Stroke-prone patients

focus on carotid surgery and stenting

Carotid endarterectomy effectively reduces the risk of stroke. Will carotid artery stenting prove to be as effective, and any safer?

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Over two-thirds of strokes related to carotid artery disease can be prevented by open carotid endarterectomy, with its established techniques, benefits and proven durability.[1,2] Percutaneous carotid stenting is now feasible, attractive and available in major vascular surgery centres, but has yet to be validated. How do clinicians identify someone prone to an arterial stroke, while managing all the other potential stroke risk factors? How should the carotid arteries be investigated? What evidence guides our selection of the best surgical intervention for each patient?

Patient identification

The stroke-prone patient can be identified with clues from the history and on physical examination.

Clues from the history

Risk factors for stroke include hypertension, smoking, hyperlipidaemia, atrial fibrillation, a close family history of stroke, coronary disease and intermittent claudication.

A minor stroke or a recent transient ischaemic attack (TIA) strongly suggests the possibility of carotid disease that may benefit from surgical intervention. Amaurosis fugax (fleeting monocular blindness) has several causes and is less threatening of stroke than of visual loss.[3]

At patients' annual check ups it is important for GPs to question carefully about transient ischaemic events or visual deficits. Frequently, patients fail to volunteer these, as they have been brief and thus may seem inconsequential.

Table 1 lists features that suggest vulnerability to an arterial stroke.

Clues on examination

Neck and skull bruits should be sought in symptomatic patients and routinely in at-risk patients (e.g. at annual check up). If present, they indicate disease of a carotid artery or one of its branches. Neck bruits correlate quite well with

IN SUMMARY

- **Transient cerebral symptoms and neck bruits are the key warnings of immediate stroke risk. A TIA is a cerebral emergency – start aspirin, and investigate promptly.**
- **Although atheroembolism from unstable carotid plaques is a frequent cause of stroke, carotid stenosis guides patient selection for surgical intervention.**
- **In the last decade, imaging of carotid disease by duplex ultrasound studies and angiography has improved in detail and safety.**
- **Carotid endarterectomy provides evidence-based stroke prevention in selected patients.**
- **Carotid artery stenting is a percutaneous alternative to open operation but is on trial; currently, its efficacy is unproven and it carries significant risks.**
- **Modern stroke prevention combines surgical intervention (both open and endovascular carotid surgery) with the medical management of multiple risk factors for stroke, such as hyperlipidaemia, hypertension and hyperviscosity.**

the coexistence of coronary atheroma; deficient (reduced or absent) leg pulses also suggest widespread atheroma.

In the neck, carotid bruits are best heard by asking the patient to stop breathing for a moment, with the stethoscope placed at the carotid bifurcation, just below the mandible. They should be distinguished from sounds transmitted from the aortic valve. With severe stenoses of the intracranial carotid siphon, bruits may be heard over the temple or orbit.

Subclavian artery stenoses and bruits are common in atheromatous patients, who often have asymmetrical brachial blood pressures: a 15 mmHg or greater difference suggests significant stenosis and impairment of subclavian or vertebral artery blood flow.

Unfortunately, 'silent' plaques without a bruit can also be very stroke-threatening. Examples are ulcerated plaques generating atheroemboli, or severely stenosing plaques reducing flow to a trickle and poised to thrombose (Figures 1a and b).

Table 2 lists presentations of carotid disease.

Investigation

Duplex ultrasound studies are the most commonly used way of confirming or evaluating disease at the carotid bifurcation. Arteriography is used in the selection of operative approach, and during operations and stenting.

Ultrasound

The cerebrovascular duplex (B-mode and Doppler) ultrasound study is a convenient, noninvasive way to triage carotid disease. It is simple but very operator-dependent. Properly conducted, it shows plaque characteristics and blood flows in the cervical part of each carotid and vertebral artery. It estimates rather than measures stenoses, according to the disturbance of blood flow caused.

Ultrasound can be repeated – for example, after 6 to 12 months to assess the progression of medically managed carotid stenosis and for surveillance after surgical management of carotid disease. Ultrasound is also good for screening patients who have a strong family history of stroke, or who have atheroma in the limbs.

