that there is also major occlusion in the head, and we cannot answer this. Furthermore, there is reason to believe that if complete occlusion has persisted for more than a week, it is unlikely but not certain that the distal clot would be completely organized so that removal would be impossible. The difficulty, of course, is in determining when occlusion took place.

At the present time patients suspected of having carotid insufficiency are handled in the following manner.

In addition to routine neurologic study which includes roentgenograms of the skull, spinal fluid examination and electroencephalogram, palpation, auscultation and compression of the carotid arteries are carried out followed as soon as possible by ophthalmodynamometry and arteriogram. If the arteriogram shows occlusion or severe stenosis of one or both internal carotids in the neck, the patient is placed on intravenous heparin immediately and operated upon the following morning. Heparin is discontinued 2 hours before operation and Hedulin, 300 mg., is begun before operation.

The operation is carried out under local anesthesia so the patient's neurologic status may be observed at all times. A shunt is kept ready in case the patient's neurologic status changes for the worse after a stenosed artery has been clamped off. So far, its use has been unnecessary. As a matter of fact, in 2 cases of old, complete obstruction on one side and severe stenosis on the other, operation was carried out on the stenosed side by simply raising the blood pressure and lowering the patient's head without any change in the patient's condition. If this simple procedure is followed, a shunt or hypothermia will rarely be necessary.

The common carotid artery is first exposed through a vertical incision along the anterior border of the sternocleidomastoid muscle and a Poppen-Blalock clamp is placed on the artery as soon as it is exposed to test further the adequacy of the collateral circulation and to minimize the possibility of dislodgment of an embolus. One cc. of heparin solution (10 mg. in 50 cc. of saline) is then injected into the artery proximal to the clamp. The bifurcation of the artery and approximately 1½ inches of the internal and external carotids are exposed by sectioning the digastric muscle and retracting the hypoglossal nerve upward. A small bulldog clamp is then placed on the external carotid. Usually a silver clip is necessary to occlude the superior thyroid. One cc. of heparin solution is then injected into the external
carotid artery distal to the clamp. In cases of complete occlusion of the internal carotid artery, no clamp is placed on this vessel. The internal carotid is then opened vertically just above the easily palpable plaque where almost invariably one will find a clot (Fig. 1A). If the clot is not too well organized, retrograde pressure from across the circle of Willis will usually blow the clot out through the incision in the artery. If this does not occur, then a catheter may be inserted and suction applied in an attempt to obtain a retrograde flow. If this fails after several attempts, the operation is abandoned. If, however, retrograde flow is obtained, a bulldog clamp is placed on the internal carotid above the incision and 1 cc. of heparin solution is injected into the artery distal to the clamp and the incision is then extended downward over the plaque and slightly below it. A cleavage plane between the intima and media is found just above the plaque and developed (Fig. 1B). This undoubtedly is the most crucial step in the operation. Review of our surgical specimens reveals that in some cases we tended to take a little media. Nevertheless, the proper plane can and should be developed without difficulty; in fact, we have been amazed at the ease with which the plaque may be shelled out. We have found the best instrument with which to accomplish this is a strabismus hook with a ball point the size of a BB shot on the end of it. If the proper plane is developed, then little if any muscularis will be removed and the remaining wall of the vessel will be sufficiently strong to hold the sutures and withstand the arterial pressure. We have always been concerned that the intima in the distal segment may be undermined by the force of the blood flow and occlude the artery again. Therefore, after the plaque has been dissected free from the media, the distal intima is elevated in one piece and amputated evenly with a knife. It is then sewed to the remaining wall of the vessel with four through-and-through No. 00000 silk sutures, having been introduced from the outside and placed equidistant around the artery (Fig. 1C). Closure of the arteriotomy (Fig. 1D) is carried out with a continuous running suture of No. 00000 arterial silk on a swedged round needle with the stitches being placed no more than 1 mm. apart. During the closure the interior of the artery is irrigated frequently with heparin solution and just before closure is completed, all clamps are removed separately and, of course, temporarily in order to flush out any clots that may have formed distal or proximal to the clamps as the case may be. Additional heparin is injected distal or proximal to the clamps as before. The endarterectomy removed segment is then re-irrigated with heparin solution and closure is completed. The order of removal of the clamps is important. The clamp on the external carotid is removed as soon as suture is completed in order to test the suture line. Even if the suture is quite adequate, there will usually be several small jets of blood from the suture line which will eventually stop. Additional sutures should be used only when large jets of blood come through the suture line. If one is satisfied with the suture, the common carotid should then be released immediately in hopes that if any thrombi are present, they will be blown up the external carotid, and quickly thereafter the clamp on the internal carotid is removed. Heparin is then started intravenously while the wound in the neck is being closed and continued until long-acting anticoagulants become effective. We cannot overemphasize the fact that hemostasis should be meticulously secured before closure of the wound in the neck is begun. We have found that the use of a drain for 24 hours minimizes the edema in the neck. The patient is usually discharged from the hospital at the end of 1 week. The long-acting anticoagulants are then continued for a month, at the end of which time the patient is brought back to the hospital for a postoperative arteriogram.
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We have attempted to restore the circulation in 22 arteries in 21 patients (Table 2). In 21 cases the carotid insufficiency was the result of arteriosclerosis and in 1 case it was caused by a subintimal clot from trauma. Fourteen arteries were completely occluded and in 8 there was severe stenosis. In only 6 of the 14 complete occlusions were we able to obtain a retrograde flow, which means, of course, that the operation was carried out too late in these 8 cases. Retrograde flow was obtained in all cases of stenosis. Of the total of 14 arteries in which a retrograde flow was obtained and circulation was restored, 7 were proved to have remained open as long as a month after operation, 1 for 2 weeks after operation, 1 for 10 days after operation, and 1 for 1 day after operation, all by arteriography. In 1 case the artery was proved to be occluded at the bifurcation in the head by arteriography at the time of operation after flow had been re-established in

