The Surgical Treatment of Extracranial Arterial Occlusion

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Patients with occlusive cerebrovascular disease present in a multiplicity of ways dependent on the extent of the disease, the vessels involved, and the anatomical vascular anatomy at the circle of Willis which varies from patient to patient. Surgery should be regarded as a prophylactic measure since once infarction has occurred, correction of the blood flow will not reverse the lesion but only prevent further destruction. Fortunately, a large number of patients first experience warning symptoms. Classically these are transient ischemic attacks involving the retina, amaurosis fugax, due to emboli; transient motor or sensory phenomena in the face, arm and less commonly in the leg when the carotid artery is involved; or episodes of homonymous hemianopsia, vertigo and dizziness when the vertebral artery is involved. Less frequently, as the artery slowly occludes with thrombus and stroke in evolution occurs, an insidious progressive neurological deficit results. In less than a third of cases the initial symptomatology may be of sudden onset causing a fixed neurological deficit which can range from a mild focal defect to a total hemiplegia. Even in these cases a careful history may elicit previous transient ischemic episodes or headaches. Sometimes focal seizures or dementia may be the presenting signs. Cases with dementia often are found to have bilateral internal carotid occlusions or severe stenosis which have not produced any motor or sensory signs.

Rarely when one artery occludes asymptotically, the collateral blood supply tends to steal blood from other arterial beds and may produce symptoms in this manner. We have some examples where one internal carotid is occluded and the other small but normal and the symptomatology has occurred on the normal side due to the other hemisphere stealing too much blood. Another example of the steal syndrome perhaps better recognized is the subclavian steal. The occlusion of the proximal subclavian artery allows the vertebral artery to supply the arm and with exercise the patient steals blood from the basilar supply and produces vertebral basilar insufficiency. In my experience the steal is not uncommon but is usually asymptomatic. With proximal common carotid occlusions an asymptomatic steal from vertebral or internal carotid to external carotid or external carotid on one side to external carotid on the other can occur.

We must always be on guard to detect the disease early. A number of patients may be diagnosed if routine examination includes auscultation of the neck vessels. The asymptomatic bruit or a patient who comes to the doctor because he can hear the bruit, should receive careful assessment. Angiography and surgery should be seriously considered since the operative morbidity and mortality is low if the cases are carefully selected. If the bruit disappears after angiography, emergency surgery is indicated if there is no neurological deficit.

Pathology-Pathogenesis

In the early 1950's and still today there lies a temptation to think of the mechanism of symptomatology as total or severe occlusion of the artery as the commonest type of pathology responsible for symptoms. As time has elapsed however, we now realize that there are a variety of mechanisms and that reduction of blood flow is just one and is perhaps less common than embolic phenomena which can arise from a stenosis, post stenotic dilatation or from a rather insignificant ulcer with little stenosis of the vessel. Furthermore, we clearly recognize emboli going to the retina because we can see them but now we know it is just as feasible to think of emboli going to the brain. Perhaps some are silent because of the size of the vessels involved and others produce symptoms when emboli are larger and obstruct more major vessels. Careful examination of the angiograms may show branch occlusions.

Another curious phenomena is the patient who continues to have transient ischemic at-
tacks after the internal carotid artery becomes occluded in the neck. This may be explained by the ulcerating plaque extending into the external carotid artery so that the emboli travel through the collateral ophthalmic supply to the internal carotid and hence to the middle cerebral artery. We have had examples of this situation where endarterectomy of the external carotid has stopped the attacks.

An unusual lesion seen only rarely is a diaphragm which occurs in the internal carotid near its origin. The diaphragm is thus not atheromatous in nature and can only be seen if the angiogram is taken in the plane of the diaphragm. Only chance will demonstrate this pathology.

**Investigations**

The reduced pulsations in the neck and the superficial temporal and facial arteries are only of help if there is a major difference. Often the internal carotid may be completely occluded and there is no detectable palpable difference. A bruit over the carotid or at the base of the neck may be very helpful in localization and occasionally, orbital and temporal bruits increase the index of suspicion. Measurement of arterial ocular tension may be helpful and thermography is said to be useful although in our series we have not used these methods regularly.