Table 1. Carotid arterial stroke-prone patients: features of vulnerability

- Transient ischaemic attacks (TIA hemispheric or syncopal)
- Amaurosis fugax (fleeting monocular blindness)
- Harsh neck bruits
- Coronary and other atheroma
- Family history of stroke
- Hypertension
- Hyperlipidaemia
- Smoker's lungs

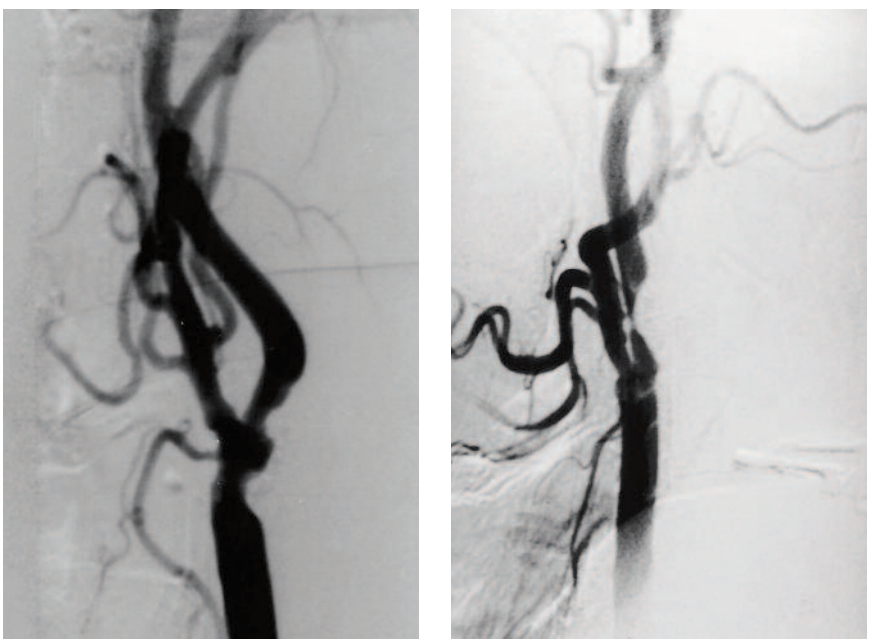
Table 2. Carotid arterial disease: presentations

- Transient ischaemic attack (TIA)
- Amaurosis fugax
- Vertebrobasilar ischaemia
- Established stroke
- Stroke-in-evolution
- Symptomless neck bruit

Arteriography

Arteriography provides the clearest detail of the carotids and their plaques, and allows precise measurements to be made of the arterial lumen and stenosis, whereas ultrasound generally provides only an estimate of the degree of stenosis at the carotid bifurcation (e.g. 'in the range 50 to 69%'). For this reason, arteriography was used as the scientific evidence basis of the best-regarded trials of carotid endarterectomy. By showing the full length of the cerebral supply vessels, from thorax to brain, arteriography still allows the most reliable patient selection for surgery.

The box on page 37 describes three current techniques of arteriography. Diagnostic arteriography using a fine arterial catheter carries small risks of bleeding at the puncture site or stroke (less than 1% in skilled hands). The two other arteriography techniques, magnetic resonance and computed tomographic arteriography, are less invasive, using intravenous injections of contrast or nontoxic contrast.



Figures 1a and b. Carotid bifurcation angiograms showing ulceration and stenosis. a (left). Ulceration. b (right). Stenosis.

Digital subtraction arteriography is also used during many carotid operations, and for all stentings, whether percutaneous or combined with an open arterial reconstruction.

Computed tomography

A CT or magnetic resonance scan of the brain will assess the integrity of the end organ for the purpose of vascular surgical decision-making. It will also rule out other stroke-threatening neurological lesions (e.g. intracranial tumours or aneurysms).

Management

The key surgical questions are:

- which carotid lesions post a significant threat of stroke?
- how urgent is their correction?
- should the patient be offered open carotid endarterectomy or percutaneous carotid stenting or open surgery combined with stenting?

