Surgical aspects of carotid-vertebral insufficiency

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THE ASSOCIATION between obstructive lesions of the extra-cranial portions of the carotid and vertebral arteries and symptoms of cerebro-vascular insufficiency has long been recognized (Todd, 1844), and with the development of reconstructive arterial surgery during the past 20 years it is hardly surprising that a great deal more attention has been focused on the condition. However, in spite of the ease of demonstration of the lesions by arteriography, and the technical competence of vascular surgeons in restoring arterial patency, the role of surgery in the management of occlusive extra-cranial arterial disease is not yet clearly defined: indeed, it is the subject of much controversy. This state of affairs results from a number of factors—a lack of basic knowledge of the natural history of the untreated disease; the uncertainty of the relationship between a demonstrated carotid or vertebral artery obstruction and ischaemic cerebral symptoms; and, not least, the lack of published results of large, properly controlled clinical trials of surgical versus conservative management 15 years after Eastcott, Pickering & Rob (1954) reported their first experience of carotid endarterectomy.

The present paper is concerned chiefly with a consideration of the role of surgery in management, but it is necessary also to review briefly current thinking on the pathology and investigation of extra-cranial cerebro-vascular disease, and to be clear as to what one hopes to achieve by restoration of full arterial patency.

Pathology

Atheroma is, of course, by far the most important cause of occlusive disease of the extra-cranial vessels. It most often takes the form of a fibrous stenosing lesion with superficial thrombosis (Fig. 1), the latter tending to lead to complete occlusion, perhaps preceded by embolization. Ulcerating lesions of the atheromatous plaque also occur frequently, and are a likely source of emboli to the intracranial arteries (Fig. 2). There is, however, a wide range of other pathological conditions which may, in varying degrees of rarity, cause extra-cranial occlusions and

FIG. 1. An extreme, smooth stenosis of the internal carotid artery.

they must be kept in mind—emboli from the heart, spontaneous dissecting aneurysms, giant cell arteritis, Takayasu's disease, scleroderma or even trauma (Little *et al.*, 1969). As an extreme rarity the vertebral or even carotid artery may be occluded by an embolus resulting from retrograde thrombosis from a vegetation in the post-stenotic dilatation of the subclavian artery distal to a cervical rib compression (Shucksmith, 1963). More common, though its role in pathogenesis may be difficult to assess in the individual patient, is tortuosity of the internal carotid vessels, often with extreme angulation or kinking in certain positions of the neck.

The demonstration of an extra-cranial arterial

FIG. 2. An irregular, ulcerating lesion of the internal carotid artery. This is unlikely to reduce blood flow, but the patient's multiple transient cerebral ischaemic episodes may well be due to liberation of micro-emboli.

obstruction in a patient with cerebral ischaemia does not necessarily mean that this is the causal lesion: indeed, as Crompton (1969) has put it, there is an embarrassing failure of correlation. For example, there have been many post-mortem studies in which an observed high incidence of arterial obstruction was unremarked by cerebral symptoms in life (Samuel, 1956; Martin, Whisnant & Sayre, 1960). The overall autopsy incidence of atheromatous disease of the extra-cranial vessels in unselected series of middle aged and elderly subjects has been variously stated to be between 40% (Martin *et al.*, 1960) and 8% (Kameyama & Okinaka, 1963), the figure obviously depending in part on whether stenoses of the cervical vessels are counted, or only complete occlusions. It follows, therefore, that not every obstructive lesion is clinically significant, and the problem is to recognize those which are. The situation is further complicated by the frequent association of intra-cranial obstructions with extra-cranial. The former often are of the greater impor-

tance in the production of symptoms, yet they may be unrecognized unless the intra-cranial vessels are routinely visualized arteriographically in the investigation of the patient.

With regard to the situation of carotid and vertebral artery lesions, it is fortunate, from the viewpoint of surgical access, that they are at the origins of these vessels in the vast majority of cases. Indeed, the internal carotid artery is rarely affected by atheroma between the sinus and the siphon, though lesions along the course of the vertebral arteries are more frequent, especially in the left (Schwartz & Mitchell, 1961). The relative incidence of carotid and vertebral lesions in post-mortem and arteriographic studies appears to be about equal (Stein *et al.*, 1962; Faris & Poser, 1963; Svare, Taveras & Stein, 1964). The occurrence of significant atheromatous lesions at other sites in the extra-cranial arteries is much less common, accounting for perhaps only 5% of extra-cranial atheromatous lesions (Svare *et al.*, 1964). Acute thrombotic and ulcerating atheroma, as opposed to slowly forming fibrotic stenosis, is much more commonly seen at the origin of the internal carotid arteries than elsewhere, and is rare in the vertebral arteries. Its other site of occurrence is in the proximal subclavian arteries, where it may lead to the rather intriguing steal syndrome (Contorni, 1960).

