Surgery in Complete Occlusion of the Internal Carotid Artery

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IT IS WELL KNOWN that vascular disease of the brain and the retina is often associated with occlusive disease of the carotid arteries. It is now possible in many cases to remove the occlusion, and such operations are being performed with increasing frequency. The technical results of operations on the common carotid, innominate, and subclavian arteries are highly satisfactory both for stenosing and complete occlusions, and at the origin of the internal carotid artery stenosing lesions are again readily operable.[1, 2] It is not yet possible to assess the functional results of these operations, as many of the patients coming to surgery have little or no neurologic disability, and the operation is performed in the hope of preventing future vascular accidents.

In patients with total occlusion in the region of the carotid bifurcation the results of surgery, both technically and functionally, are even more uncertain and for this reason we report our experience from the period of 1958 to 1960 in 15 such cases in whom surgical treatment was undertaken.

Clinical Features

There were 10 men and five women whose ages ranged from 33 to 75 years. Twelve patients presented with a hemiparesis, two with monocular visual symptoms, and one with focal seizures. The onset was associated with loss of consciousness in only two patients, and previous transient symptoms were remarked upon by seven patients.

There were no unusual features on clinical examination; the diastolic blood pressure was over 120 mm. Hg in only one patient, and one patient had mitral stenosis and atrial fibrillation. A palpable diminution or absence of the appropriate carotid pulse was noticed in eight patients. Two patients had a bruit over the occluded carotid and several others had systolic bruits over the carotid bifurcations on the other side. Ptosis and meiosis of the pupil on the side of the carotid lesion were noticed in four patients, but facial sweating was unaltered. One patient showed unilateral optic atrophy, and three patients seen during an attack of blindness showed retinal edema and arterial occlusion. In one patient friable microemboli were seen passing through the retinal vessels during an attack of monocular blindness.[3]

A hemiparesis, of variable severity, was present in 12 patients; in all, the arm and face were more severely affected than the leg. Six had a homonymous hemianopsia.

One patient had the unusual combination of internal carotid occlusion on one side with ophthalmic artery occlusion on the other, due to multiple embolism from an atrial myxoma.

Pressure Measurements of Ophthalmic Artery

In 13 patients ophthalmic artery pressures were measured with the Bailliart ophthalmodynamometer, and results are shown in figure 1. Most cases showed some diminution of pressure on the affected side, the most marked difference appearing in systolic rather than diastolic readings and in early rather than late cases.

After operation, in which pulsatile back flow was established, ophthalmic pressures returned to normal (four cases).

Angiographic Findings

Percutaneous puncture of the common carotid artery was done in thirteen patients. In the remaining two patients the diagnosis was made on the absence of the common carotid pulsation. Exposures were taken on completion of injection and 2 and 6 seconds thereafter.

The criterion of carotid occlusion was ab-

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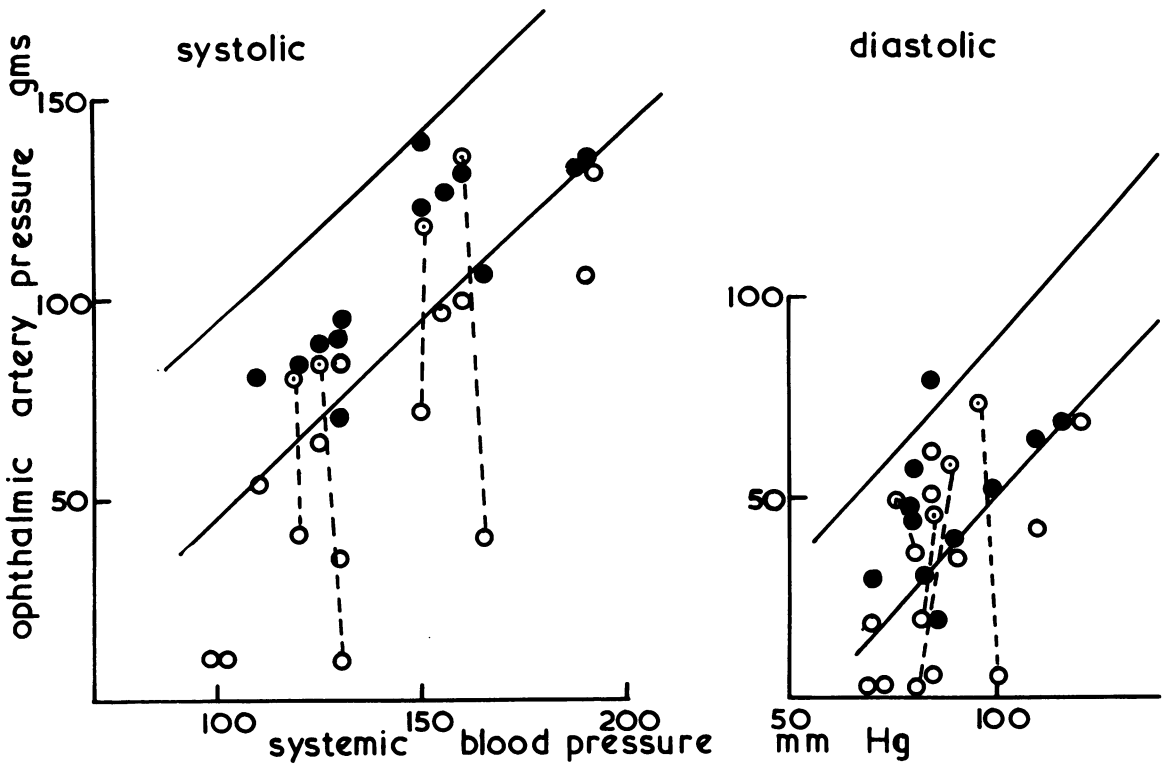