Angiography is the definitive means of diagnosis and the route used depends upon the suspected site of the disease. If the vessels of the arch are involved then trans femoral catheterization with arch, neck and skull films is the best means of assessing the total cerebral blood flow. Care should be exercised however to determine that the femoral artery at the site of puncture is not extremely diseased. Often we have contented ourselves with a right retrograde brachial and percutaneous left carotid angiogram when the suspected lesions are in the internal carotid artery. When lesions are suspected in the vertebral system an arch study or bilateral brachial angiograms are done. Careful assessment of the lesions; single, multiple and the collateral circulation of the circle of Willis, is carried out in preparation for surgery. Should the patient have bilateral carotid stenosis or occlusion on one side and stenosis on the other or an absent A1 segment of the anterior cerebral on the side of the lesion to be repaired, then a bypass shunt would be used at the time of surgery. It is indeed important therefore to assess the collateral flow pre-operatively to avoid any ischemia at the time of temporary occlusion.

Carotid compression is not warranted as the information gained from such a test is not reliable. It also may be complicated by fracturing of the plaque, thrombosis of the vessel or embolization.

**Selection of Patients for Surgery**

Most patients presenting with transient ischemic attacks or a minor stroke will be candidates for surgery even if they have had a previous coronary occlusion. Age has not been a major factor. The oldest patient in the series is 79 years of age. Attention should be paid to the physiological age rather than the chronological age as some patients at 75 look like 65 and are better risks than a patient of 45 with wide spread, rapidly advancing vascular disease and a history or previous coronaries. Indeed, the younger patients with hypertension and a bad vascular family history may have difficult arterial tissue to deal with at the time of surgery. The plaques may be very extensive and the upper margin may not return to normal intima thus leaving a rough upper edge. This is particularly true of diabetics whose intima is extensively involved and a good endarterectomy is not possible. Mild diabetics may be considered for surgery but severe ones should be avoided. Serious cardiac decompensation, multiple coronaries, renal disease and general debility are all contra-indications for surgery. Acute cerebral infarction should also be a contra-indication and if the patient is drowsy, this means a large infarct, and surgery would only result in a red infarct or death.

In the sixty to eighty age groups the lesions are surprisingly discreet occupying a one to two centimetre length of artery and terminating above in normal intima. In fact the plaque splits the intima into inner and outer layers so when dissection is carried up to the upper margin of the plaque, the deeper intima is left on the vessel wall creating a bevelled edge to the upper end of the resection. Thus the repair
may be of better quality in this age group.

**Pre-operative Management**

Pre-operatively most patients are maintained on anti-coagulants (Dicoumeral) with prothrombin times less than 23 seconds, less than twice the control.

**Surgical Technique**

Care must be taken not to operate upon the patient if the prothrombin time is above the stated level. Heparin, 100 International Units per kilogram of body weight, is given at the time of endarterectomy if a shunt is to be used. Also during the procedure hypothermia to 30 degrees Centigrade is used unless the vessel is totally occluded. Care is taken to maintain the blood pressure at normal or above normal throughout the procedure as a minor drop may further embarrass the circulation to the brain. In normotensive patients the blood pressure is elevated 30 mm. Hg. during the occlusion time to increase perfusion. In hypertensive patients every attempt is made to maintain the blood pressure, or elevate it slightly if in our judgement the patient's cardiac reserve will tolerate it. Monitoring of the blood pressure and E.E.G. throughout the procedure is done and used to assess the adequacy of the shunt to be certain ischemia is not taking place. The occlusion time without a shunt is usually kept under 20 minutes. However, if the collateral circulation is adequate longer periods will be tolerated. General anaesthesia is used to facilitate hypothermia, comfort for the patient and the low anaesthetic morbidity does not warrant the use of local anaesthesia.

The technique of dissection and exposure will be demonstrated in a film.