It is therefore important when considering investigation and treatment of extra-cranial cerebro-vascular disease to practise four vessel angiography as a routine, whenever possible, otherwise multiple and perhaps significant lesions are liable to be missed in a considerable proportion of cases. Similarly, as has already been pointed out, intra-cranial angiograms should also be obtained routinely: for example, Hutchinson & Yates (1957) found that 50% of a series of patients coming to autopsy as a result of a cerebral infarct had both intra- and extra-cranial disease associated. Other investigations have suggested that a combination of intra- and extra-cranial arterial occlusion is the important factor in the production of many cerebral infarcts (Kameyama & Okinaka, 1963; Weiner, Berry & Kundin, 1964). It may well be, of course, that a proportion of these intra-cranial lesions are the result of emboli from a proximal internal carotid lesion, rather than being primary atheroma.

Only by having full information about the state of the intra-cranial vessels can the possible pathological significance of any accessible extra-cranial lesion be judged, and surgery be applied more rationally.

The natural history of atheromatous extra-cranial cerebro-vascular disease

The difficulties in obtaining clear-cut useful information about the natural history of occlusive

extra-cranial cerebro-vascular disease are considerable, stemming first from the impossibility in a patient with cerebral infarction of being certain on clinical grounds of the primary site of the arterial occlusion (Lascelles & Burrows, 1965). Similarly, clinical differentiation between embolic and occlusive lesions as the cause of an infarct is usually equally uncertain. Even more fundamental is possible uncertainty in distinguishing between cerebral haemorrhage and infarction. Complete angiography would facilitate a proper study of natural history, but its hazards, particularly in the elderly, preclude its use as a routine investigation, so that this useful tool is only applied in selected cases.

In general, therefore, it is not possible in the current state of knowledge to predict the effects of untreated extra-cranial cerebro-vascular disease, and this greatly increases the difficulty of defining the place of surgical treatment. Rather better information is available about one more readily defined form of cerebral ischaemia, namely the transient variety, in which the episodes of neurological deficit last no longer than an hour or two. When these attacks are associated with demonstrable stenotic disease in the extra-cranial vertebral or carotid vessels they are regarded by vascular surgeons as the best indication for arterial reconstruction: it is therefore important to consider what is known of prognosis in the untreated patient.

Transient cerebral ischaemia appears to have its effects about equally often in the vertebral and carotid territories, as judged by clinical investigation. Of eighty-two such patients followed by Acheson & Hutchinson (1964), half developed a stroke during the period of follow-up of 3 years, the average interval between the onset of ischaemic attacks and the stroke being 13 months. Strokes appeared rather more often in their series in patients with vertebral ischaemia than carotid, and resulted in total disability or death in about a third of the patients. Marshall (1964) showed that stroke occurred earlier in carotid ischaemia than in vertebro-basilar, at 14 and 23 months respectively, in the untreated patients in his large series. He emphasizes (1969) that as the risk of a completed stroke is high in transient ischaemic attacks associated with a carotid stenosis treatment is urgently required. This urgency is not so marked in vertebral lesions. The variability with which progression to stroke occurs in patients with transient cerebral ischaemia is well known, and attacks may cease spontaneously at any time. Rather curiously, the risk of stroke does not seem to be related to the frequency of the attacks (Bradshaw & Casey, 1957). Parry (1969) suggests that once a patient has had a stroke he has a 20% chance of having another within a year and a 10-40% chance of being dead within 5 years.

The special investigation of patients with suspected extra-cranial carotid or vertebral artery disease *Angiography*

Angiography is the mainstay of investigation. There is still some controversy as to whether direct puncture of the neck vessels is desirable, or whether aortic injection through a catheter passed via a peripheral artery is safer and better. The modern trend is towards the latter technique. Wood & Hilal (1969) have laid down four basic guide lines for the choice of arteriographic technique, and these have much to commend them: first, all four vessels should be visualized throughout their cervical passage from their origins; second, multiple radiographic projections may be needed to give complete information about the significance of plaques involving only part of the vessel circumference, and the significance of kinks in tortuous vessels; third, the whole intra-cranial circulation must also be visualized to exclude other cerebral pathology and to show the extent of intra-cranial atheroma; finally, the arteriographic technique used must have a minimal morbidity.

Apart from showing the existence of disease in the intra- and extra-cranial arteries, arteriography will also demonstrate the site and extent of collateral arterial pathways, and a knowledge of these may be of importance in planning surgical treatment.