Before surgery

A matter of urgency

Logic requires effective preventive measures before stroke occurs. The threat of stroke correlates with instability of the carotid atheroma as well as the severity of stenosis it produces. Plaque ulceration allows repeated atheroembolism of particles to the brain, just as important as flow-critical carotid stenoses of 95 to 99% that may occlude or thrombose.

Thus, a TIA is a cerebral emergency – akin to myocardial ischaemia – so start aspirin (see later) and investigation at once. Definitive surgical treatment should be sought as soon as possible, and preferably within a day or two of a TIA. Repeated (or 'crescendo') TIAs are the most urgent situation; delays can kill. Established neurological deficits (i.e. strokes) usually require two to six weeks for the brain infarct to stabilise and heal prior to definitive carotid surgery; despite continuing research, no anticoagulant or other treatment is yet proven to limit or speed

the recovery of the acute stroke. The box on this page outlines the optimal timing of surgical intervention.

Australia is well supplied with ultrasonographers, high quality angiographers, neurologists and vascular surgeons, so quick, safe diagnosis is readily available. When diagnostic doubt exists, a neurologist should be involved in the management team.

Pending further investigation

Platelet inhibition

Aspirin, 100 mg/day, is appropriate from the start, as most angiography and carotid surgery is neurologically safer with platelet stickiness inhibited.

Maintaining perfusion

High blood pressure, usually present, should not be dropped precipitately until after any critical carotid stenosis is relieved surgically.

Selection for surgery

Any advice about surgical or nonsurgical carotid management needs to be clearly understood by the patient, carefully weighing up possible risks and benefits. Stable or dormant carotid lesions should not be treated 'just because they're there' – for example, if discovered in passing during coronary angiography.

Long term stroke minimisation may not apply to patients harbouring a cancer in smoker's lungs, or with severe cardiac or other disease limiting their life expectancy.

Success in carotid therapy correlates well with the number of patients being selected and operated on annually by the vascular surgery team, and depends very much upon timely treatment.

Surgery

Carotid endarterectomy is still the proven approach for correcting carotid lesions. The scope of an open operation may now be extended transluminally by ballooning and stenting techniques, guided angiographically in the operating or endovascular theatre. Percutaneous carotid stenting is being developed and evaluated. Before it can be widely recommended, we must await its technical refinement and proof, in randomised trials, of its efficacy, safety and durability.

Carotid endarterectomy

Carotid endarterectomy has been refined over many years since first performed in 1953 by Michael DeBakey in Houston, Texas.[4] Its safety and efficacy in stroke prevention has been proven by many well accepted studies.

The worldwide North American Symptomatic Carotid Endarterectomy Trial (NASCET) had earlier established the superiority of open carotid endart - erectomy over medical treatment, in reducing strokes by 17% in symptomatic patients with a carotid stenosis that was greater than 70%.[1] The NASCET analysis has now concluded, confirming the benefit of surgery also for patients with moderate degrees of carotid stenosis (greater than 50%).[2] Benefit correlates with the severity of stenosis measured by angiography, and is sustained in the long follow up period.

Asymptomatic patients with a greater than 60% carotid stenosis also benefit from a carotid endarterectomy when performed at a high standard,[5,6] which Australian vascular surgeons have established. The major European studies have been reanalysed using NASCET angiographic criteria; their results concur.

Carotid artery surgery – optimal timing

- Before the next stroke occurs
- When neurological deficit has stabilised
- Within 24 hours, if the carotid stenosis is flow-critical or thrombosing
- Occasionally, during a stroke-in-evolution

Carotid arteriography – current techniques

Digital subtraction arteriology (DSA)

DSA involves catheterisation of the femoral or brachial artery, nowadays a simple and relatively safe procedure in skilled hands, using modern apparatus. Most angioradiologists and vascular surgeons encourage day-patient carotid DSA, unless the patient is unstable or has arteries that are difficult to access . Of the three techniques described here, DSA most clearly shows the full length of all four cerebral vessels and the circle of Willis, and best defines any stroke-threatening lesions.

