Anaesthesia for carotid endarterectomy

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Summary  Cerebrovascular disease is a major public health problem in the Western world. Carotid endarterectomy reduces the risk of stroke in patients with symptomatic carotid stenosis, but itself is associated with appreciable morbidity and mortality. Careful patient selection is therefore necessary, and this is informed by several large multi-centre trials. The relevant anatomy and pathology are first outlined below with a description of the basic surgical technique. The relative merits of general and regional anaesthesia are discussed, with particular emphasis on detailed pre-operative assessment. The role of various cerebral monitoring techniques is explored together with the common and serious complications that may be encountered in the perioperative period.

Introduction

Cerebrovascular disease is the third largest cause of death in the developed world after heart disease and cancer. Each year approximately 130,000 people in the UK suffer a stroke, and many more will have a transient ischaemic attack (TIA). Within a year of these cerebrovascular events, a third of patients will have died (usually a cardiovascular death), and of the survivors, nearly 40% are dependent on others for help with activities of daily living.1 In 1996, the financial burden to the NHS and social services was estimated at £2.3 billion, or around 5.8% of NHS and social services expenditure.2 It is estimated that 5–12% of new strokes are caused by carotid artery occlusive disease which is amenable to surgical intervention in the form of carotid endarterectomy (CEA).

Epidemiology and patient selection

Since CEA carries a risk of death or disabling stroke, appropriate case selection is crucial in determining which patients are likely to benefit from intervention. This selection process is informed by several large randomised controlled trials. The North American Symptomatic Carotid Endarterectomy Trial (NASCET) randomised patients with carotid stenosis of 70–99%, and recent stroke or TIA in the territory of the affected artery, to receive either maximal medical therapy or surgical endarterectomy. This demonstrated that in those that were surgically managed, there was a 17% absolute reduction in incidence of stroke in the territory of the stenosed carotid artery at 2 years.3 The more
recent European Carotid Surgery Trial (ECST) randomised 3024 patients across 97 European centres with symptomatic disease to CEA or medical management. The trial showed that CEA was worthwhile for patients with a carotid stenosis of at least 80%, with a number needed to treat of nine to prevent one death or disabling stroke. The perioperative incidence of major stroke or death was 7%, irrespective of the degree of stenosis.

Asymptomatic patients (i.e., those who have not had a stroke or TIA) with carotid artery stenosis may not gain the same benefit from endarterectomy as symptomatic patients. This is in part because almost half of future strokes in the territory of the stenosed artery in this group are either lacunar or cardioembolic in nature, and thus would not be prevented by endarterectomy. Nevertheless, the large European Asymptomatic Carotid Surgery Trial (ACST) enrolled asymptomatic patients with at least 60% carotid artery stenosis and randomised them to endarterectomy or medical management. Over a 5-year follow up period, the rates of stroke were approximately halved in the endarterectomy group (6.4% vs. 11.8%), even when the perioperative stroke and mortality risk (3% in this study) was taken into account. The biggest benefit appeared to be in those patients under 75 years of age.

In those with symptomatic disease, timing of the procedure is also important. Data from various trials show that the greatest long-term risk reduction for stroke is in those who have CEA within 2 weeks of their presentation. This is due to the fact that plaque found overlying a carotid stenosis in recently symptomatic patients usually has a far more unstable profile.

Overall a complex picture is shown. Trial data show in general terms which groups of patients should be offered CEA, but cannot be taken in isolation. Anaesthetic fitness for surgery is important, as are ethnic and sex differences between patients which may influence the likelihood of a successful outcome.

**Pathophysiology**

Atherosclerosis accounts for over 90% of cases of carotid stenosis. Other rarer causes include radiation, dissection, arteritis and fibromuscular dysplasia. Several well-known risk factors increase the predisposition to carotid atheroma formation, including hypertension, hyperlipidaemia, diabetes mellitus and smoking. Atherosclerosis is characterised by remodelling of the vessel wall, with infiltration of inflammatory cells, lipid-laden macrophages and connective tissue, reviewed expertly elsewhere. This process may develop over several decades until a mature plaque develops, characterised by a central lipid core with a fibrous, endothelium-lined cap overlying the luminal side. Due to arterial remodelling, the lumen may not be compromised in the early stages, but such narrowing usually occurs with time. Eventually rupture of the fibrous cap exposes the thrombogenic lipid core to the blood with thrombus formation, vascular occlusion and stroke.