**TABLE 2**

<table>
<thead>
<tr>
<th>Surgical results of endarterectomy</th>
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<tbody>
<tr>
<td>Number of patients operated upon</td>
</tr>
<tr>
<td>Number of arteries operated upon</td>
</tr>
<tr>
<td>Total occlusions</td>
</tr>
<tr>
<td>Retrograde flow obtained</td>
</tr>
<tr>
<td>No retrograde flow obtained</td>
</tr>
<tr>
<td>Severe stenosis</td>
</tr>
<tr>
<td>Retrograde flow obtained</td>
</tr>
<tr>
<td>Circulation restored</td>
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</tbody>
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the neck. Presumably the retrograde flow obtained in this case was from the posterior communicating or ophthalmic arteries. In 3 cases the arteries thrombosed after surgery. No anticoagulants were used in these cases. In the 10 arteries that remained patent, anticoagulants were used in all except the traumatic case (Case 1).

Much to our chagrin after re-establishing the circulation in 2 patients (Cases 1 and 3) who had complete hemiplegia preoperatively, we found on repeated carotid arteriograms complete occlusions of the middle cerebral arteries, apparently from emboli that had occurred before operation. In these 2 cases while some improvement has occurred, it certainly could not be attributed to the surgery. In the remaining 7 cases with 8 arteries, 2 patients have returned to normal and 3 patients (Cases 2, 6 and 7) have only slight residual impairment. In 1 patient (Case 8) with complete occlusion on one side and extreme stenosis on the other, operated upon 3 weeks ago, there has been no improvement. None of these has had any further episodes suggesting ischemia or embolism. A final patient (Case 9), a woman who had complete hemiplegia 2 days after arteriography and in whom endarterectomy was carried out with some transitory improvement, died 2 days after operation. Necropsy revealed the artery to be patent, but the right hemisphere was infarcted.
It is a dangerous matter to draw any sweeping conclusions on the basis of such a small series of cases. However, it is our belief, at the present time at least, that in the majority of cases endarterectomy is the operation of choice although, as we pointed out above, circumstances may well demand other procedures and one should certainly be ready to use whatever means are necessary to re-establish the blood flow in the carotid. Furthermore, we are by no means certain that anticoagulants are necessary postoperatively. However, we have a very strong feeling that we would much prefer to deal with a postoperative hemorrhage in the neck than a postoperative thrombosis at the operative site, particularly if this is associated with an embolus to one of the major vessels in the head. It is certainly obvious, however, that if this or any other operative procedure on the carotid artery directed toward re-establishing circulation to the brain is to be worth while, it must be performed before the propagating thrombus has organized to the point that it cannot be removed and before the patient has sustained permanent brain damage.

