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Surgery of Carotid Artery Stenosis

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Hunt (1914) first drew attention to carotid artery stenosis and thrombosis as a cause of stroke. Eastcott, Pickering, and Rob (1954) demonstrated that surgical correction of stenosis was possible, with relief of symptoms in one patient. Since then series of patients have been reported by Rob and Wheeler (1957), De Bakey, Crawford, and Fields (1961), and Edwards and Gordon (1962), to mention a few authors.

Atherosclerotic narrowing at the origin of the internal carotid artery is the usual pathological abnormality, and it is remarkable how often the process is limited to this short segment of the vessel. The mechanism by which this may produce the characteristic symptoms of intermittent contralateral stroke or intermittent homolateral blindness has given rise to much speculation and research. Ross Russell (1961) demonstrated emboli in the experimental animal and also in the branches of the ophthalmic artery during attacks in patients. However, anticoagulant therapy does not necessarily relieve the patient's symptoms. Cerebral anoxia secondary to haemodynamic changes originating at the site of the

carotid stenosis has been invoked to explain the symptoms. While the numerous congenital anomalies of the circle of Willis tend to support these theories, cerebral-flow studies (Nylin, 1961) have so far been unable to confirm any overall change in blood-flow during the attacks.

Thrombosis of the internal carotid artery is often the sequel to carotid artery stenosis, and hemiplegic stroke the resultant risk, but stroke does not necessarily result from a complete occlusion. There are several well-documented case reports in which one or both carotid arteries are completely occluded, without symptoms having been experienced by the patient. There are, however, no clinical data to relate the risk of thrombosis to the degree of carotid stenosis, or to the time lapse which may intervene between these events.

The results of surgical correction of stenosis are satisfactory in the early case, and poor in the completed stroke secondary to thrombosis (Rob, 1959; Crawford, De Bakey, and Fields, 1961). The obvious explanation is that the anoxic cortical cell undergoes irreversible change after four minutes and that it is impossible to undertake surgical correction within this time limit.

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The purpose of this paper is to present the results of surgical treatment of carotid artery stenosis and thrombosis in patients coming under the care of one surgeon in 1957–64.

Clinical Features

In this survey 67 operations were performed on 57 patients. In two cases operative exploration failed to reveal an internal carotid stenosis in patients who had a suggestive history and an angiogram demonstrating irregularity of the carotid arterial lumen; therefore 65 endarterectomies were done in 55 patients. All these patients were operated on by the same surgeon (J. R. K.).

Fifty-one (93%) patients presented with intermittent cerebrovascular insufficiency. The characteristic symptoms were episodic in nature and fell into three main types: homolateral visual symptoms, speech symptoms, and contralateral hemiparesis. The attacks were intermittent and brief, sometimes lasting only a few minutes. The extent of residual signs and symptoms was variable. Often no evidence of the attack could be found. Thirty-one (57%) patients presented with more than one main symptom (Table I).

TABLE I.—Presenting Symptoms in 51 Patients With Intermittent Carotid Insufficiency

No. with visual symptoms	20
.. " speech	18
.. " contralateral hemiparesis	44
.. " more than one main symptom	31

The majority of the patients were males (50 males, 5 females). The highest age incidence was in the sixth and seventh decades (Fig. 1). A certain family history of vascular disease was obtained in 14 (26%) patients, but in many no information was available. Only one patient suffered from diabetes. Twenty-seven (50%) had a diastolic blood-pressure of 100 mm. Hg or over. Twenty (36%) patients had overt symptoms or signs of associated vascular disease. No cases of dissecting aneurysm or "pulseless disease" occurred in this series; there was no serological evidence of syphilis.

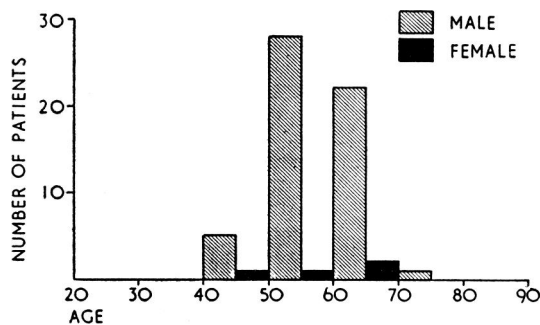


FIG. 1.—Age and sex incidence of patients with carotid artery stenosis.