Defects in the vascular endothelium may encourage plaque formation by allowing lipid-laden monocytes to adhere to and access the subendothelium through expression of abnormal cell adhesion molecules. These monocytes subsequently mature and develop into lipid-laden “foam cells”. Smoking, hypertension and diabetes mellitus are all associated with endothelial dysfunction, though the precise mechanisms are still being elucidated. Although larger plaques are correlated with a greater degree of stenosis and greater incidence of stroke, it is not the size of the plaque but rather its composition that determines this; an “unstable plaque” with a thin fibrous cap, little smooth muscle, a high concentration of metalloproteinases and a large lipid core may be at greatest risk of rupture.

**Anatomy**

The Circle of Willis (COW) is the vascular network supplying oxygenated blood to the brain. It is supplied by the internal carotid arteries (“anterior circulation”) and the vertebrobasilar artery (“posterior circulation”). The COW is composed of an anterior communicating artery, and paired anterior cerebral, internal carotid, posterior communicating and posterior cerebral arteries. The anterior cerebral and posterior communicating arteries are branches of the internal carotids, while the posterior cerebral arteries are branches of the basilar artery, itself formed from the union of the paired vertebral arteries.

Several common anatomical variations exist; commonly, the posterior cerebral artery is small and the ipsilateral posterior communicating artery is large, meaning that the “posterior” cerebellar circulation on that side is in fact derived from the internal carotid artery rather than the vertebrobasilar system.

A decrease in blood supply from one tributary (such as the territory of a narrowed carotid artery)
can in theory be compensated for by increased supply from the other arteries feeding the COW. Evidence from SPECT scanning has shown this to be the case in some, but not all patients. Interestingly, the ACST trial showed that patients who underwent CEA suffered significantly fewer strokes in the contralateral anterior circulation than medical controls. This was ascribed to the COW allowing enhanced flow from the “unblocked” carotid to compensate for reduced supply elsewhere. The COW provides the vascular anaesthetist with a means of maintaining brain perfusion by ensuring an adequate driving pressure to the contralateral carotid artery while cross-clamps are in place on the surgical side.

**Surgical conduct of carotid endarterectomy**

The patient is positioned supine with the head extended and rotated to the contralateral side. An incision is made over the anterior border of the sternomastoid muscle from 2 cm below the angle of the mandible to just above its insertion. The muscle is dissected free from surrounding tissue and retracted laterally. The internal jugular vein is mobilised and retracted medially, and the carotid sheath is then opened. The vagus nerve is identified within the sheath, and the common carotid artery (CCA) is exposed and mobilised. The bifurcation, internal and external carotid arteries (ICA, ECA) are then exposed circumferentially. Care is taken to identify and protect the hypoglossal and carotid sinus nerves which lie above the ICA. After heparinisation, vessel clamps are applied to the common, internal and external carotid arteries. At this stage, a shunt may be placed either prophylactically or if compromised cerebral perfusion is suspected. This involves insertion of a hollow conduit into the ICA distal to the plaque, which is allowed to bleed back removing potential air emboli before proximal insertion into the CCA.

Using an operating microscope, an incision is made in the CCA proximal to the plaque and extended into the ICA. Once exposed, the plaque is dissected away from the intima working distally. Any intimal flaps created in this process are trimmed or tacked to the vessel wall with sutures in order to leave a smooth non-atherogenic lumen. The exposed intima is irrigated with heparinised saline, and the arteriotomy is closed with a continuous monofilament suture. A synthetic or venous patch may be incorporated into the arteriotomy closure to prevent narrowing which risks subsequent stenosis and re-occlusion. Prior to complete closure, the clamps are briefly removed in turn allowing debris to be flushed out of the system. Following closure and inspection of the suture line, the clamps are removed in sequence beginning with the ECA followed by the CCA. This allows any remaining debris to be flushed into the ECA rather than entering the cerebral circulation. Finally the ICA clamp is removed, and the superficial layers are closed.

As an alternative to the conventional technique described above, eversion endarterectomy employs a transverse arteriotomy at the level of the carotid bulb. Although evidence from randomised controlled trials suggests a lower rate of re-stenosis, this has not been shown to reduce the incidence of neurological adverse events.