Dangers of arteriography

Many workers have pointed out the higher incidence of neurological complications of angiography in patients with ischaemic arterial disease, compared with that in patients being investigated for cerebral tumours and other non-ischaemic conditions. However, De Bakey and his colleagues (Crawford, De Bakey & Weibel, 1969) report a mortality of less than 1% from angiography in an enormous number of investigations in occlusive extra-cranial cerebro-vascular disease. The danger of arteriography is perhaps greatest when direct vessel puncture is employed, especially by less experienced radiologists, the chief hazard lying in the intra-mural injection of the contrast medium. This may result in acute stenosis or obstruction of the artery, and to a less extent in the detachment of atheromatous emboli at the site of puncture. For example, it is a common surgical experience to find a complete internal carotid occlusion at operation, due to recent red thrombus superimposed on an atheromatous plaque, in a patient in whom arteriography only a few days before had shown a tight stenosis. This situation may sometimes have been precipitated by manual compression of the vessel to stop bleeding after removal of the arteriography needle.

It is to avoid these complications of direct needle puncture that non-selective aortic arch injection is

so often advocated now, a short catheter being passed up the left brachial artery, preferably, to just beyond the aortic valve. A bolus of contrast material is very rapidly injected, and, ideally, multiple exposures are made with a biplane apparatus. The injection may be repeated with the patient changed in posture if this appears to be indicated by the first series of films. This technique gives X-rays of good diagnostic value for both the extra- and intra-cranial arteries, and also shows well the origins of the common carotid, vertebral, subclavian and innominate arteries.

Intravenous arteriography by the simultaneous injection of large volumes of contrast medium into a superficial vein in each arm has been advocated as a much simpler technique, avoiding any possible arterial trauma, but the resulting arteriograms are frequently of too poor quality for diagnostic usefulness, and the method has not been widely adopted.

Other methods of studying the effects of ischaemic disease on the cerebral blood

While arteriography is essential in the proper investigation of patients other simpler techniques have been employed, particularly as screening tools, and in the follow up of patients after surgery. None is wholly reliable, but three are still in the experimental stage: thermography, isotope techniques, and Doppler ultrasonography. Thermography (Wood, 1964, 1965; Wood & Hill, 1966; Mawdsley *et al.*, 1968) is particularly applicable in internal carotid artery disease as the frontal and supra-orbital branches of the ophthalmic artery supply the skin on the forehead. The temperature of this area and of the inner canthus of the eye is reduced in over 80% of patients in the presence of a carotid obstruction or significant stenosis. The Doppler ultrasonographic technique (Brisman *et al.* 1970) is also applicable to cervical carotid disease, indicating when carotid blood flow is reduced. O'Brien *et al.* (1967) and others have applied isotope scanning methods to the intact skull, with the object of detecting diminished hemispheric blood flow in the presence of ischaemia due to extra-cranial disease, but the correlation with arteriographic findings has been poor.

It seems unlikely that any of these indirect methods currently available can be developed to give constantly reliable information about blood flow, and arteriography remains the essential investigation in all patients in whom surgery is contemplated. It must routinely demonstrate the entire extra- and intra-cranial course of all the major vessels supplying the brain.

The effects of cerebral ischaemia due to extra-cranial carotid and vertebral artery disease

A wide variety of clinical presentations of cerebral

ischaemia occur, ranging from the major dramatic stroke to the 'little stroke' or transient ischaemic attack, the latter tending to recur with varying frequency. Clinical differentiation between carotid and vertebro-basilar ischaemia may be difficult, symptoms depending so much on the ability of the collateral circulation to compensate for diminished blood flow in one major vessel, but certain observations may be helpful in pointing to the artery involved (Williams, 1969).

In carotid disease anterior cerebral symptoms are rare, except in bilateral occlusion, because of the normally good cross-flow via the anterior communicating artery. It is the middle cerebral artery territory which is so often affected, resulting in the well recognized syndromes of speech disturbances, hemiplegia, in which the legs may be spared due to adequate collateral blood flow, and hemianopic field defects. Vertebro-basilar ischaemia also produces some well recognized symptoms (Williams & Wilson, 1962), such as vertigo, visual disturbances, which are often transient, drop attacks, and numbness and clumsiness of the arm. Epileptic fits may occur, and occipital headaches are not uncommon. Vertebro-basilar symptoms tend to be recurrent, and are of wide variety.

However, the symptoms of carotid and vertebro-basilar ischaemia are variable, and may co-exist together, so that when arteriography has been decided upon it is essential, as has already been stressed, to visualize all four vessels in both their extra-cranial and intra-cranial course.

Selection of patients for angiography

It is clear that only a small proportion of patients with symptoms suggestive of carotid or vertebral artery lesions will be candidates for surgery, but before surgery itself can be considered those patients who will be submitted to arteriography must be selected. This is a task of paramount importance, yet inevitably controversial and difficult in the present state of knowledge. The selection process will be initiated by the physician or neurologist who habitually sees the whole range of patients with cerebro-vascular ischaemia and other cerebral lesions. He has three diagnostic steps to take before deciding that a patient should be submitted to arteriography (Williams, 1969).