Figure 1

Ophthalmic artery pressure measurements on 13 patients. The lines indicate the range of normality (95 per cent confidence limits) for patients over 50 years. Closed circles, normal side; open circles, occluded side. Postoperative change indicated by dotted lines.

sence of filling of the intracranial artery, and there were two kinds of angiographic appearance.

Group I. Complete hold-up of the dye occurred in the internal carotid artery within 1 to 3 cm. of the origin (eight cases). Collateral vessels were usually well developed.

Group II. Apparent complete hold-up of the dye occurred in the first part of the internal carotid artery. In later films the dye was seen filling or partially filling the upper part of the artery as far as the base of the skull (five cases). Collateral vessels were not seen in this group.

This appearance indicates an occlusion of the upper part of the internal carotid that is not quite complete. When the artery is blocked at this site, retrograde thrombosis tends to occur throughout the length of the vessel to within a short distance of the origin. The final appearance is of complete occlusion at the origin of the artery, irrespective of the initial site of the thrombosis.

In two cases belonging to angiographic group II, with an intracranial occlusion and slow flow up the artery, it was possible to verify the site of the block by angiography on the operating table, dye being injected directly into the cervical part of the internal carotid artery.

Postoperative angiograms were done on only two patients as the restoration of normal carotid pressure was checked more easily by ophthalmodynamometry. There were no ill effects after arteriography.

Pressure Measurements of Carotid Artery

Pressure measurements were attempted on five occasions by a Statham strain-gage manometer, the artery being punctured with a no.-1 needle. It was not found possible to record the pressure on both sides of a complete occlusion at the origin of the internal carotid

Table 1

Results of Operation (Fifteen Carotid Arteries Explored)

A. Pulsatile backflow established	5	
Patent postoperatively (ophthalmodynamometer)	4	(Case 4 not examined)
Subsequent rethrombosis (detected by palpation)	3	Case 4. Rethrombosis at 5 days Second operation; thrombosed again; no change Case 11. Rethrombosis at 2 days; died Case 10. Rethrombosis at 4 weeks; died
No subsequent rethrombosis	2	Case 2. Slight improvement postoperatively; died 3 months later from cancer; carotid patent at autopsy Case 6. Complete recovery (9 months)
B. Backflow not established	10	
Improved on follow-up	5	Case 1. Complete recovery 3 years Case 3. Complete recovery 2 years Case 7. Improved, working 9 months Case 9. Improved, at home 6 months Case 13. Improved, no more attacks 3 months
Unchanged or worse	3	Case 5. Working 18 months Case 8. Working 1 year Case 14. In hospital 6 months
Died	2	Case 12. One week postop.; autopsy, carotid blocked at syphon Case 15. Died postoperatively; autopsy, multiple emboli

owing to thrombosis in the distal portion of the artery. In cases belonging to angiographic group II, where the occlusion occurred at the base of the skull, measurements gave confirmatory evidence of a distal occlusion, the pressure being equal in internal and common carotid arteries apart from slight damping in the distal record.

Operation

Through an incision parallel to the sternomastoid muscle, from the tip of the mastoid process to the level of the cricoid cartilage, the common carotid artery and its two branches were exposed. Approximately 7 cm. of the internal carotid artery were isolated. The lower end of the obstruction was usually at the point of bifurcation. No effort was made to do a temporary bypass of the obstruction.