**Pre-operative assessment**

Carotid endarterectomy is associated with significant mortality and morbidity and so the importance of pre-operative decision making stems also from the fact that the operation itself is not a cure and carries with it significant inherent risks. The aim is to reduce the risk of future stroke, but at the same time it will bring much of that long-term risk into a shorter time period. Thus, the decision to operate is a fine balance between the risks of the medically managed vascular disease versus medical management combined with a surgical intervention. Many patients are now being considered for surgery in the weeks following presentation with symptoms. This reduces the time available to investigate all the relevant co-morbidities. Locally agreed peri-operative guidelines may well become more useful in guiding satisfactory pre-operative assessment for vascular anaesthetists and their respective surgical teams in the future. In a pooled analysis of outcome data from three major trials, the 30-day mortality rate was 1.1%. Of these, 20 of 35 deaths were due to perioperative stroke. The combined stroke and death rate at 30 days was 7.1%. The majority of cases of perioperative stroke result from thrombosis or embolism rather than haemodynamic changes induced by the cross-clamping. The second commonest cause of death is from myocardial infarction. In a series of 506 patients scheduled for CEA, coronary angiography revealed severe, correctible coronary artery disease (CAD) in 37% of patients suspected of having CAD, and in 16% thought to have no CAD. Renal impairment, diabetes mellitus, ischaemic heart disease and congestive cardiac failure all increase the
likelihood of a myocardial event in the postoperative period. Although only a relatively small number of patients suffer this complication, it has been shown in a series of 75 patients undergoing CEA that a significant rise in troponin I (TnI) occurs in up to 13% of patients. The consequences following a troponin rise are hard to predict, but there is evidence to suggest that a raised TnI following vascular surgery is associated with a reduction in short and longer term survival. Hence, perioperative optimisation with beta-blockers, anti-platelets and statins are important pre-operative considerations. Assessment should therefore focus on the patient’s cardiac risk, since the presence of carotid artery atheroma is a predictor of ischaemic heart disease, which may be unmasked by the stress of the perioperative period. Uncontrolled hypertension is a risk factor for post-operative neurological dysfunction, and patients with a diastolic blood pressure of >110 mmHg pre-operatively should be deferred until adequate control has been obtained. Identification of high-risk surgical populations can be achieved by using validated scoring indices such as Lee’s revised cardiac risk index. The American College of Cardiology and American Heart Associations’ perioperative cardiovascular guidelines are useful to further risk-stratify and guide clinicians on how best to further evaluate patients. Investigations such as stress echocardiography, myocardial perfusion scans or exercise ECG may be considered subject to local availability and expertise. In addition to cardiopulmonary assessment, the patient’s neurological status should be documented. This allows comparisons to be made in the immediate post-operative period, where any decrease in function may prompt re-exploration. The neurological examination should include motor function, cranial nerve examination and some assessment of speech, swallowing and language as a minimum.

**Conduct of general anaesthesia**

The decision between general and regional anaesthesia depends on anaesthetic and surgical preference in most centres. While local anaesthetic techniques have several theoretical advantages (discussed later), robust evidence does not yet exist to advocate this approach over general anaesthesia as a matter of routine. There are however studies that do suggest a reduction in perioperative complications with regional anaesthesia. The results of the GALA trial (GA versus RA) are eagerly awaited. This trial, which is powered to recruit approximately 5000 patients, hopes with its results to better inform clinicians regarding the best approach to this hotly debated area of practice.

A careful induction should be performed, with attention to the patient’s blood pressure throughout. Placement of an arterial line pre-induction will facilitate this aim. Sufficient opiate should be given to attenuate the pressor response to intubation; the presence of a cuffed endotracheal tube is in the author’s view mandatory given the limited access to the patient’s airway once surgery is underway. Large bore intravenous access should be secured and monitoring should include ECG leads II and V5 to allow detection of myocardial ischaemia with greater sensitivity.