First, he must decide that the symptoms are most likely to be due to cerebral ischaemia: second, he must decide that the cause of the ischaemia is most likely to be mainly in the extra-cranial arteries, and that there is no evidence of gross intra-cranial artery disease: third, having made the two previous decisions he must finally decide whether other general aspects of the patient's health and age will permit surgery, should this be proved feasible.

There is no point in performing arteriography unless it is to be followed by operation, if indicated, unless some other cause for the symptoms, e.g. tumour, is suspected.

The preliminary selection is thus completed before any patient reaches the surgeon, so that an awareness of the possible role which surgery may play in treatment is necessary in physicians routinely seeing patients with cerebral ischaemia. The final decision whether or not to operate now lies with the surgeon, and he will be influenced in large part by the nature of the lesions shown by arteriography. Two courses are open to him: he may attempt to reconstruct almost every lesion in which this is possible, or he may modify his selection by reference to the neurological state of the patient, by his assessment of the possible benefits of restoration of normal arterial patency, and in consideration of the possible risks of surgery in the individual. It is clear that much controversy is again possible, so that the rationale and risks of surgery must be considered in some detail.

Rationale of surgical treatment of occlusive carotid and vertebral artery disease

If the results of large controlled clinical trials of surgical versus non-surgical treatment were available much of the controversy over the role of surgery would disappear. In the absence of this statistical material the place of surgery must be justified by arguments setting out the theoretical advantages of restoring blood flow to normal, and converting an irregular, atheromatous arterial segment to a regular, smoothly lined one of normal or larger calibre. It is necessary to consider separately internal carotid stenoses, carotid occlusions and vertebral artery lesions.

Internal carotid artery stenosis

A carotid stenosis may produce cerebral ischaemic symptoms either because it reduces blood flow distally to a critical extent which cannot be compensated for by the collateral arrangements, or because the irregular surface of the lesion leads to liberation of emboli, often small and multiple.

It originally seemed that the former, a diminished blood flow beyond the stenosis, provided a simple explanation for a major stroke or transient cerebral ischaemic attack. Poiseuille's law is often quoted in support of this, it being suggested that a progressive reduction in blood flow occurs as the stenosis increases. However, this law applies to flow in tubes with a free outflow: in arteries there is a peripheral resistance which is normally so large that the additional resistance imposed by a proximal stenosis is insignificant till this has reached a critical point, only after which is distal blood flow markedly

reduced. Brice, Dowsett & Lowe (1964) measured this critical stenosis in man by applying a micrometer screw clamp to healthy carotid arteries which were being tied for the control of intra-cranial berry aneurysms. The degree of stenosis was thus calculable, and blood flow proximal and distal to it was measured with an electro-magnetic flow-meter. Their conclusion was that blood flow was only lessened when the lumen area was reduced to between 2 and 5 mm², corresponding to about an 85% stenosis in an average-sized artery. In the long irregular stenoses seen frequently in atheromatous lesions, however, it may be that rather lesser degrees of stenosis reduce blood flow. For example, if blood pressures are measured above and below a stenosis by intra-arterial needle at the time of operation a systolic gradient is frequently found when the stenosis appears on the arteriogram to be of about 50%. Such a pressure gradient, under conditions of stable peripheral resistance, is a good indication of a reduced blood flow (Figs. 3 and 4), and after successful reconstruction of the stenosis it should disappear.

FIG. 3. An advanced, irregular, high grade stenosis probably leading to marked reduction of blood flow (see Fig. 4).

FIG. 4. The operative pressure recordings made on the vessels illustrated in Fig. 3. Pressures below (top) and above (bottom) block, before disobliteration. There is a marked systolic and diastolic pressure gradient suggesting a large reduction in blood flow beyond the stenosis.

It is thus likely that a stenosis has to be of severe degree before ischaemic symptoms can be attributed to it, and when this degree of stenosis is reached it is difficult to see how it may be associated with multiple transient ischaemic attacks rather than a frank infarction. Denny-Brown (1960) and others, however, have suggested that if hypotensive episodes occur in a patient with a carotid or vertebral stenosis these may lead to a temporary reduction in blood flow beyond the stenosis, and hence to a transient cerebral ischaemic episode. Millikan (1965) considered the role of transient hypotensive episodes in the production of 'little strokes', and suggested that there may often be associated intra-cranial occlusions with a collateral circulation normally of only a marginally successful quality. Vascular spasm is no longer invoked as a cause.

Much attention has centred on the causation of strokes by emboli liberated from the cervical atheromatous lesion. While a large embolus may occlude the middle cerebral artery to cause death or major disability, more interesting are the showers of micro-emboli (Gunning *et al.*, 1964) which are now widely held to be a cause of transient cerebral ischaemic attacks. There is no doubt that surgical restoration of a smooth lumen can prevent further embolization, and this has been invoked as a major justification for operation. However, note must be taken of the very variable natural history of transient cerebral ischaemia, with liability to spontaneous remission, and the good results claimed for anticoagulant therapy. Marshall (1969), however, only advocates the use of anticoagulants in this condition in three circumstances: where the atheromatous lesion is beyond the reach of surgery, in particular in the carotid siphon; to tide a patient over till surgery can be arranged; and, rarely, in patients who for general reasons are not regarded as suitable subjects for surgery.