A generous exposure is preferred to allow adequate exposure of the internal and common carotid arteries. This facilitates the use of a shunt and also if the plaque is larger than anticipated an extensive incision in the common and internal carotid may be made without being hampered by inadequate exposure. Some of the failures can be attributed to inadequate exposure and too rapid dissection of the plaque, hence not obtaining a smooth upper intimal cut. The common, internal and external carotid arteries are not completely dissected out of their beds. There is no need to do this and it only produces bleeding deep to the artery and may damage the carotid sinuses. After dissection Papaverine is injected into the adventitia. This dilates the internal carotid artery and gives a thicker wall to sew up after completion of the endarterectomy.

Then the artery is opened and retrograde flow is checked in the internal carotid. If a shunt is needed it is now inserted. We use an internal shunt as it is easy to insert and remains out of the way during the plaque resection. The plaque is now peeled out of the vessel. Care must be taken to remove all of the plaque from both the internal and external carotids. Usually in the common carotid one has to leave some thickened intima but a sharp cut is made so the edge is smooth. The bed of the endarterectomy is then washed thoroughly with saline and all tags which will float up are removed. Failure to be meticulous and not rush this part of the procedure will result in irregular upper and lower intimal margins as well as tags projecting from the inner surface which can cause embolic and thrombotic problems post-operatively. Artificial prosthetics are not used as the high failure rate of these arterial grafts has proven the patient's own tissue to be superior. We rarely use a vein patch but occasionally the diameter of the internal carotid artery is too small and this is expanded by a vein patch. Transposition of the external carotid artery to the internal carotid artery is not a good procedure as it takes away collateral blood flow.

The vessel is closed with 5–0 prolene which does not saw through the vessel when the suture is snugged up tight.

**Vertebral Arteries**

Endarterectomy of the vertebral arteries is not as successful as the plaque strips poorly and the vessel wall is too thin. In these cases a vein patch is necessary rather than endarterectomy to ensure adequate lumen size. These plaques rarely ulcerate. A better procedure is to transfer the vertebral artery to the thyrocervical trunk if it will reach. For the left vertebral the chest should be entered to give sufficient exposure. The left subclavian artery needs thoracic and neck exposure. If endarterectomy can not be
accomplished due to extensive disease in the aorta, then a bypass jump vein graft from the carotid artery to the distal subclavian artery is usually used. Similarly, proximal common carotid and middle cerebral jump vein grafts may be used.

Post-operative Management

The neck size is checked regularly to determine the presence of any post-operative clot. Patients are mobilized within 24 hours after their blood pressure has been checked. Some patients may become mildly hypotensive due to carotid sinus irritation. The patients are maintained on Dicoumeral for six months with regular checks on their anti-coagulant levels. A repeat angiogram is done at six months follow-up.

Results

We first commenced carotid artery surgery after 1953 when I had occasion to operate with Dr. Hamlin and Dr. Sweet in Boston. Earnest attention was given to looking for cases in 1954 but the numbers were meager because referring doctors were unaware of what to look for and thus early diagnosis was rare. Therefore our earlier cases were frequently varying degrees of completed strokes. Patients suffering transient ischemic attacks and those with bruits found upon examination had to await the increasing awareness of the referring physician. Since 1954 the number of patients treated surgically has greatly increased as it has become clear that with selection and early diagnosis, prophylactic surgery is useful and the low morbidity of 2% and mortality of 2% indicates the low risk involved.

Patients with a total carotid occlusion with no major infarct, operated upon under three days have a good chance of a patent carotid 75% of the time. After the three day interval has passed the only group in whom the artery could be opened were those who presented with transient ischemic attacks. Successful reconstruction was possible in approximately half of these cases. The block was focal and made up of calcium. There was no clot so the vessel above had no intimal break and the blood in it remained unclotted. Presumably some sluggish flow from collateral circulation had kept it patent.

In the stenotic group the long term patency rate was 96% and only a few patients have turned up with recurrent stenosis. Usually these could be traced to peri-arterial scarring although two patients have shown recurrence of atheroma.

Summary

The conclusions therefore must be that the recurrence of atheroma is late, some ten to twenty years, and not common following endarterectomy. Excellent patency with smooth vessels may be attained and low morbidity and mortality indicates that we have extended this group of patients’ life span. I have not encountered any reported series of medical therapy or no therapy where the results are as good. It is true that this is not a controlled series but I would have to be shown satisfactory evidence before I could morally decline to operate in properly selected patients.