Before opening the artery heparin (5,000 to 10,000 units) was given intravenously and allowed to circulate for 1½ to 2 minutes before clamping the common and external carotid arteries with Glover's multitoothed clamps. An incision was then made into the termination of the common carotid artery, along the bulb and into the internal carotid artery. If there was any back bleeding from the internal carotid artery, the artery was clamped. A formal endarterectomy was then done, and the thrombus together with the thickened atheromatous intima was easily separated and removed. Downwards into the common carotid the intima was thin and was divided. Distally, however, the thickened intima extended well up the internal carotid artery. The cut edges of the intima were sutured back to the media to prevent dissection of the coats when the flow of blood was reestablished. Before suturing the incision with 6.0 black silk a sucker and corkscrew reamer were passed up the internal carotid artery in an attempt to remove thrombus in the upper reaches of the artery. In early cases the artery was found to contain a soft, black thrombus. It has been our experience that suction alone was seldom successful in removing the clot, and we relied on a corkscrew reamer. The instrument was gently rotated into the open internal carotid artery for a short distance and then removed. A portion of thrombus was usually brought out with it, and the process was repeated rotating the reamer farther up the artery until the base of the skull was reached. By this means the last piece of a propagated thrombus was sometimes successfully removed and pulsatile back flow was es-

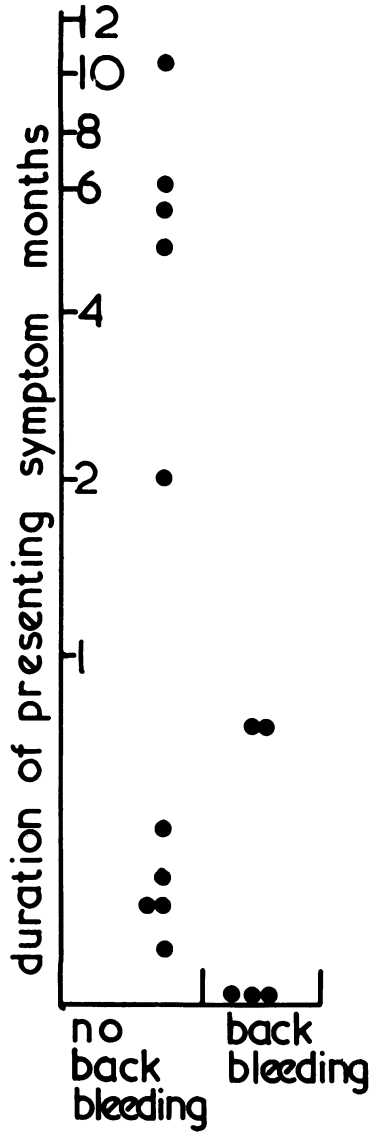

Figure 2

Duration of symptoms in technically successful cases and in those found to be inoperable.

tablished. The wound in the neck was closed in layers.

Results of Operation

A summary of the follow-up information is given in table 1 and shows that technically successful operations were achieved in five of 15 patients. Three of the carotid arteries subsequently thrombosed, although all five patients had been maintained on anticoagulants during the period in hospital. Half the patients in whom operation was unsuccessful showed some improvement postoperatively and two were completely normal 3 years later. Of the four patients who died three were comatose and gravely ill before operation, one (case 10) had only a moderate hemiparesis and was unquestionably made worse by operation. This may have been due to an embolic blockage of an intracranial vessel or to a short period of hypotension during operation.

Pathology

The pathology of the arterial occlusion was deduced from inspection of the artery at operation, from histologic examination of the endarterectomy specimen or from autopsy. In eight cases there was evidence of extensive atheroma at the origin of the internal carotid, and atheromatous plaques were removed by blunt dissection. All these cases showed recent thrombus on the surface of the plaque, and it appeared likely that the last stage of obliteration of the arterial lumen was one of thrombotic occlusion. Recent thrombus extended upward into the cervical portion of the internal carotid (five cases) and downward into the common carotid (two cases). It also extended into the external carotid artery on two occasions and probably into the ophthalmic artery on one occasion. In two cases the occlusion was embolic, one a case of mitral stenosis and the other of atrial myxoma. In both, the embolus impacted in the intracranial portion of the internal carotid and the angiogram showed apparent occlusion near the origin in early view, with delayed flow up the cervical carotid that was visible in the later films.

In one case thrombotic occlusion occurred 12 hours after an injury to the face and neck. The patient was a young man with minimal atheroma. In one case multiple arterial thrombosis including both femoral and one internal carotid occurred over the course of 6 weeks. There was no evidence of arteritis or of blood disorder, and atheroma was only moderate. In two cases belonging to angiographic group II, the internal carotid artery was narrowed and nonpulsatile in its cervical course. There was no evidence of atheroma or thrombosis at the bifurcation on opening the artery, and only slight back-bleeding occurred. Both these patients had long histories, and in one the

onset of symptoms may have been related to a gunshot wound of the neck 20 years previously. It is also possible that the internal carotid artery was congenitally hypoplastic.