It would seem that where transient ischaemic attacks are associated with a carotid stenosis a good theoretical case for vascular reconstruction does

exist. If the attacks are due to micro-embolization they will stop, and in a severe grade of stenosis restoration of normal distal blood flow will be achieved.

Progression of untreated carotid stenoses to complete occlusions. It is well known from serial arteriograms over a period, particularly in the limb vessels, that stenoses tend to progress to complete occlusions, and when this occurs in the internal carotid artery a major stroke may be the sequel. For example, a previously observed cervical bruit, due to a carotid stenosis, may be noted to have disappeared when the patient presents with a major stroke, the stenosis having become an occlusion, with a consequent severe and uncompensated reduction of cerebral blood flow. Similarly, post-mortem examination in patients dying of stroke frequently reveals recent occluding red thrombus superimposed on a long-standing stenosing atheromatous lesion. Finally, the occlusion by clamping of a stenotic internal carotid artery at operation under local anaesthesia may sometimes result in the patient's loss of consciousness, with recovery if the clamp is quickly removed, thus suggesting the effect of natural progression of the stenosis to occlusion if sufficient collateral pathways did not appear.

A reasonable case, from the prophylactic viewpoint, may thus be made out for surgical treatment of carotid stenoses to prevent them becoming complete occlusions. Further, should occlusive disease develop in the other cervical arteries then a better collateral potential has been provided. Four arteries must always be better than three, and so on.

Hazards of endarterectomy for carotid stenosis. However desirable reconstruction of a carotid stenosis might appear from the above, it is also necessary, before advocating it, to be aware of the possible ill-effects of the operation. The actual mortality rate of carotid reconstruction varies widely in different reported series, ranging, for example, from an 8–9% hospital mortality in a collected series of carotid reconstructions of all types in 1329 patients reported by Rainer *et al.* (1966), to one death in fifty-one operations for carotid stenosis reported by Kenyon & Thompson (1965). Certainly, the sort of mortality generally quoted today in operations for stenosis is about 2 or 3% (Bland *et al.* 1970).

However, a consideration of the risk of serious neurological damage, short of death, occurring at operation is hardly less important, and here reported figures vary much more widely than those quoted for mortality: consideration of the literature and personal experience would suggest that about 5% of patients suffer some degree of neurological

deterioration as a result of operation, though recovery occurs in a significant number of these.

Another problem which arises when claiming that operation has produced neurological improvement, or a cessation of transient ischaemic attacks, is the difficulty of proving that the reconstruction has remained patent. For example, transient ischaemic episodes due to liberation of emboli will certainly be stopped if total occlusion of the internal carotid artery results, and if the collateral circulation is adequate no neurological sequelae will arise. Apart from arteriography there is no certain method of proving that the internal carotid artery remains patent, and the surgeon is rarely any more anxious to suggest, than an apparently well recovered patient is to accept, further arteriography with its discomfort and slight hazard.

Blaisdell, Lim & Hall (1967), however, have made what is perhaps the most complete arteriographic follow-up of extra-cranial arterial reconstructions to date. Out of 100 patients ninety-nine were proved to have patent reconstructions at the close of operation. Ninety-five of these patients had repeat arteriography between 10 and 60 days later and in ninety-four the reconstruction remained patent. Further arteriography in fifteen patients available for study 5 years after operation showed none to have re-occluded, but two had become stenosed.

There is thus evidence that if the carotid reconstruction is carried out with meticulous care, and proved to be patent at the termination of operation later re-occlusion is rare.

An occasionally observed sequel to carotid reconstruction is a spell of extreme hypertension, and hypotensive measures may need to be instituted to reduce the risk of a cerebral haemorrhage. The cause of the hypertension is not certainly known, but may be ascribed either to cerebral ischaemia during operation or to carotid sinus stimulation.

Internal carotid artery occlusion

When complete occlusion of the internal carotid artery occurs it is liable to be associated with a severe degree of brain damage, unless the collateral circulation is abundant. Once cerebral infarction has occurred there can be little hope of reversing the results by reconstructive carotid artery surgery. If infarction has not occurred then the natural collateral circulation is adequate. In these circumstances, therefore, the justification for attempting surgery would be the resulting improvement in the collateral potential as a prophylaxis against the effects of occlusions developing later elsewhere. If a stroke is in a state of evolution, however, restoration of blood flow may reduce the final extent of the neurological deficit. Hard evidence that this occurs is difficult to find.