CASE REPORTS

Case 1. M.E.D. (#206330). A 16-year-old male Golden Glove boxer was admitted on Jan. 19, 1956, 1 day after having been defeated by a decision in a Golden Glove boxing bout. He was not knocked down or rendered unconscious. He went home without complaint. The next morning, however, he was unable to get out of bed and could not use his right side. He became progressively stuporous. The family was able to arouse him at intervals during the day, but he had been unable to speak.

Examination revealed a stuporous, uncooperative patient with right hemiplegia and aphasia without evidence of increased intracranial pressure.

A left carotid arteriogram (Fig. 2A) revealed an incomplete occlusion of the internal carotid artery at the bifurcation in the neck. The left carotid artery was explored and a subintimal clot just above the bifurcation (which by the time of surgery had completely occluded the artery) was evacuated and a retrograde flow was obtained. The artery was closed. There was no immediate postoperative change in the condition of the patient.

By the 18th postoperative day he was alert and eating fairly well, but there had been only minimal improvement in his hemiparesis. At that time a postoperative left carotid arteriogram (Fig. 2B) was performed and revealed the artery in the neck to be patent. However, there was complete occlusion of the middle cerebral artery presumably from a preoperative embolus.

He was last heard from 21 months later at which time his family physician reported he had an impaired, but useful speech and could walk with a spastic gait, but had no significant improvement in the arm—altogether about the improvement one would expect from an occlusion of the middle cerebral artery. There is no reason to believe he was benefited by the operation.

Case 2. W.T. (#207359). A 50-year-old white male was admitted on Feb. 1, 1956 with a chief complaint of aphasia and right hemiplegia of 1 week’s duration. His present illness began 3 or 4 months previously with onset of attacks of numbness of the right arm and leg, which he would rub violently during the few minutes that
these attacks lasted. A week before admission, while working at his desk in a hospital, there was sudden development of right hemiplegia and aphasia. In the intervening week he had recovered some power in the leg and was beginning to move his arm and fingers. There had been no improvement in his speech.

Examination revealed an alert, but very emotional middle-aged individual. There was a complete expressive aphasia, but there was reason to believe he understood most, if not all, of what was said. There was no hemianopia. There was, however, a right central facial weakness and marked weakness of the right arm and leg, but there was some power in all of the muscles in these extremities. No sensory deficit could be elicited. Palpation of the carotid artery in the neck revealed no definite abnormality.

The day following admission bilateral carotid arteriograms (Fig. 3A) showed complete occlusion of the internal carotid artery on the left. There was collateral circulation through the external carotid and ophthalmic arteries which allowed faint filling of the intracranial carotid circulation showing apparent occlusion of the ascending frontoparietal artery, no doubt the result of a small embolus from a thrombus distal to the point of occlusion in the neck. There was a marked stenosis of the internal carotid on the right at the same level (Fig. 3B). On Feb. 6, 1956 carotid endarterectomy was performed. Heparin and Hedulin were administered for 2 days, at the end of which time the prothrombin time was at the desired level and heparin was discontinued.
Postoperatively there was rapid improvement in the power in the arm and useful return of speech. On Feb. 21, 1956, left carotid arteriogram showed the internal carotid artery to be patent (Fig. 3C). There was a fusiform dilatation of the site of the operation and a slight constriction in the internal carotid artery distal to this dilatation which represented the upper limits of the endarterectomy.