Discussion

The clinical observations in the present series are in accord with other reports on larger numbers of patients.[4] It is often impossible to diagnose a carotid thrombosis on clinical grounds alone. The combination of ocular and cerebral symptoms was present on six occasions and seven patients described transient ischemic attacks in the months or years preceding the major stroke. Carotid pulsation in the neck was palpably diminished in eight patients including two with common carotid occlusion. Carotid bruits were variable; the presence of a bruit did not exclude a total occlusion. Four patients had a partial Horner's syndrome but this was not correlated with duration of obstruction and occurred in both acute and chronic cases. It was presumably due to an interruption in the blood supply to the sympathetic trunk. Angiography showed an unexpectedly large proportion of cases in which the obstruction appeared to start at the base of the skull, and the appearances in these cases of a slow laminar flow of dye up the stagnant or nearly stagnant vessels are noteworthy. These findings are not diagnostic of carotid occlusion but may occasionally be seen affecting both carotid arteries in acute hydrocephalus from tumor or subarachnoid hemorrhage.[5]

When considering the results of treatment in extracranial carotid disease it is helpful to distinguish between carotid narrowing and carotid occlusion. Narrowing is extremely common and occurs in about one third of random autopsies, frequently affecting more than one of the main vessels.[6] Most cases are symptomless but when symptoms do occur they are often transient and repetitive, both in the eye and in the cerebral hemisphere.[7] The pressure in the upper part of the carotid artery measured directly at operation or indirectly by ophthalmodynamometry is usually normal, and histologic examination of the intima removed by endarterectomy usually shows fresh thrombus on a basis of atheroma. It is probable that many of the transient symptoms suffered by these patients are due to embolism from a diseased portion of artery, a process that is known to occur in other situations, e.g., the subclavian artery in association with a cervical rib.[8] If the diseased intima can be removed by endarterectomy, the transient attacks may cease. Assessment of such cases is difficult, however, since attacks may cease spontaneously, and also because patients are often maintained on anticoagulants.[9] If embolism is frequent and if the area of stenosis cannot be removed, it may be advisable to ligate the internal carotid, provided a good cross circulation can be demonstrated angiographically.

When a partial occlusion becomes complete, the symptomatology and approach to treatment are different. Here the brain is entirely dependent on collateral vessels and the pressure in the upper part of the affected carotid artery is reduced, particularly when the occlusion occurs rapidly as when the artery is ligated.[10, 11] Under these circumstances the collateral blood flow to the hemisphere on the affected side is liable to become insufficient when the systemic blood pressure falls.[12] Many patients undergo spontaneous carotid occlusion without symptoms and the lesions may be discovered incidentally at autopsy; in others, possibly those with more advanced arterial disease, the collateral vessels are inadequate and a hemiplegia ensues.

Pulsatile backflow, which is the prerequisite of a technically successful operation, was achieved in five of 15 patients, about the same proportion of successful results as reported by others in total internal carotid occlusions.[1, 7, 13] In the remainder, either the site of the occlusion was inaccessible or the thrombus had organized and extended throughout the length of the vessel. The successful operations were all carried out within a few days of the onset of hemiparesis (fig. 2) and it is possible that had the other patients been seen earlier, most of the operations would have been successful.

Functionally, results were even less impressive. In the present series two of the tech-

nically successful operations were followed by some postoperative improvement and one returned completely to normal, but many of these with inoperably occluded arteries also showed improvement. It is not surprising that thrombendarterectomy has little or no influence on recovery from existing symptoms, since the brain quickly suffers irreversible changes as a result of ischemia, and it follows that the only justifiable reason for operating on a completely occluded internal carotid is the belief that the prognosis with regard to further strokes will be improved if four instead of three patent vessels are supplying the brain. When one artery is severely narrowed by atheroma there is a probability that the other arteries (and other parts of the same artery) will also be affected, and this may possibly be a limiting factor in the development of collateral vessels. However, the incidence of cerebral infarction following carotid ligation is no higher in arteriosclerotic patients than in young patients.[14]

On the other hand, the not infrequent recurrence of thrombosis following endarterectomy, the fact that total occlusion takes place by thrombosis, and the possibility of embolism of loose thrombus during operation or at the time when total occlusion is taking place all suggest that the medical control of thrombosis might be of more benefit than surgery. Prothrombin inhibitors of the coumarol type seem to be doubtfully effective in controlling arterial thrombosis (three arteries in the present series rethrombosed while the patient was on adequate treatment with phenindione) and a more potent antithrombotic agent possibly with fibrinolytic or antiplatelet action is urgently needed.

Summary and Conclusions

The clinical features, findings on investigation, and results of operation are reported on 15 cases of complete thrombotic occlusion of the internal carotid artery. Backflow was established in five cases but no additional clinical improvement was noted when these cases were compared to others in whom operation was unsuccessful.

We cannot recommend surgery at the present time to patients with a moderate or severe hemiplegia associated with a complete angiographic block, with the prospect of relieving existing cerebral ischemia. It seems justifiable only as a possible insurance against further strokes, and its effectiveness in this regard is unknown.

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