Adequate cerebral perfusion must be maintained to minimise the risk of cerebral ischaemia secondary to a reduction in cerebral perfusion after cross-clamping. This requires normocapnia, adequate arterial oxygenation, near baseline mean arterial pressure (MAP) and adequate venous drainage. Some authorities would advocate the maintenance of a MAP 20% above baseline values to enhance perfusion through the COW during cross-clamping. Some of this evidence comes from case reports that suggest that supra-normal MAP and small increases in cerebral oxygen content can reduce cerebral ischaemia at watershed areas within the brain leading to significant clinical improvements. Given that many of these patients will be elderly smokers with significant ventilation/perfusion mismatch, near patient analysis of arterial blood gas samples will allow accurate calibration of the capnograph against PaCO2. This is helpful because hypocarbia must be avoided due to its deleterious effects on an already compromised cerebral circulation through cerebral vasoconstriction. Hypocarbia may precipitate significant cerebral ischaemia, while hypercarbia may produce cerebral steal and should also be avoided.

Intra-operatively drugs which must be readily available include heparin, given prior to cross-clamping to reduce the incidence of thromboembolic events. A dose of 5000 IU (approximately 100 IU/kg) is usually given as a bolus, but consideration should be given to dosing on a weight basis for patients at extremes of body size. A vasopressor and short-acting beta-blocker should also be immediately to hand. During surgery stimulation of the carotid sinus may precipitate extreme bradycardia or even asystole; the anaesthetist must be vigilant and have a vagolytic drug at the ready. Removal of the surgical stimulus and or application of lidocaine to the carotid sinus are usually very effective manoeuvres. However, the
incidence of post-operative hypotension is more common after this intervention.

Either a volatile or intravenous technique is suitable for maintenance of anaesthesia; the aim is to minimise cerebral metabolic rate (CMRO$_2$) and therefore oxygen demand. Volatile agents with low blood: gas solubility coefficients such as sevoflurane and desflurane offer the advantage of faster emergence times and therefore earlier neurological assessment. Avoidance of long-acting opiates such as morphine and pethidine will also help achieve this goal. Nitrous oxide should be avoided; as well as increasing CMRO$_2$, it presents a risk of worsening any air embolus that becomes entrained into the distal cerebral circulation around the time of cross-clamping.

Normoglycaemia should be maintained, since hyperglycaemia has been associated with a worse neurological outcome in conjunction with cerebral ischaemia. Maintenance of normothermia is also important; while mild hypothermia (33–34°C) may improve outcomes in traumatic brain injury; this is offset by undesirable problems such as post-operative shivering and coagulopathy in elective surgical patients. Hyperthermia should also be avoided, as this will increase CMRO$_2$.

Care should be taken to assess the adequacy of ventilation in the post-operative period, to ensure blood pressure remains within acceptable limits, and to assess the return of neurological function once the patient is able to cooperate. As a minimum standard, motor function, facial symmetry, speech and language function should be assessed. Any new deficit should prompt urgent review by the surgical team who may wish to consider Doppler imaging of the carotid arteries with the option of re-exploration.

The majority of patients can be cared for post-operatively on a vascular surgical ward with appropriately trained staff, although level 2 facilities will occasionally be required for patients with multiple co-morbidities or those who develop post-operative haemodynamic instability that requires intravenous vasoactive infusions. Those patients that go back to a ward should ideally be monitored with invasive arterial monitoring in recovery for 2–3 h post-operatively. The majority of complications such as stroke and wound haematoma occur within 8–12 h of the end of surgery.

**Awake carotid endarterectomy**

Over the previous decade, there has been considerable debate amongst anaesthetists over the best anaesthetic technique for CEA. Nowadays CEA is commonly performed under local anaesthesia. Those who advocate LA point to evidence that supports a reduction in perioperative morbidity and possibly even mortality. Other advantages include early detection of cerebral ischaemia as awake patients can be more readily neurologically assessed, a reduction in shunting requirements and shorter hospital stay. Despite these apparent advantages, there is a lack of high-quality evidence to support regional over general anaesthesia as the technique of choice. It may be the case that GA has compensatory advantages such as better operating conditions, neuroprotection (by reducing cerebral metabolic demand), and reduced patient catecholamine levels which might reduce myocardial ischaemia. A recent Cochrane review analysed 48 trials (7 randomised) and over 25,000 patients, but was unable to demonstrate a convincing safety benefit of LA over GA. Although analysis of the non-randomised trials (the majority of the patients) suggested LA was associated with reduced mortality, myocardial infarction, perioperative stroke and pulmonary complications, this was not supported by the higher quality randomised controlled trials studied. The Europe-wide (GA versus LA) GALA trial has now finished recruiting and should hopefully provide better quality evidence to help inform vascular teams in the not too distant future.