Bilateral disease was present in 27 (50%) patients. This was manifest by symptoms or signs in 17 (31%) patients, but was revealed by angiography in 10 others without symptoms.

The clinical symptoms fell broadly into three main categories: (1) intermittent stroke with full recovery, (2) intermittent stroke with minor residua, and (3) completed stroke.

The majority with confirmed carotid artery stenosis had a bruit audible on auscultation. A thrill was rarely palpable over the affected artery. The neurological signs varied from normal to extensive contralateral hemiparesis and aphasia. Field defects were a frequent finding in patients with visual symptoms.

In all patients angiography was used to confirm the diagnosis. The technique varied from a unilateral percutaneous carotid angiogram in initial cases to a bilateral carotid angio-

gram (Fig. 2) or an arch aortogram later in the series. Electrocardiography and serological tests for syphilis were performed on all cases. Electroencephalography was used when necessary to exclude other diagnoses.

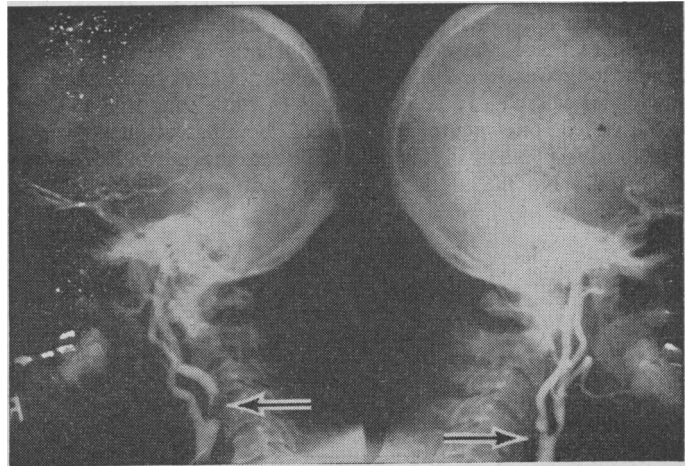


FIG. 2.—Carotid angiogram showing bilateral internal carotid artery stenosis.

Surgical Procedure

The operative procedure entails clamping of the carotid arteries on the affected side for sufficient time to permit an adequate endarterectomy. These time intervals varied between 8 and 35 minutes, with a mean of 16 minutes. To protect the brain during this period three differing techniques were employed: (1) General anaesthesia with hypothermia to 30° C. induced by surface cooling. This was used in 38 (69%) patients. (2) General anaesthesia with plastic-tube bypass from the common to the external carotid artery was used in four (7%). (3) Local anaesthesia produced by blocking cervical second, third, and fourth nerves as they emerge from the vertebral foramina was used in 13 patients (24%). During the operation an observer carefully watched the patient for signs of impending cerebral anoxia, and, if they developed, general anaesthesia was immediately induced, the patient intubated, and a bypass inserted to restore carotid blood-flow. This was necessary in only one patient in this group.

The bifurcation of the carotid artery is explored through a skin-crease incision in the neck, the atheromatous plaque is identified by gentle palpation, and the vessels are dissected to permit easy clamping. With the clamps applied heparin 50-100 mg. is injected into the common and internal carotid arteries. The plaque is then exposed by a longitudinal arteriotomy, the endarterectomy completed, and the arteriotomy closed with a continuous arterial suture. Vein or plastic patches to widen the lumen of the endarterectomized vessel were not used in any patient in this series. The wound is then closed in layers with a drain, which is removed after 24 hours.