By this time the patient could converse reasonably well and his problem of
stenosis of the right internal carotid was discussed with him. It was his desire to have this plaque removed, so on Feb. 24, 1956 an endarterectomy on the right was performed. The same anticoagulant regime was instituted.

Postoperatively his course was uneventful and he was discharged from the hospital March 2, 1956, a week after the second operation.

The patient returned 4 weeks later for right carotid arteriogram (Fig. 3D) which showed the vessel to be open. He was discharged the following day to continue his anticoagulants.

The patient was last seen approximately 21 months after his first operation. He had done fairly well, his speech had continued to improve and he could converse slowly without loss of words. He stated when he attempted to speak rapidly or became excited, his speech would become difficult. He had had one convulsive seizure 19 months postoperatively, presumably the result of the healed infarct. Examination revealed a useful speech with a minimal expressive aphasia. The cranial nerves were normal. The only residual of his hemiparesis was slight hyperactivity of the deep reflexes. No bruit could be heard over the carotid arteries and palpation revealed normal pulsation and no evidence of unusual dilatation.

Case 3. S.J.S. (#226574). A 71-year-old white male was admitted on Aug. 30, 1956, having been in an automobile accident and rendered temporarily unconscious 36 hours previously. Approximately 6 hours before admission a left hemiplegia had developed quite rapidly, and it had been thought by his family physician that he had an intracranial hemorrhage. His past history was significant in that 2 months previously he had an episode of transitory amnesia during which he became lost for 2 hours while driving his automobile.

Examination revealed a stuporous, elderly man with complete left hemiplegia. There was some tenderness over the right carotid bifurcation. Vital signs were within normal limits. Spinal fluid examination showed normal pressure without significant abnormalities in the fluid. Roentgenogram of the skull was negative.

A right carotid arteriogram (Fig. 4A) showed complete occlusion of the internal carotid artery just above the bifurcation in the neck. It was our opinion that there was little likelihood that re-establishment of the circulation would result in improvement. However, after discussion with the family, it was decided to explore the artery. On Sept. 1, 1956 the artery was exposed and an arteriosclerotic plaque was found completely occluding the artery for a distance of approximately 1 cm. Distal to the plaque was a small thrombus and when this thrombus was removed, there was a good retrograde flow from the head. The plaque was removed and the artery was closed. Intravenous heparin was started. Two weeks later the arteriogram was repeated (Fig. 4B) which showed the artery to be patent in the neck, but the middle cerebral artery at the bifurcation of the internal carotid intracranially was occluded, no doubt the result of an embolus from the thrombus distal to the point of occlusion in the neck.

Postoperatively there was gradual, slow improvement.

The patient was last heard from a year postoperatively. He had learned to walk, but with the typical gait of a hemiplegic. The left arm was useless.

As can be seen, this is about the usual recovery one would expect from a completely occluded middle cerebral artery and had nothing whatever to do with the re-establishment of the carotid circulation.

Case 4. W.S.M. (#243736). A 67-year-old white male was admitted on March 3,
1957 because of intermittent episodes of numbness and weakness in his right arm and leg and progressive mental deterioration. Four years previously he had been in an automobile accident and as a result pain had developed in the arm. A few weeks subsequent to the accident he had an attack of acute depression. Following this there were intermittent episodes of numbness and weakness of the left side of the body for a year or so, which finally disappeared. His present illness began 1 year before admission when the patient's family noticed progressive mental de-

erioration and episodes of confusion. Approximately 3 months before admission he began to have episodes of transitory numbness and weakness of the right arm and leg.

Examination revealed an elderly man with mild mental confusion, related particularly to time and occurrence of recent events. The right common carotid artery and bifurcation were hard, cord-like and pulseless. There was a bruit over the left carotid bifurcation. Compression of this artery for more than a few seconds resulted first in dizziness and then loss of consciousness. The rest of the findings were negative except for generalized hyperactivity of the deep reflexes, greater on the left, and bilateral Hoffmann's sign.