There are many ways to monitor cerebral perfusion (discussed later), but all have fairly poor specificity and sensitivity in detecting inadequate conditions. It is because of this that many surgeons shunt all patients under GA regardless of the monitoring used; with an awake patient adequate perfusion can be readily assessed by changes in speech, cerebration and motor power, particularly at the time of common carotid clamping. A significant change in any of these variables would prompt the insertion of a shunt thus allowing far more selective use of, and potentially minimising the risk attributed to this intervention. Shunts are used in approximately 65% of patients under GA and 10% under LA. Disadvantages of local anaesthesia include the difficulty in managing the airway in an emergency such as late cerebral ischaemia after the endarterectomy has started and the high degree of patient and surgeon co-operation required. There are of course specific complications related to the various regional techniques that must be anticipated. Numerous problems are encountered that need consideration pre-operatively; patients must empty their bladder prior to theatre, judicious use of intravenous fluids, getting the patient into a comfortable position in which they must stay for perhaps up to 3 h. Some patients
find it very claustrophobic and can become restless which is not uncommon in the elderly; this can potentially endanger the patient and staff and clearly sedation is not an easy option. There can be difficulty in providing adequate anaesthesia in those who have a high carotid bifurcation (which will be noted on the pre-operative duplex scan) because heavy retraction on the angle of the mandible is very uncomfortable. These patients invariably need supplemental local anaesthetic from the surgeon. CEA under LA is not an easy option, quite the opposite; it is labour intensive. In the event of intra-operative agitation or airway problems, the anaesthetist and surgeon need to have a plan for managing these difficult situations. It does however give anaesthetists another option, and may be of benefit in patients in whom general anaesthesia is thought to confer an unacceptably high risk, for example those with left ventricular outflow tract conditions such as severe aortic stenosis or hypertrophic cardiomyopathy. Local anaesthetic infiltration, deep or superficial cervical plexus blocks, cervical epidural blockade or a combination of these techniques may be used. Failure rates for cervical plexus blocks are of the order of under 3%, and less than 5% for cervical epidural. However life-threatening complications are more common with cervical epidural. A description of these techniques is provided below.

(i) **Local anaesthetic infiltration:** This requires a motivated patient and surgeon. Local anaesthesia must be used judiciously, with care taken to avoid intravascular injection. Excessive volumes of local anaesthetic may distort the operative field making surgery technically more difficult. Success with only local infiltration is limited and regional techniques are more commonly used with infiltration as an adjunct.

(ii) **Superficial cervical plexus blockade:** The superficial cervical plexus supplies innervation to the skin of the anterolateral neck through the anterior primary rami of C2–C4 (see Figure 1). The conduct of the technique is well described. The patient is positioned supine with head facing slightly away from the side to be blocked. A 22G needle is inserted at the midpoint of the posterior border of the sternocleidomastoid muscle, from where four nerves emerge: the transverse cervical, supracleavicular, greater auricular and lesser occipital nerves (see Figure 2). Local anaesthetic is infiltrated deep to the first fascial layer encountered both caudad and cephalad. A volume of around 10 ml is usually sufficient. Since motor block is not sought, a weak local anaesthetic solution such as bupivacaine 0.25% may be used.

(iii) **Deep cervical plexus blockade:** The deep block has been described by Winnie et al. and by Moore. This can be considered a block of the pre-vertebral space around C2–C4 spinal nerves as they emerge from the vertebral

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**Figure 1** Branches of the superficial cervical plexus.
foramina. The anterior rami of C2–C4 supply the superficial and deep cervical plexuses. The deep cervical plexus innervates the deeper structures including the muscles of the anterior neck. Successful blockade will also provide surface anaesthesia of the neck, occipital and pectoral regions. The patient is positioned as for superficial cervical plexus blockade.