In each patient a histologically confirmed atheromatous plaque was removed, but the characteristics of these plaques (Fig. 3) varied considerably. In 47 operations sufficient data

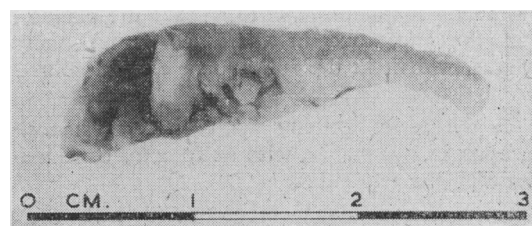


FIG. 3.—Typical atherosclerotic plaque removed at operation.

were available to classify the pathological specimens as follows :

1. The most frequent lesion (Fig. 4) was a firm atheromatous plaque producing a localized stenosis of the internal carotid artery, the effective lumen varying from 1 to 2.5 mm. in diameter (Fig. 4, type V). Calcium deposit was a common finding in these plaques. In one instance a fresh thrombus 5 mm. long was found distal to the plaque, but not occluding the distal vessel. It is interesting to note that this patient experienced one acute episode of hemiparesis six weeks previously, presumably from a similar thrombus becoming detached. No mural thrombi were noted in the remaining plaques.

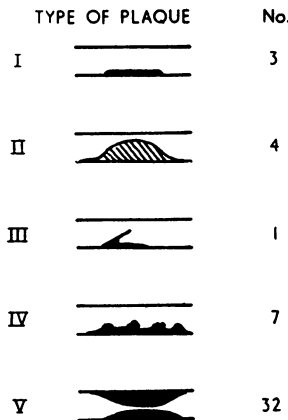


FIG. 4.—Analysis of pathological data in 47 carotid endarterectomies.

2. A roughened area of arterial intima with irregular calcified projections and small mural thrombi was the next commonest finding. This type of lesion was removed by endarterectomy in seven patients. This was the second most common finding in the partially stenosed group (Fig. 4, type IV).

3. A web-like piece of atheroma projecting into the lumen of the vessel was removed in one patient (Fig. 4, type III).

4. Haemorrhage into a plaque was observed in seven operations. This was a localized dissection, usually posterolateral, of the carotid plaque, and in no way related to the needle puncture for carotid angiography. When the plaque was mobilized the semi-fluid content of old blood or pale autolysed semi-solid content had to be carefully evacuated before the endarterectomy could be completed (Fig. 4, type II).

5. A flattened intimal plaque, causing no appreciable narrowing of the vessel and little or no radiological change, was encountered in three patients. This type of plaque, however, was associated with a clearly audible murmur over the affected side (Fig. 4, type I).

In the four patients with completed stroke and carotid artery thrombosis, operation under general anaesthesia demonstrated a tight stenosis (type V) in three and a dissection (type II) in one. Both varieties were associated with distal thrombosis extending to the cavernous portion of the vessel; although it was possible to remove the plaque in each case, and the thrombus in three cases, no neurological recovery was evident in the subsequent clinical course of the patient.

Later in the series patients with bilateral stenosis were operated upon in two stages, with an interval of four to eight weeks between the two sides.

The post-operative management varied with the anaesthetic technique used and the extent of the neurological abnormality present. Patients operated on under local anaes-

thesia were ambulant on the first post-operative day. Under optimum circumstances the patient was fit for discharge on the third day, and the sutures were removed as an out-patient.

Results

For analysis of the results in this series the patients can be divided into two main groups: (1) those with intermittent strokes, suffering from a stenosed internal carotid artery; and (2) those with completed strokes, suffering from an acute thrombosis of the internal carotid artery.

Group 1

The patients in this group (Table II) recovered from the operation with one exception: he developed ventricular fibrillation during hypothermia and required cardiac massage and electrical defibrillation. This patient died 18 hours after operation, and at necropsy was found to have had extensive coronary artery disease. The remaining 50 (98%) patients were discharged from hospital and are included in the follow-up analysis. There were 42 survivors in June 1964 (Fig. 5). Of these, 30 (71%) had no residual neurological signs before operation and have had no recurrence of symptoms. Those with moderate neurological disability have made considerable improvement but have not entirely returned to normal. Where severe neurological change was present (5 patients, 10%) little improvement followed operation. Patients with a demonstrable field defect retained this abnormality. Twenty-three (45%) of these patients have received post-operative anticoagulant therapy. In the later patients in this series this therapy was omitted.