Magnetic resonance arteriography (MRA)

MRA is useful for patients with x-ray contrast sensitivity, or poor arterial access for catheter arteriography. It is excellent for defining intracerebral lesions; however, it is expensive, and prone to artefacts and lack of image clarity in the neck and chest. Patients with some pacemakers, heart valves and ferromagnetic prostheses cannot undergo MRA.

Computed tomographic arteriography (CTA)

CTA acquires pictorial information in x-ray 'slices' or 'spirals', then reformats the data to show the arteries longitudinally, like an arteriogram. It requires a contrast injection, and is subject to patient movement and processing artefact. It can be combined preoperatively with a CT scan of the end organ, the brain.

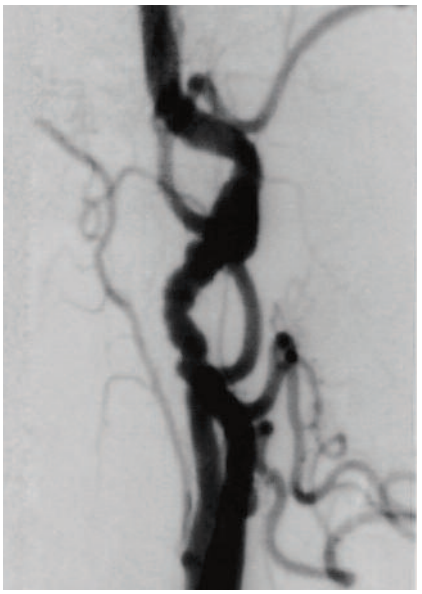


Figure 2. Internal carotid artery showing fibromuscular hyperplasia stenosing ridges.

Technique

Carotid endarterectomy involves an incision in the neck, allowing control of the carotid artery and its branches. The arteries are clamped, then opened by an arteriotomy which enables the atheromatous plaque to be shelled out. This relieves any stenosis and leaves a smooth flow surface lined by external elastic lamina.

If collateral circulation to the brain via the circle of Willis is insufficient, a temporary internal carotid shunt is used to maintain cerebral flow. The arteriotomy is then closed by monofilament suture, although some arteries require repair with an arterial patch or graft. Blood transfusion is very rarely needed.

Some anaesthetists use regional anaesthesia, but most patients, even elderly ones, are suitable for carotid surgery under general anaesthesia. This allows best control of the heart, which is the main operative risk. Further, carotid endarterectomy can be performed synchronously with a coronary bypass operation, in patients who have the dual indication.

Complications

In busy vascular surgery units around Australasia, a combined mortality and major morbidity rate of 4 to 6% is experienced for carotid arteriogram plus endarterectomy. The main dangers are myocardial ischaemia (1 to 2%), stroke (1 to 2%), cranial nerve damage, and bleeding.

The patients at greatest operative risk are those with bilateral carotid disease, uncorrected coronary disease, diabetes, recent stroke, as well as women and continuing smokers.

Aftercare

The hospital stay for carotid endarterectomy is usually two to five days.[7] Aspirin, aspirin/dipyrimadole (Asasantin SR) or clopidogrel (Iscover, Plavix) is continued indefinitely in patients.[8] Return to driving and work is usual in one to two weeks if the neck wound has healed comfortably.