Using Lofstrom’s technique, the transverse process of C6 (Chassaignac’s tubercle) and mastoid process are identified (Figure 3). A line is drawn between the two which should run along the posterior border of the sternocleidomastoid muscle. Points approximately 2, 4 and 6 cm caudally from the mastoid process are marked along this line. These points will correspond to the transverse processes of C2, C3 and C4. After subcutaneous skin infiltration with local anaesthetic, a fine bore needle is inserted at each of these marks in turn, maintaining a slightly caudal angle until transverse process is contacted. It is then withdrawn slightly, and, after negative aspiration, local anaesthetic is injected. Cephalad angulation should be avoided, since this may risk cervical cord injury if the needle is inserted too deeply; the transverse processes rarely lie more than 2.5 cm deep to the skin.
Approximately 4 ml of 0.25% bupivacaine at each level will provide adequate anaesthesia. An alternative approach is to inject a larger volume (10 ml) at the level of the fourth cervical vertebra. Complications of deep cervical plexus blockade (see Table 1) include phrenic nerve palsy, local anaesthetic toxicity (especially if inadvertent vertebral artery injection occurs) and total spinal anaesthesia, and can occur despite an apparently straightforward procedure. It is thus mandatory to have full monitoring, skilled assistance and facilities for intubation and ventilation in the event of complications.

Cervical epidural: This technique has its advocates, but is not practiced much in the UK, mostly due to fears relating to serious potential complications. An epidural catheter can be sited at C7–C8 or the space below. The catheter can be topped up with a dilute local anaesthetic solution such as bupivacaine 0.125% to achieve sufficient anaesthesia for surgery. Many anaesthetists prefer the hanging drop technique to localise the epidural space at this level, since the space is smaller and the “give” with loss of resistance techniques is less pronounced than caudally. Cervical epidural blockade is not without risk of complications. Aside from potential damage to the cervical cord, failure and respiratory embarrassment are potential problems.

A recent retrospective audit of 1828 carotid endarterectomies compared complications of combined superficial and deep cervical plexus block with cervical epidural over a 10-year period. A significantly higher failure rate was shown in the epidural group (6.9% vs. 3%), as well as a higher rate of life-threatening complications (2% vs. 0.3%), leading the authors to conclude that cervical epidural is not the first choice for CEA and should only be practiced by those with experience in the technique. Even then, the complication rate and severity of complications are very prohibitive.

### Cerebral monitoring and protection

The aim of cerebral monitoring in CEA is to determine if clinically significant cerebral ischaemia is occurring. If so, a shunt can be sited to bypass the cross-clamps and hopefully improve cerebral perfusion. The placement of a shunt is not without risk. It may dislodge emboli and cause stroke, or cause injury to the distal internal carotid artery, with increased incidence of late re-stenosis. For CEA under local anaesthesia, clinical monitoring of the patient’s motor power, speech and cerebration is the gold standard. Under general anaesthesia, a variety of surrogate measures have been developed.

### Electroencephalography (EEG)

The EEG signal is affected by cerebral ischaemia and is used by many centres to guide shunt requirements. It is heavily operator-dependent and requires a trained neurophysiologist. It does not reliably detect ischaemia of deeper structures, and in comparison with clinical assessment in awake CEA patients, has been shown to have poor sensitivity. In a recent case series, 13 of 32 patients judged to require a shunt based on clinical assessment showed no evidence of ischaemia by EEG criteria.

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<th>Complications of regional anaesthetic techniques for carotid endarterectomy.</th>
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<td><strong>Superficial cervical plexus block</strong></td>
<td><strong>Deep cervical plexus block</strong></td>
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<td>Intravascular injection (rare)</td>
<td>Injection into vertebral, artery</td>
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<td>High volumes of LA leading to toxicity</td>
<td>Subarachnoid or epidural injection</td>
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<td>Commonly need augmentation of block</td>
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Somatosensory evoked potentials (SSEPs)

Needle electrodes are used to stimulate the contralateral median nerve at the wrist. Sensing electrodes are placed on the scalp overlying the ipsilateral primary somatosensory cortex in the postcentral gyrus. This area of the brain is supplied by the middle cerebral artery, and thus will be affected by reduced flow in the corresponding internal carotid artery. A stimulus lasting 200 ms with a monophasic square wave pulse (usually 20–30 mA) is applied to the median nerve at fixed intervals (typically 3–6 Hz). The latency and amplitude of the evoked response are measured from the sensing electrode. The signal of interest is the cortical N20 peak; a 50% decrease in amplitude or a 10% increase in latency of this are usually taken as reflecting significant ischaemia, and may prompt the insertion of a shunt. Interpretation of these signals is complicated by general anaesthesia, which can in itself affect the signal. Specialised equipment and trained personnel are required. A sample trace is shown in Figure 4.