TABLE II.—Results of Operation in 51 Patients With Intermittent Carotid Insufficiency

Survivors (June, 1964)	42
Full recovery	30 (70%)
Partial recovery	7 (17%)
Little improvement	5 (10%)
Operative deaths	1
Subsequent deaths	8
C.V.A.	4
Unrelated	2
Unknown	2

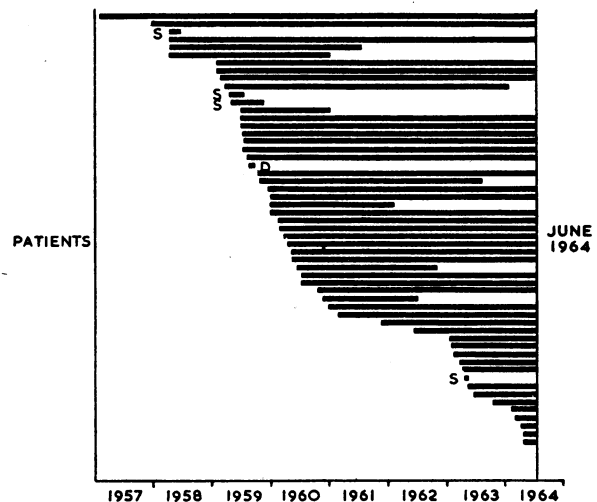


FIG. 5.—Follow-up of all patients in series: bilateral operations not indicated. S indicates completed stroke. D indicates operation death.

One patient had a recurrent stenosis at the same site 18 months after operation. He underwent a successful reconstruction of the internal carotid artery, using a homograft. This graft has recently (four years later) thrombosed and further treatment is being contemplated. In the remainder post-operative angiography was not done as a routine, and

indeed was unacceptable in the majority of patients with normal or improving neurological function. When doubt existed on the patency of the internal carotid artery, angiography was performed (Fig. 6).

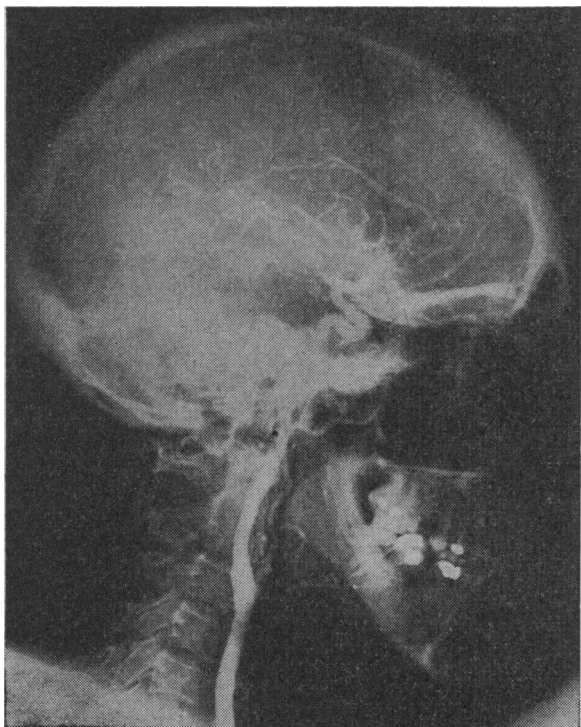


FIG. 6.—Carotid angiogram one year after endarterectomy.

Eight patients have died since operation. Four of these deaths occurred from acute cerebrovascular accidents, but in three cases this was not confirmed by necropsy. The remaining patient had an acute cerebellar haemorrhage, demonstrated at necropsy. Three of these patients had a systolic blood-pressure of over 180 mm. Hg, and all four were on anticoagulants at the time of death. The other four died of causes not related to their carotid artery disease, but one died from intestinal haemorrhage while on anticoagulant therapy.