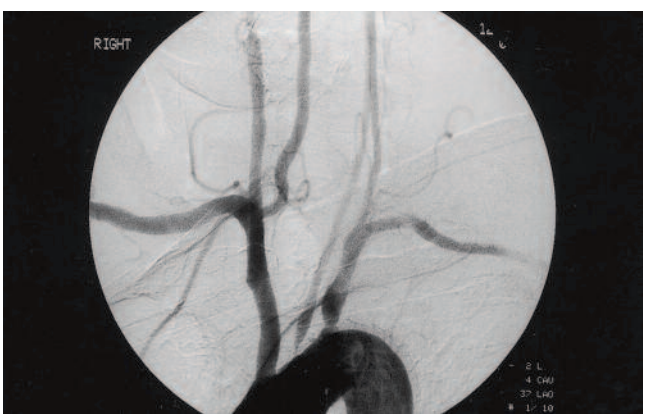
Results

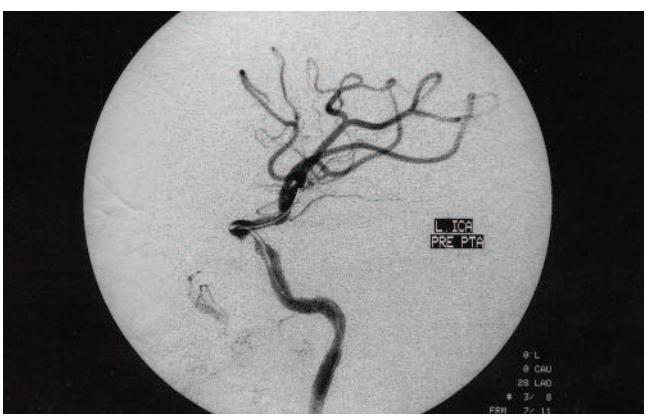
The vast majority of patients have an enormous reduction in their chance of developing a stroke, provided they are selected appropriately for operation. Each year, about 1% of patients experience a further stroke, often remote from the operated hemisphere. About 5% of patients show late restenosis, usually benign, and reoperations are few.

Carotid stenting

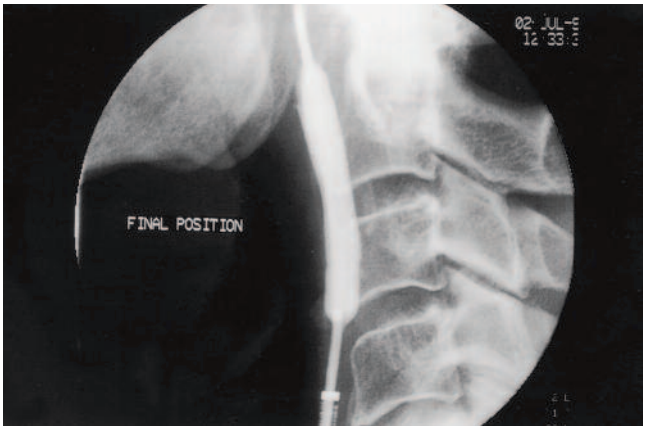
The technological fascination of endovascular surgery is undeniable. Endovascular surgery techniques are being used to balloon and stent many arteries round the body, including some carotid arteries. Patients often hope for and request such 'keyhole' solutions. But however tempting, the risks, efficacy and durability of carotid stenting are yet to be established.[9]

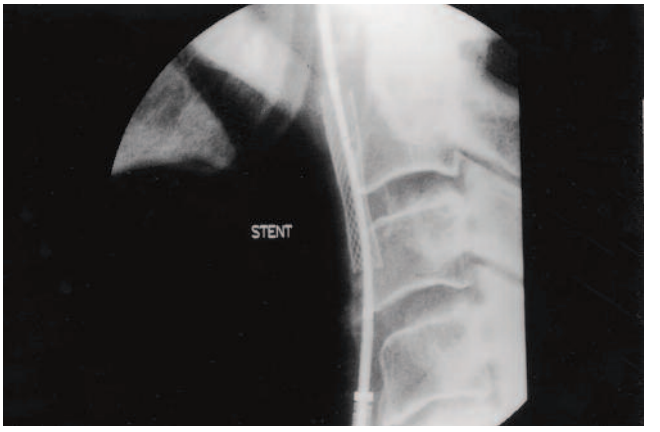
Although some arterial lesions respond well to balloon dilatation (angioplasty) without needing a stent, others rebound at once, or restenose over some weeks or months. So, many arteries will be stented primarily, particularly small ones like the 4 mm diameter carotid arteries, and 2 to





Figures 3a and b. Lesions treated in tandem with carotid bifurcation endarterectomy. a (left). Severe intrathoracic stenosis of left common carotid artery origin. b (right). Severe stenosis of the intracranial siphon undergoing transluminal balloon dilatation (intraoperatively).





Figures 4a and b. Carotid artery stenting via a percutaneous transfemoral catheter. a (left). Atheroma being dilated with a 5 mm diameter balloon. b (right). The self-expanding stent in place.