A diagnosis of carotid occlusion on the right and carotid stenosis on the left was made and on March 4, 1957 a left carotid arteriogram (Fig. 5A) revealed stenosis of two-thirds of the internal carotid artery at the bifurcation in the neck. Two days later vertebral angiography (Fig. 5B) was performed which was interpreted as
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Fig. 5. Case 4. (A) Marked stenosis of left internal carotid artery in the neck. There was complete occlusion of right common, external and internal arteries. (B) Vertebral arteriogram showing adequate vertebral circulation and partial filling of anterior and middle cerebral arteries. (C) Postoperative arteriogram showing patency of left internal carotid artery. Subsequent films (not shown) revealed normal intracranial carotid circulation.

being normal and, furthermore, filled the carotid circulation poorly on both sides. Because of the acute carotid insufficiency when the left carotid was compressed, we were afraid that the artery could not be occluded for an operation without severe brain damage. A shunt was prepared and on March 8, 1957, under local anesthesia, the common carotid was exposed and a Poppen-Blalock clamp was applied, surprisingly without loss of consciousness or hemiplegia. This in all probability was the combined result of the recumbent position and increased blood pressure inci-
dent to injection of procaine around the carotid sinus. Endarterectomy was then performed.

Postoperatively the patient was put on heparin and Hedulin was started. The following day the prothrombin time was high enough to allow discontinuation of the heparin. His postoperative course was uneventful. There was marked improvement in his mental status, in fact his family stated he was perfectly normal. He had no more episodes of hemiparesis.

The patient was re-admitted 1 month later for a postoperative carotid arteriogram (Fig. 5C) which showed the artery to be patent and in good condition.

![Image](image_url)

**Fig. 6. Case 5.** (A) Marked stenosis of right internal carotid artery. Intracranial carotid circulation (not shown) revealed absence of ascending frontoparietal branch of right middle cerebral artery. (B) Postoperative arteriogram showing patency of right internal carotid. Intracranial circulation (not shown) revealed no change.

Six months postoperatively he had had no further mental disturbance. He had had one episode of mild dizziness and was examined by his internist who found his blood pressure to be "low." He has had no episodes of hemiparesis. Examination revealed he was mentally clear and alert, and he had no abnormal neurological findings. Ophthalmodynamometric readings showed the left eye to be 80, the right eye 40.

**Case 5. L.B. (§254042).** A 75-year-old white female was admitted on June 10, 1957 with a left hemiparesis. Twenty-four hours previously she had had sudden onset of weakness of the left arm and a few minutes later she collapsed to the floor. By the time of admission she had improved considerably. Past history was of interest in that for 2 years she had had intermittent episodes of dizziness and a feeling as though she were going to faint.
Examination revealed an elderly lady, somewhat mentally confused, with minimal dysarthria and marked left hemiparesis, more severe in the arm.

On the 2nd hospital day bilateral carotid arteriography was performed which revealed bilateral carotid stenosis, more severe on the right side (Fig. 6A) and apparent occlusion of the ascending frontoparietal branch of the middle cerebral artery, presumably the result of an embolus which originated just distal to the area of stenosis in the neck. In hopes that further emboli could be prevented, endarterectomy was performed on the right side with removal of a large plaque, and postoperatively she showed gradual improvement in speech and power of the left arm and leg. She was discharged 2 weeks later with only slight residual weakness in the arm and leg and normal speech.

At the time of her re-admission on July 23, 1957 for repeated arteriography, she was clear, mentally alert and could speak distinctly and coherently. There was slight residual hemiparesis. The arteriogram revealed the artery to be patent (Fig. 6B). She was discharged the following day. She was last seen 4 months postoperatively at which time she was essentially normal and had had no further difficulty.

Case 6. E.F.H. (#254304). A 65-year-old man was admitted June 12, 1957 with a moderate expressive aphasia. Approximately 2 years previously he had had sudden onset of numbness and clumsiness of his right hand followed by weakness which cleared. Two weeks before admission he had awakened with an incomplete aphasia which had improved slightly.