Even if a case can be made out for attempting to reconstruct complete internal carotid artery occlusions, restoration of flow, in practice, is frequently impossible. This is because occlusion of the internal carotid at its origin is so frequently followed by superimposition of thrombus up the length of the unbranched vessel into the skull, and this thrombus rapidly becomes adherent to the vessel wall and cannot be removed from below: if no bleed-back can be obtained from the cranial end of the artery restoration of onward flow is not possible and nothing useful can be achieved by operation. The time taken for this irrevocable situation to arise is very variable, restoration of flow being impossible to achieve within 6 hr of a stroke in one patient while it may be restored in another months afterwards. An explanation in the latter case may sometimes lie in the existence of an aberrant ascending pharyngeal artery which arises above the occlusion and permits some collateral flow through the distal internal carotid and thus prevents thrombosis: in other cases an apparently complete occlusion on the arteriogram does have a small patent channel through its centre permitting some flow to occur. In a collected series of 151 complete occlusions explored by various surgeons, flow could only be restored in forty-three patients, and the number in whom the neurological state was thought to be improved was ten (Gillespie, 1967). Of particular interest is the fact that several authors reported that as much clinical improvement took place in those patients in whom flow could not be restored as in those in whom it was, though it may be that the long-term prognosis is worse in the former group.

There is thus little hope of achieving useful immediate results by operating on patients with complete carotid occlusions and strokes, particularly if several days have elapsed (Thompson, Austin & Patman, 1967), so that the possible prophylactic value of a successful reconstruction must remain speculative (Perdue *et al.*, 1970).

Hazards of attempted reconstruction of complete internal carotid occlusions. When complete occlusion of the internal carotid artery occurs the external carotid usually becomes a most important collateral pathway for cerebral blood flow, especially via its anastomotic connections with the branches of the ophthalmic artery round the orbit. It is therefore essential that flow in the external carotid is not impaired by operation on the internal carotid, or a worsening of the patient's neurological state is likely. Indeed, when flow in the internal carotid cannot be achieved, a formal reconstruction of a stenosed external carotid artery (Fig. 5) may be worth while to facilitate its usefulness as a collateral (Deithrich *et al.*, 1968).

FIG. 5. There is a complete occlusion of the internal carotid artery and marked stenotic disease of the external. Reconstruction of the latter might be worth while if flow cannot be re-established in the internal carotid artery.

A further, more recently recognized, hazard of operating on complete internal carotid occlusions associated with a recent stroke is the risk of haemorrhage into the cerebral infarct, with serious or fatal results (Bruetman *et al.*, 1963; Wylie, Hein & Adams, 1964). The extent of this risk cannot certainly be expressed, and depends greatly on the neurological state of the patient at the time of operation, and also on the interval of time elapsing between the stroke and the surgery. The risk seems to be greatest in patients operated on very soon after a major stroke, and in whom the neurological state is in rapid evolution, either improving or worsening. It is now widely accepted that if operation is to be attempted in such patients it should be delayed for at least a month: by this time, of course, the chance of restoring flow will have been much reduced.

The acutely kinked internal carotid artery as a cause of cerebral ischaemia. Acutely angulated loops of a redundant tortuous internal carotid artery may be termed 'kinks' (Figs. 6 and 7), and such kinks may

FIG. 6. An acutely angulated or 'kinked' internal carotid artery.

produce functional stenoses (Najafi *et al.*, 1964). Occasionally atheroma may form in a kink leading to structural stenosis, or even to liberation of micro-emboli. However, it must be stressed that all other possible causes of cerebral ischaemia should be excluded before considering operating on a kink. When a kink does appear to be associated with symptoms, these are usually of the transient ischaemic type, and they may be produced by certain neck movements. It is only when the symptoms are of the transient ischaemic type that operation is worth carrying out, and the experience is that the ischaemic episodes are stopped.

Embolic occlusion of the internal carotid artery

The occlusion by an embolus, usually of cardiac origin, of the internal carotid artery is an uncommon cause of a stroke. As no time elapses to permit the development of a collateral circulation the stroke is liable to be profound. The chances of improving the situation by embolectomy are obviously small, but if this is done at all it must be done quickly, and the risk of producing haemorrhage into an infarct, if flow is re-established, must be accepted. The writer,

Fig. 7. The operative appearance of a 'kinked' internal carotid artery in a patient who suffered from multiple transient cerebral ischaemic attacks. These ceased after reconstruction of the vessel.

for example, has operated on five patients under the age of 35 years with embolic carotid occlusion and profound stroke of less than 8 hr duration. Flow could be restored only in three of the five, and in only one of these three was there a dramatic complete recovery within a couple of hours of operation.

It would therefore appear that carotid embolectomy for stroke is not likely to be a very fruitful field for surgery.