Ten patients have been operated on for bilateral lesions. Four underwent bilateral simultaneous endarterectomy under general anaesthesia and hypothermia. Two of these developed a marked hemiparesis immediately after operation. Both made a reasonable recovery in four to six weeks. More recently the bilateral cases have been staged, using cervical block anaesthesia, with an interval of four to eight weeks between each side. Only one of the patients treated in this way experienced any complication—recovering rapidly from a transient mild hemiparesis.

Group 2

The four patients in this group had a completed stroke following an acute thrombosis of the internal carotid artery. None showed any improvement after operation, and all died in the early post-operative period. At operation the distal segment of the internal carotid artery contained propagated thrombus in each case.

Discussion

Two clear conclusions can be drawn from this series of patients. The late results of carotid endarterectomy for stenosis and recurrent stroke are good, but the results for stenosis and thrombosis with completed stroke are poor.

These concur with the experience of Rob (1959) and Crawford *et al.* (1961). The early diagnosis of carotid artery stenosis is therefore most important. The intermittent nature of the visual defects and episodes of incoordination or paralysis, particularly when associated with vascular murmurs at the root of the neck or over the carotid bifurcation, throws suspicion on the extracranial vessels, and angiography is indicated in all such patients. In the early patients carotid angiography was adopted, and arch or vertebral disease was excluded by clinical assessment, in particular by auscultation. However, the fact that 56% had bilateral disease, of which 19% were symptom-free, indicates the need to demonstrate all four cerebral vessels.

The technique of the actual carotid endarterectomy has remained standard, but the methods of protecting the brain during the enforced period of ischaemia have varied. Of the three methods, hypothermia to 30° C., while facilitating the procedure for the surgeon, carries a distinct risk of ventricular fibrillation in the ageing atherosclerotic patient. The use of a temporary plastic-tube bypass has not been entirely satisfactory, owing to the danger of kinking, thrombus formation, and interference with the endarterectomy, which procedure requires the greatest precision. Local anaesthesia on the conscious patient, although less convenient for both patient and surgeon, has proved most satisfactory, as it is possible to observe cerebral function continually and correct any signs of fatal anoxia before irreversible changes have occurred. Hyperbaric oxygen at 2 atm. has been used to protect the cerebral cortex during such surgery, but further experience will be required with this technique.

The patients with bilateral disease originally had both sides dealt with simultaneously. However, this resulted in post-operative cerebral complications in two of the four patients. Better results have been achieved when the operation was staged with an interval of four to eight weeks. From this it would appear that the cerebral circulation may require a period of time to adjust to the altered haemodynamics produced by relief of the stenosis.

Vein or plastic patches to widen the endarterectomized vessel have not proved necessary in view of the short length of the typical stenosis and the relative freedom of the distal extracranial carotid artery from atheromatous deposits. Cloth patches have the added disadvantage of providing a nidus for thrombus formation, with subsequent danger of cerebral embolism. Anticoagulant therapy has been recommended and extensively used in the treatment of cerebrovascular disease (Carter, 1961; Siekert, Millikan, and Whisnant, 1961). In our series 45% of patients were so treated in the post-operative period. Of the eight deaths occurring since operation, four were due to a cerebrovascular accident. Each of these patients was on anticoagulants, and it is interesting to note that three of them had arterial hypertension. An additional death due to intestinal haemorrhage occurred in this group.

The cause of the symptoms of intermittent stroke associated with carotid artery stenosis is of particular interest. Denny-Brown (1951) has suggested haemodynamic crises as a possible cause. Edwards, Gordon, and Rob (1960) have described two cases in which such episodes were associated with a fall in arterial pressure; the first during an attack of ventricular tachycardia, and the second from the vasodilation produced by a hot bath. No such precipitating factors were evident in the patients in the present series. Gunning, Pickering, Robb-Smith, and Ross Russell (1964), in a detailed study, related the pathological material obtained at operation or necropsy to the symptomatology in 16 patients. They have demonstrated that platelet-fibrin thrombi build up on the carotid plaque and become detached as micro-emboli which lodge in the retinal vessels and probably in the brain, to produce the typical episodes of retinal or cortical insufficiency.