Examination revealed a mentally clear elderly man with a moderate expressive aphasia. On his 2nd hospital day bilateral carotid arteriography (Fig. 7A) revealed a very high-grade stenosis in the internal carotid artery in the neck on the left and an occlusion of the ascending frontoparietal artery on the left. Right carotid arteriogram revealed minimal carotid artery disease. Vertebral arteriogram was performed, revealing normal-appearing vertebral, basilar and posterior cerebral vessels. On the 5th hospital day left carotid endarterectomy was performed with removal of a large plaque.

Postoperatively his course was uneventful and he was discharged on the 16th postoperative day with moderate improvement in his aphasia.

He was re-admitted for follow-up arteriogram on July 24, 1957. His aphasia was markedly improved in that he could complete sentences and got into difficulty only when he attempted to speak rapidly. Repeated left carotid arteriogram showed the vessels to be patent (Fig. 7B). He was discharged the following day.

He was seen 4 months postoperatively at which time there was only slight hesitation in speech with a prolongation of words when speaking rapidly.

Case 7. D.B.W. (#260119). A 67-year-old white male was admitted Aug. 4, 1957 approximately 36 hours after he had fallen to the floor on his hands and knees and told his wife he felt “drunk.” He was able to get in bed with her help and said that he was very dizzy and even worried about falling from the bed. He then went into deep sleep from which his wife could not arouse him. He was taken immediately to the local hospital where he was unresponsive on admission. Approximately 6 hours after his admission to the local hospital he aroused and spoke to the family by name, but was confused about place and events. After only a few minutes, he again lapsed into semicoma. He remained unresponsive from this point until a few minutes before admission to this hospital when he again aroused and began talking with the family, but was still very confused and lethargic.
His past history was significant in that during the past few months he had some memory deficit and personality change.

Examination revealed a very confused elderly man with no localized neurologic deficit. Both carotid pulsations were palpable and equal. Spinal fluid revealed normal pressure without significant abnormalities. Roentgenograms of the skull were negative. Bilateral carotid arteriograms (Fig. 8A) under local anesthesia revealed bilateral internal carotid artery disease with fairly marked stenosis on the right side and moderate stenosis on the left, both being at the bifurcation in the neck. We were by no means certain that the bilateral stenosis that had been demonstrated in this patient was sufficient to produce his symptoms. In fact, it seemed probable that his vertebral circulation was involved as well. He was observed for several days during which time his mental status did not change and on Aug. 10, 1957, after considerable deliberation, it was elected to do a right carotid endarterectomy. This was carried out without difficulty. Intravenous heparin was begun and continued for 24 hours until the oral anticoagulants became effective. There was immediate marked improvement in his mental state. In fact, by the time he returned from the operating room he was quite clear and talkative. At the time he was discharged from the hospital on the 9th postoperative day, the family stated he was mentally normal. However, he still complained of dizziness.

One month later he returned for postoperative arteriogram which showed the artery to be patent (Fig. 8B). He had a partial amnesia for a part of his previous hospital stay. He still complained of some dizziness, but was otherwise normal.
Case 8. R.A.B. (267370). A 59-year-old white male was admitted Oct. 15, 1957 because of a left hemiplegia of 1 year's duration and progressive mental deterioration and progressive aphasia for about 3 months. His illness began in June, 1956, when he had intermittent bouts of clumsiness and falling associated with transitory left hemiparesis which would last approximately 5 minutes. By November, 1956, a persistent left hemiparesis had developed. Approximately 3 months before we saw him, there developed considerable difficulty with speech, mental deterioration and emotional instability.