Final selection of patients with carotid artery lesions for surgery. As has been discussed, arterial reconstruction may have either a curative role in stopping transient cerebral ischaemic attacks and improving the neurological state of patients with stroke in evolution, or have a prophylactic role in preventing carotid stenoses progressing to occlusions, and improving total cerebral blood flow in extreme stenoses and occlusions to reduce the effects of other subsequently developing occlusive lesions, either intra- or extra-cranial. It is therefore possible to justify operating on almost every patient with an anatomically correctable lesion. Results of surgery, however, suggest that certain groups of patients are most likely to benefit, and that some lesions are rarely worth reconstruction.

Surgery probably has its most curative role in the treatment of multiple transient cerebral ischaemic attacks associated with a stenotic internal carotid artery lesion. Whatever their mode of production the attacks usually cease after operation, and would seem that the benefit of surgery far outweighs the risks. Much less consistently, restoration of blood flow in a patient with a stroke in evolution may be associated with marked improvement in the neurological picture, and it is in this group of patients that reconstruction is most controversial. A patient with a completed stroke, however, cannot be cured or improved neurologically by carotid reconstruction and should probably not be subjected to operation if the stroke has left a severe neurological deficit. Similarly, in complete carotid occlusion operation is seldom worthwhile as restoration of flow is not possible in a high proportion of patients.

The value of the other or prophylactic role of surgery in restoring normal blood flow in tightly stenosed or occluded carotid arteries is more speculative. However, as atheroma tends to affect more than one of the extra-cranial arteries there must be a potential benefit in having as many primary arterial pathways open to maintain collateral circulation as possible, and this is particularly so if intra-cranial occlusions or congenital anomalies impair the efficiency of the circle of Willis (Kamuyama & Okinaka, 1963). Patients who have not recovered well neurologically, but have one or more tightly stenosed vessels, can reasonably be submitted to surgery. The risk of major cerebral infarction from their carotid stenosis, particularly if bilateral, progresses to complete occlusion, far outweighs any hazard of operation. Similarly, patients who have recovered from an infarct and have one occluded internal carotid artery with stenosis of the other are in real danger of a future major stroke. In them it is perhaps worth trying to restore flow in the occluded vessel first, to increase collateral brain blood-flow, and, if this is not possible, to ensure optimum patency in that external carotid artery. The stenotic internal carotid vessel should then be dealt with, a shunt usually being essential to maintain cerebral blood flow during the reconstruction. There would seem to be little doubt about the future value of successful reconstruction in such a patient (Fig. 8a and 8b).

Vertebral artery lesions

The commonest, and certainly the most accessible site of occlusive disease in the vertebral artery is just at its origin from the subclavian artery. The lesion may be a stenosis (Fig. 9) or a complete occlusion, and in the latter case propagated thrombus may extend far up the vessel. Vertebral artery lesions situated elsewhere in the vessel are rarely amenable

FIG. 8. This 36-year-old patient was recovering from a left hemiplegia. The completely occluded right internal carotid artery (Fig. 8a) was explored first, but blood flow could not be re-established. As the left internal carotid artery showed a significant stenosis (Fig. 8b) it was deemed essential to reconstruct this vessel as a prophylactic procedure a month later.

to surgical access and reconstruction so will not be considered further.

An important feature of the vertebro-basilar system is that the basilar artery may be adequately perfused by either one of the vertebral arteries alone, if patent, so that vertebral artery lesions tend only to produce symptoms when they are bilateral. The exceptions to this are where one vertebral artery is dominant, the other being very small, or where the vertebral arteries do not both join the basilar artery. It should also be stressed that when significant vertebral artery lesions are present there are lesions in the other extra-cranial arteries in a high proportion of cases (Crawford *et al.*, 1969).

The decision to operate on vertebral artery lesions will be taken when there are symptoms suggestive of predominantly vertebro-basilar insufficiency and marked disease of both vertebral artery origins. It is usually necessary only to restore normal flow in one vertebral artery. Complete occlusions are rarely

suitable for reconstruction. Frequently, operation on a vertebral artery will be carried out at the same time as a carotid reconstruction.

The technique of operation for internal carotid artery lesions

Carotid stenosis

In the early days of reconstruction of carotid stenoses much thought was given to methods of reducing the risk of a cerebral infarct when flow was completely stopped during the time the common, internal and external carotid arteries were occluded by the arterial clamps. Techniques used to minimize this ischaemic risk included operating under hypothermia, complicated external shunts, internal shunts, operating under local anaesthesia, or a system of temporarily waking the patient for a trial period of arterial occlusion (Coleman & Gillespie, 1963) so as to permit close neurological observation. Further experience, however, has shown

FIG. 9. A smooth stenosis of the left vertebral artery near its origin.

that these techniques, with the exception of the use of the internal shunt in certain circumstances, are unnecessary. It is now the almost universal practice to conduct the reconstruction under general anaesthesia, care being taken to keep the patient's blood pressure at or slightly above the preoperative level, to use oxygen rich anaesthetic mixtures, and to maintain slight hypercarbia (Young *et al.*, 1969).