Stump pressure

Measurement of stump pressure gives an indication of pressure in the COW, and by inference, the adequacy of the cerebral circulation. Following cross-clamping of the common and external carotid arteries, a needle attached to a pressure transducing system is placed either into the common carotid distal to the clamp (into the “stump”) or into the internal carotid. The pressure measured is derived from backflow through the internal carotid artery, and is therefore a reflection of that in the COW. Many levels have been proposed as a threshold for shunt insertion, usually around 40 mmHg is considered appropriate. Although simple to perform, this technique lacks sensitivity. In a recent case series of 314 patients undergoing awake CEA, stump pressure was measured and correlated with clinical evidence of neurological deficit. This showed that although specific (97.4%), a stump pressure of 40 mmHg had poor sensitivity for detecting clinically significant ischaemia (56.8%).

Transcranial Doppler (TCD)

Use of the Doppler probe allows blood flow velocity to be measured during CEA. Bone impedes the passage of ultrasound waves, but various “windows” of thinner bone exist in the skull. The transtemporal window (the petrous part of the temporal bone) allows visualisation of flow in the middle cerebral artery in 90% of patients. Velocity rather than flow is measured unless the cross-sectional area of the artery is known. Assuming this remains constant, trends in velocity will reflect trends in flow, a significant decrease prompting shunt insertion. TCD has been validated as a reliable parameter for judging the effects of carotid cross-clamping on cerebral blood flow, hence it is a useful adjunct in the decision making process with regard to the need for intra-operative shunting. TCD can also be used to detect signals which reflect the formation and dislodgement of microemboli during the dissection phase of the operation. Evidence exists that a high number of microemboli signals ( > 50 h /C0 ) correlates with increased incidence of post-operative neurological dysfunction.

An analysis of 1058 CEA patients monitored with TCD showed a fall in MCA velocity of >90% to be a strong predictor of perioperative stroke, while a fall of >50% had a positive predictive value of only 32% for the detection of neurological dysfunction in a series of 103 patients undergoing awake CEA. Even with an experienced operator, inadequate views are obtained in 10% of patients.

Near-infrared spectroscopy (NIS)

NIS is a measurement of the wavelength and intensity of the absorption of near-infrared light by a sample, and has been used during CEA to influence the decision on intra-operative shunt insertion. Optodes placed on the skin emit light of near-infrared wavelength (800–2500 nm). This light penetrates scalp and brain tissue, and is absorbed to varying degrees by the haemoglobin in the arteries, veins and capillaries, allowing a composite value for tissue oxygenation to be obtained. This can be expressed as a percentage tissue oxygenation index (TOI), or regional cerebrovascular oxygen saturation (rSO2). Normal values are around 60–65%. While it has been demonstrated that cross-clamping lowers rSO2, determining a reliable cut off threshold signifying

![Figure 4 SSEP of left N20 area from a patient undergoing left CEA. Shunt placement causes return of normal signal amplitude lost during cross-clamping (modified from Manninen et al.36).](image-url)
cerebral ischaemia has proved more difficult. In a study of 94 patients having CEA under regional anaesthesia, a fall of 20% from baseline was shown to have a high negative predictive value (97.4%), but a very poor positive predictive value (33.3%). In other words, a patient who does not show a 20% fall in rSO2 is very unlikely to have significant cerebral ischaemia, while a patient displaying such a fall has only a one in three chance of this being the case. A more recent study of 167 patients compared NIS with Doppler and cerebral function monitoring for the detection of ischaemia. A drop of 13% in the TOI was both highly sensitive (100%) and specific (93.2%) for ischaemia as monitored by these control methods.