3 mm diameter coronary arteries. The aim of implanting a stent is to widen the carotid artery lumen and stabilise its lining.

At this time, stroke-prone patients suitable for carotid stenting include those with the following conditions:

- neck irradiated, or otherwise difficult to approach by conventional operation
- tandem lesions 'inaccessible' in the intrathoracic or intracranial carotid arteries
- early restenosis after carotid surgery or ballooning
- web-like stenoses of fibromuscular hyperplasia (Figure 2)
- carotid aneurysms, dissections and injuries.

Patient selection for carotid stenting requires catheter arteriography (see the box on page 37). Lesions found to be in tandem with ones at the carotid bifurcation may take priority in treatment – for example:

- severe atheroma in the innominate or proximal carotid artery (Figure 3a)
- siphon stenosis amenable to transluminal ballooning (Figure 3b).

Technique

There are two approaches to implanting a stent, both using x-ray guidance in an endovascular arteriogram facility. In one approach, the carotid artery is selectively catheterised via a puncture of the femoral or brachial artery, under a local anaesthetic (Figures 4a and b). In the other, the carotid artery lumen is directly catheterised, proximally or distally, for stent placement during an open operation on the carotid artery, usually under a general anaesthetic .

In both approaches, the catheter system is guided through the lesion, then the stent is deployed and expanded, the apparatus withdrawn and a completion angiogram done. Figure 5 shows a balloon catheter being inserted, with x-ray guidance, to dilate a stenosed origin of the common carotid artery before placing a stent.

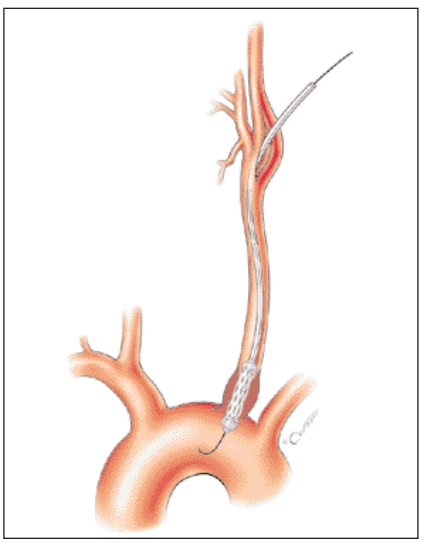


Figure 5. During carotid bifurcation endarterectomy a balloon catheter is x-ray guided to dilate the stenosed common carotid origin and place a stent.

Stents are permanent metallic implants that, as stated earlier, aim to stabilise the diseased carotid lining against generating emboli, as well as maintaining the luminal diameter. Some stents are self-expanding springs, while others require expansion by an angioplasty balloon to achieve the desired diameter. All are foreign bodies, subject to thrombosis if blood flow slows, and prone to stenose by an ingrowth of excessive endothelium or neointima. So, platelet inhibitors are used during the procedure and lifelong afterwards.

Complications

Arterial ballooning and stenting work better in larger arteries; hazards multiply when small arteries like the carotids (as well as popliteal, tibial and coronary arteries) are tackled. The complications of carotid stenting include stroke, arterial damage or occlusion, and restenosis.

Stroke. A risk of cerebral atheroembolism exists whenever a diseased carotid, brachiocephalic or subclavian artery is catheterised. Guide wires and catheters

can easily disrupt the fragile atheromatous plaques. At present, percutaneous stenting of a carotid artery carries an 8 to 15% risk of stroke, in series reported around the world;[10] and, at this level, some series have been abandoned. Special embolus-trapping catheters are being developed to try to improve the safety of 'closed' or percutaneous stenting procedures.

Arterial damage or occlusion. Stents in the neck are inevitably subjected to flexion and torsion, more so than stents in protected areas like the abdomen and pelvis; cerebral arteries also tend to have delicate walls. Better stent materials and configurations for the neck are being sought.

There is extreme difficulty in retrieving carotid stent complications, especially acute occlusion, thrombosis or rupture of the artery and displacement, fracture or maldeployment of the stent. The candidate for carotid stenting is often the very patient for whom surgical access to repair a disaster is difficult or impossible – perhaps near the skull base or deep in scar or irradiated tissue.