Where the arteriograms show a tight bilateral carotid stenosis, or a complete internal carotid occlusion on the contra-lateral side to a stenosis to be reconstructed, the risk of serious cerebral ischaemia during operation is greatly increased, and here an internal polythene shunt should be used routinely. Preoperative carotid compression tests, and ophthalmodynamometry with carotid compression, may give some indication of the likely result of operative carotid occlusion, but the absence of ill-effects on such testing does not certainly rule out the risk of cerebral infarction during operation.

The technique of carotid reconstruction is illustrated in Fig. 10a-d. In this instance the arteriotomy, after endarterectomy, has been closed with an onlay

FIG. 10 (a-d). The technique of carotid bifurcation endarterectomy illustrating the use of the temporary polythene shunt, and patch graft closure of the arteriotomy. Actual removal of the atheromatous material is easily accomplished if care is taken to enter the correct separation plane.

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FIG. 10b

patch graft. This may either be of vein or lightweight Dacron material, and it serves to widen the inflow to the internal carotid artery (Fig. 11). There is much divergence of surgical opinion as to the value of the patch graft in this situation and it is by no

FIG. 10c

FIG. 10d

means always necessary. The desirability of removing all the atheromatous material sufficiently far distally in the internal carotid artery to reach a healthy and well-adherent intima must be stressed. Any distal intimal shelf which has to be left must be carefully tacked down to avoid dissection of blood into the wall of the artery when flow is restored. The external carotid artery origin must also be endarterectomized in the same way as this vessel may be an important collateral channel for cerebral blood flow.

FIG. 11. Operative photograph of a vein patch graft illustrating the marked increase resulting in the size of the vessel.

In common with many other surgeons the writer finds no use for heparin during or after carotid reconstruction: it will not redeem the results of technical imperfection, while a proper reconstruction needs no heparin to ensure continuing patency.

Carotid occlusion

When the internal carotid artery is completely occluded there is little risk of cerebral ischaemia occurring during operation except in extensive multi-vessel disease where blood flow via the external carotid collateral pathway is vital for cerebral oxygenation. The likelihood that flow up the internal carotid cannot be restored in complete occlusions has already been discussed, and in this instance every effort must be made to ensure that external carotid artery flow will be maximal. This may sometimes involve using part of the internal carotid artery in a plastic repair of the origin of the external (Fig. 12a-d) (Jackson, 1967).

The kinked internal carotid artery

This relatively common arteriographic finding is probably seldom the cause of symptoms, but, when it appears to be, the elongated internal carotid artery can be dealt with as illustrated in Fig. 13a and b.

FIG. 12 (a-d). If flow in a totally occluded internal carotid artery cannot be restored its origin may be used after endarterectomy to reconstruct a stenotic external carotid artery, thus increasing this vessel's collateral potential.

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FIG. 12b.

This is much more satisfactory haemodynamically than excising a portion and reconstituting this relatively small calibre vessel by end-to-end suture.

Technical hazards of operation on internal carotid artery lesions

The main risk of carotid reconstruction is a worsening of the patient's neurological state, either as a result of the liberation of emboli during handling of the vessels, or by the cutting off of a vital part of the cerebral circulation during the time the arterial clamps are applied. Rarely, the hypoglossal or vagus

FIG. 12c.

FIG. 12d.

nerve may be injured, and the cervical branch of the facial nerve may be divided or temporarily damaged by a retractor when the skin incision has to be carried upwards close to the angle of the mandible, leading to some paresis of the corner of mouth.

The technique of operation for orificial vertebral artery stenosis

Anaesthesia is similar to that for carotid reconstruction. The vertebral artery origin is approached by a supra-clavicular incision. The clavicular head of the sternomastoid is divided, and the jugular vein and the tissues medial to the scalenus anterior muscle are separated to reach the arching subclavian artery, from which the vertebral arises superiorly. The arteriotomy is made through the anterior wall of the lower part of the vertebral artery and extends

FIG. 13 (a and b). Reconstruction of an acutely 'kinked' internal carotid artery.

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FIG. 13b

into the subclavian artery. After endarterectomy the vessel may be closed directly, if it is large enough, otherwise a vein or Dacron patch graft is inserted.

The technical hazards of vertebral artery reconstruction include lymphatic fistulae, Horner's syndrome from sympathetic damage, pneumothorax, and the turning of stenotic into occlusive disease as a result of postoperative thrombosis. All these complications are rare, and are largely avoidable with careful technique.

Conclusion

From both the curative and prophylactic aspect a good case can be made out for reconstructive surgery in atherosclerotic disease of the internal carotid and vertebral arteries. The risks of arteriography and operation are acceptably low, and the likelihood of reconstructions remaining patent seems high.

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