Post-operative complications

Hypertension may worsen neurological outcome, and can exacerbate the hyperperfusion syndrome. Intra-operative bradycardia is often successfully treated with local anaesthesia to the carotid sinus nerve by the surgeon; however, it commonly causes hypertension in the post-operative period and so is not used prophylactically, but rather only if necessary. Clearly, pain as a causative factor should be excluded in the first instance. Otherwise management with nitrates or beta-blockers is useful to maintain the blood pressure to within 20% of pre-operative levels. Swift management of hypertension in recovery is thus important as it can jeopardise outcome. Hypertension is also implicated in the development of wound haematoma, which, coupled with generalised airway oedema or intracerebral haemorrhage. Symptoms can range from headaches and irritability to fits, coma and death. It tends to present a week after surgery. Carotid body damage, if bilateral, theoretically may cause ventilatory inadequacy in the presence of hypercapnia or hypoxia. Bilateral CEA is rarely performed and so this complication is very uncommon.

Hyperperfusion syndrome is also a rare but serious complication of CEA. It is thought to result from excessive blood flow through brain tissue following restoration of flow, coupled with impaired autoregulation of that area. This may result in cerebral oedema or intracerebral haemorrhage. Symptoms can range from headaches and irritability to fits, coma and death. It tends to present a week after surgery and so most patients have gone home and present to an emergency department. A high index of suspicion should be maintained; if suspected, radiological imaging should be arranged urgently and tight blood pressure control maintained as well as consideration given to the use of osmotic diuretics and steroids to help reduce cerebral oedema.

Carotid stenting

Interest into the use of carotid artery stents (CAS) to manage symptomatic carotid disease has increased in recent years. This is largely due to advances in interventional radiology and because CAS has theoretical advantages. CAS should avoid the need for general anaesthesia, potential cranial...
nerve injury and the complications of wound infection and bleeding. It may have a role in treating those with previous radiotherapy or burns to the neck where a surgical approach would be potentially difficult and dangerous. However, the benefits of CAS compared with surgery do not appear to be substantiated by the current literature in terms of primary outcome measures. A review of randomised trials which compared surgery with CAS failed to show any morbidity or mortality advantage that could warrant a change in practice, and a recent trial was terminated early on grounds of safety due to the hugely increased incidence of stroke in those randomised to stenting. Quite apart from this, there are reports of significant post-operative haemodynamic instability that requires more investigation. This is likely to be due to stimulation of the carotid sinus by the angioplasty balloon, coupled with stent deployment in this area. This leads to increased parasympathetic discharge causing hypotension and/or bradycardia. This can occur almost immediately (in theatre), but a significant number of patients also develop haemodynamic instability up to 8 h after the stent is deployed. A retrospective analysis which looked at nearly 500 patients undergoing carotid stenting reported an incidence of severe hypotension of 7%. These findings were observed during carotid stenting reported an incidence of severe hypotension of 7%. These findings were observed during a significant period of post-operative care.47 Quite apart from this, there are reports of significant post-operative haemodynamic instability that requires more investigation. This is likely to be due to stimulation of the carotid sinus by the angioplasty balloon, coupled with stent deployment in this area. This leads to increased parasympathetic discharge causing hypotension and/or bradycardia. This can occur almost immediately (in theatre), but a significant number of patients also develop haemodynamic instability up to 8 h after the stent is deployed. A retrospective analysis which looked at nearly 500 patients undergoing carotid stenting reported an incidence of severe hypotension of 7%. These findings were observed despite pre-medication with fluids and atropine. It is worthy of note that neurological complications were not significantly increased in those who developed cardiovascular instability. Haemodynamic instability after endarterectomy has a similar incidence; however, most endarterectomy patients are not pre-medicated with atropine. The elderly and those with ischaemic heart disease were those most commonly affected.

Although not common practice at present, anaesthetists may find their vascular colleagues performing CAS in the future if clinical improvements are made with this procedure. Anaesthetic considerations for CAS will depend upon the approach (at present it is usually a cervical or femoral arteriotomy) and upon local practice with regard to the location in which the procedure is carried out. Post-operative admission to critical care units for haemodynamic support may be greater than with endarterectomy and this could therefore have medical and financial implications.

Conclusion

Carotid endarterectomy can bring major public health benefits when patients are selected appropriately. By careful attention to detail in all phases of the procedure, the anaesthetist can make a significant contribution to a successful outcome. While general anaesthesia is currently the mainstay of practice, this may change more in future if high quality evidence demonstrates the superiority of regional anaesthesia. The principles of careful neurological and cardiovascular assessment and the provision of optimum conditions for cerebral perfusion will however, remain unchanged.

References

23. GALA Trial Homepage: <http://www.dcn.ed.ac.uk/gala/>.