Operative removal of a carotid stent and restoration of a carotid artery is a very major undertaking.

Restenosis. The process of restenosis bedevils current stentings all around the body and is particularly hard to manage in a carotid artery. Up to 30% of stents restenose within the first year. Irradiation of the lesion during stent insertion, aiming to minimise the unwanted neointimal or myoendothelial ingrowth, which so often compromises the stented lumen of a small calibre artery, is being studied.

Aftercare

Most stented patients stay overnight in hospital for initial antithrombotic therapy with heparin or dextran (Dextran 40 Intravenous Infusion BP, Dextran 70 Intravenous Infusion BP, Macrodex, Rheomacrodex). Then, they remain long term on platelet inhibitors such as aspirin or clopidogrel.

Just like after any arteriogram, the integrity and comfort of the femoral artery puncture will determine the patient's return to activity, work and driving during the next few days.

Results

Surgeons and stroke neurologists in Australia are studying the morbidity and durability of cerebrovascular stenting, just as closely as they have analysed the effectiveness of open carotid endarterectomy. The lack of neurologist scrutiny and the short or nonexistent follow up in many series reported so far leave us unsure of the benefit to risk ratio for patients considering a carotid stenting procedure. Further, the ideal stent is not available yet and many types are being

implanted – the mixture of devices and techniques still evolving make the assessment of results difficult.

A proper randomised comparison of carotid stenting versus endarterectomy, the 'CREST' study, has just been funded by the US National Institute of Health and will eventually provide us with the required quality of evidence.

Cost and availability

In Australia, stents cost from about $500 to $2000. Disposable catheters and equipment for the implantation add further to the material cost, as does the expensive arteriographic guidance required for stenting. Compare the five dollar cost of an arterial suture to sew up the carotid after an endarterectomy!

Australia is fortunate to have centres of expertise where endovascular surgeons, neurologists and interventional radiologists collaborate closely to evaluate the new technique of stenting for safeguarding the cerebral blood supply. As is appropriate with new treatments, teams proceed cautiously, concentrating experience in a few hands and auditing outcomes carefully.[11] On offer are open balloon arterioplasty for selected carotid and siphon lesions, and closed stenting for some innominate, subclavian and carotid artery lesions.

After surgery

Follow up surveillance

Operated or stented carotids merit non-invasive follow up. Carotid lesions progress at an unpredictable rate, and are prone to sudden plaque disruption.

For operated carotids, duplex Doppler ultrasound studies after 3 to 12 months may be used to check the treated carotid vessel as well as any progression of atheroma in the opposite artery.

In this developmental phase of stenting, follow up is by six-monthly duplex ultrasound studies of the stent, together with neurological examination.

Risk factor modification

The atheroma process affects the arteries overall and is a progressive, currently irreversible, degenerative disorder. With or without surgical intervention, the stroke-prone patient needs multipronged medical management for cerebral and cardiovascular risk reduction. Table 3 lists the mechanisms by which arterial stroke can occur; risk factor modification addresses many of these mechanisms.

Stroke risk factors that need excluding, eliminating or controlling include hyperlipidaemia, diabetes, smoking, hypertension and other haematological factors.

Treating hyperlipidaemia

The lipid profile will guide metabolic therapy with a balanced low fat diet, regular

Table 3. Carotid arterial stroke: mechanisms

- Atheroembolism (from carotid artery or aorta)
- Occlusion and distal ischaemia
- Thrombosis with propagation
- Thromboembolism (from heart)
- Platelet embolism (from plaque ridges)
- Stenosis and hypotension
- Polycythaemia and hyperviscosity

exercise, reducing overweight, and, if indicated, lipid lowering drugs. Metabolic factors are especially important if the atheroma is prematurely severe, as when significant carotid artery disease is found in young patients.

Treating hypertension

Long term reduction of hypertension is beneficial, and remediable renovascular stenoses are commonly found in these carotid patients. As mentioned earlier, any sudden lowering of systolic hypertension should be avoided until a severe carotid stenosis has been excluded or corrected. A stroke may be caused by suddenly dropping the perfusion pressure for rocky arterial beds, healing cerebral infarcts or cerebral capillaries with already borderline flow.