While platelet-fibrin micro-emboli undoubtedly occur, it would appear from the operative material in this series that this is not the whole explanation. The patient with a type I plaque (Fig. 4) is at risk on two counts: he may develop micro-emboli, or a haemorrhage into the plaque may occlude the artery or convert the lesion into type II. Such a localized dissection occurring in atheromatous plaques has been described in detail by Winternitz, Thomas, and LeCompte (1938) and Winternitz (1954).

The type II plaque has two possible sequels: it may rupture and discharge its semi-solid content into the internal carotid artery with resultant cerebral emboli and leave the smooth web-like plaque (type III) or the rough necrotic area (type IV), or it may organize to the most commonly found type V. The risk of trial compression over the carotid artery bifurcation in patients with such a lesion is obvious, and may account for some of the neurological signs elicited during clinical examinations. The patient with a type IV plaque is at particular risk from emboli of either platelets or detached debris, and Gunning *et al.* (1964) have suggested this possibility. It is also possible that both type I and type IV plaques may build up to a type V. On numerous occasions we have noted similar lesions to those described while operating on occlusive vascular disease in the abdominal aorta and peripheral vessels of the limbs. Presumably they also produce small emboli which for the most part are lost in the vascular bed of the limbs and pass unnoticed by the patient. However, when the end-organ of the arterial supply is an essential organ, such as the brain, the need for removing a potential source of such emboli is clearly indicated.

Summary

The clinical investigation and results of 65 carotid endarterectomies in 55 patients presenting during 1957–64 are analysed. Fifty-one patients had intermittent attacks of cerebral insufficiency produced by carotid artery stenosis, and four had completed stroke secondary to carotid artery thrombosis. In the former group 42 patients were alive and well in June 1964, 30 of these being completely free from

symptoms; there was one operative death. All four patients in the latter group died within one month of operation.

Ten patients with bilateral disease were operated upon; staging of the operation at an interval of four to eight weeks is recommended in such cases.

Hypothermia was used in 38 patients, plastic-tube bypass in 4, and local anaesthesia in 13. The relative merits and demerits are discussed.

An analysis of the atherosclerotic plaques encountered in 47 operations permitted classification into five different types. The possible mechanisms of formation and the relationships to symptomatology are discussed.

It is a pleasure to acknowledge the contribution of the physicians and neurologists who referred the patients, and particularly of Dr. C. H. Edwards, of St. Mary's Hospital; also the help of the many general practitioners who assisted in the follow-up of the patients. We are also indebted to Dr. David Sutton for the arteriographic studies, to Dr. P. N. Cardew for photographic reproductions, and to Professor W. T. Irvine for access to the records of the earlier patients in this series.

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Effects of Tolbutamide Upon Gastric Secretion and Emptying

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Brit. med. J., 1965, **1**, 1464–1466

It was suggested that endogenously produced glucagon and insulin might play a physiological part in controlling gastric secretion and emptying (Aylett, 1962a).

This communication deals with the response of gastric secretion and emptying to stimulation by intravenous tolbutamide in duodenal-ulcer subjects. The method of measuring gastric secretion and emptying was adapted from that of Hunt (1954), as described by Aylett (1962b).

Method

Quantitative water test meals were given, marked with a known amount of phenol red. The patients fasted from 10 o'clock the previous night; starting at 9 a.m. the 750-ml. test meal was poured into the stomach via a polyvinyl tube passed

through the mouth. This meal was allowed to "digest" for 20 minutes, then the unemptied gastric contents were recovered by suction, using an electric pump. Each "meal" was preceded by a washout of 250 ml. of tap-water. A similar washout was given after the meal to check the completeness of recovery.

Estimations were made, on recovered gastric contents, of the concentrations of acid, chloride, potassium, sodium, and phenol red, and of the phenol-red content of the final washout. With the use of Hunt's equations, these measurements, together with the volume of recovered gastric contents and of the final washout, enabled the 20-minute output of these gastric electrolytes to be calculated, together with the volume of gastric contents emptied.

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