Examination revealed an elderly man who cried intermittently during the taking of the history and the examination. He was able to answer only simple questions. He had an incomplete global aphasia, greater in the expressive realm. There was marked left hemiparesis, most marked in the arm and hand. The internal carotid pulsation on the right seemed to be absent and there was a decrease of pulsation above the bifurcation on the left. There was a definite systolic bruit over the left bifurcation. On Oct. 16, 1957 bilateral carotid arteriograms (Fig. 9A and B) demonstrated complete occlusion of the right internal carotid artery at the bifurcation in the neck and a 95 per cent stenosis of the left internal carotid as well as absence of the left external carotid. It was realized that little if anything could be done for this man. Certainly there was no chance of improving the left hemiplegia. However, we were not so certain that re-establishing the flow on the left might not help
his speech. The matter was discussed with the family and it was decided to attempt left endarterectomy. This was carried out without difficulty. The patient was placed on intravenous heparin which was continued for 24 hours until long-acting anticoagulants became effective. The patient’s postoperative course was complicated by the onset of intractable hiccoughs which persisted for approximately 3 days. There was no essential improvement in his speech or emotional instability.

On Oct. 28, 1958 a postoperative left carotid arteriogram (Fig. 9C) demonstrated both the internal and external carotids to be patent with good flow to the intracranial vessels. He was discharged 2 days later with no significant improvement.

![Fig. 9. Case 8. (A) Marked stenosis of left internal carotid artery in the neck. (B) Complete occlusion of right internal carotid artery in the neck. (C) Postoperative arteriogram showing patency of left internal carotid artery. (See text.)](image)

**Case 9. M.L.B. (#268680).** A 67-year-old white female was admitted Oct. 24, 1957 in a state of mental confusion with no evidence of paralysis. According to her family she had been mildly hypertensive for approximately a year and had something described as a “small stroke” approximately 4 months prior to admission. Since that time she had had some mental confusion, but had had no particular complaints. Three days prior to admission mental confusion became marked and she was unable to recognize members of her family.

Examination showed a mentally confused, elderly woman who was uncooperative. There were no other significant neurologic findings. Bilateral carotid arteriography (Fig. 10A) demonstrated 80 per cent stenosis of both carotid arteries in the neck. Arteriosclerotic plaques were noted throughout the intracranial vessels; particularly there was marked narrowing of both anterior cerebral and middle cerebral arteries. Two days following arteriography, on morning rounds she was found with
a complete left hemiplegia. She was taken to surgery immediately, endarterectomy was performed and a retrograde flow was obtained. On returning to the ward there was no essential change in her condition. In approximately 12 hours there was some return of function in the arm and moderate return of function in the leg. The following morning, however, she again was found with a complete left hemiplegia, and another right carotid arteriogram (Fig. 10B) revealed that the internal carotid artery and middle and anterior cerebral arteries on the right were still patent. The patient expired 12 hours later.

At autopsy the pertinent findings were referable to the entire brain. There were multiple areas of encephalomalacia, many of which were old, but there was a new infarction which involved most of the right parietal lobe. There was severe generalized arteriosclerosis, particularly in the anterior and middle cerebral arteries. The right common carotid artery was patent at the site of the endarterectomy. In retrospect there is considerable doubt whether the carotid insufficiency was the cause of her symptoms. It is more likely that insufficiency of the anterior and middle cerebals was the main cause of her symptoms and of her death.

ADDENDUM

Since this paper was prepared all the surviving patients reported above have been followed and there has been no essential change in their neurologic status. None has had any further episodes suggestive of ischemia and embolism and none has had any evidence of aneurysm formation at the site of the endarterectomy.
Since that time we have operated upon an additional 13 arteries in 10 patients. Six arteries in 5 patients were totally occluded and in only 2 could a retrograde flow be obtained and circulation be restored. There were 7 arteries in 5 patients in which there was marked stenosis. Retrograde flow was obtained and circulation was re-established in all cases, making a total of 9 additional arteries in which the circulation has been re-established. Up to the present time postoperative arteriograms have been done on 5 of these arteries and the artery has been patent in each case. The remainder of the postoperative arteriograms in the surviving patients are yet to be done. One patient died of an intratracheal hemorrhage after tracheotomy necessitated by a postoperative hematoma. Another patient with bilateral occlusion died. He was in decerebrate rigidity at the time we first saw him. Of the remaining 8 patients operated upon, 4 have returned to normal, 1 has improved and in 3 there has been no change.

REFERENCES
CAROTID INSUFFICIENCY


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