Platelet inhibition

Platelet inhibition with aspirin is beneficial preoperatively and intraoperatively, and is usually continued for life, in view of disease in other vessels. Clopidogrel or ticlopidine (Ticlid, Ticlopidine Hexal, Tilodene) can further reduce other cardiovascular events.

Anticoagulation

Anticoagulation with warfarin (Coumadin, Marevan) is indicated in two groups of patients:

- those with atrial fibrillation or prosthetic cardiac valves
- those with brainstem or vertebrobasilar ischaemia due to basilar artery disease (confirmed on angiography).

Both groups will benefit from additional stroke protection with warfarin but with some added risks of bleeding even when well monitored. In those with basilar artery disease, the subclavian and vertebral arteries may need treating as well as the carotid arteries.

Heparin is unproven in acute stroke, but may be used to discourage a trickling carotid lumen from thrombosing until an operation can be arranged. Heparin, dextran, abciximab (ReoPro), urokinase (Ukidan) and other thrombolytics may be used during angiosurgical intervention.

Treating hyperviscosity

Viscosity is an often neglected aspect of blood circulation, and the hyperviscosity of dysproteinaemia or polycythaemia should be treated; venesection or haemodilution may be necessary. Smoking contributes to stroke risk by, among other things, increasing red cell rigidity and viscosity.

Conclusion

Open carotid endarterectomy has been thoroughly examined and proven in the management of the stroke-prone patient. Carotid stenting, like other forms of endovascular surgery, is attractive to clinicians and patients; however, it needs similar close scrutiny before it can be recommended and widely introduced as a stroke-preventive.[12] Major vascular surgery units and stroke neurologists around Australia and the world have the responsibility of proving the potential of this daring, new approach to preventing strokes. MT

References

1. Barnett HJ, Taylor DW, Eliasziw M, et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. N Engl J Med 1998; 339: 1415-1425.
2. Clinical alert: benefit of carotid endarterectomy for patients with high-grade stenosis of the internal carotid artery. National Institute of Neurological Disorders and Stroke Stroke and Trauma Division. North American Symptomatic Carotid Endarterectomy Trial (NASCET) investigators. Stroke 1991; 22: 816-817.
3. King J. Loss of vision. Mod Med Aust 1990; 33(5): 52-61.
4. DeBakey ME. Successful carotid endarterectomy for cerebrovascular insufficiency. Nineteen-year follow-up. JAMA 1975; 223: 1083-1085.
5. Endarterectomy for asymptomatic carotid artery stenosis. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. JAMA 1995; 273: 1421-1428.
6. Hobson RW 2d, Weiss DG, Fields WS, et al. Efficacy of carotid endarterectomy for asymptomatic carotid stenosis. The Veterans Affairs Cooperative Study Group. N Engl J Med 1993; 328: 221-227.
7. Lord R. Overnight hospital stay for carotid endarterectomy [reply to letter]. Med J Aust 1998; 168: 640.
8. Taylor DW, Barnett HJ, Haynes RB, et al. Low-dose and high-dose acetylsalicylic acid for patients undergoing carotid endarterectomy: a randomised controlled trial. ASA and Carotid Endarterectomy (ACE) Trial Collaborators. Lancet 1999; 353: 2179-2184.
9. Clinical practice guidelines. Prevention of stroke. The role of anticoagulants, antiplatelet agents and carotid endarterectomy. Canberra: NHMRC, 1997.
10. Jordan WD Jr, Voellinger DC, Fisher WS, et al. A comparison of carotid angioplasty versus endarterectomy with regional anesthesia. J Vasc Surg 1998; 28: 397-403.
11. Bladin CF, Davis SM, Burton K, et al. The use of percutaneous transluminal angioplasty (PTA) for the treatment of extracranial atherosclerotic vascular disease. The Australian Association of Neurologists (AAN). Aust N Z J Med 1998; 28: 654-656.
12. Hankey GJ. Transient ischaemic attacks and stroke. Med J Aust 2000; 172: 394-400.