Regional anaesthesia for carotid endarterectomy (CEA) became commonplace in the UK in the 1990s, but significant changes in anaesthetic techniques have taken place since the last major review was published.1 Regional anaesthetic techniques have evolved—in particular, the use of nerve stimulators and ultrasound to detect peripheral nerves. This review will focus on the whole process of regional anaesthesia for CEA, including: preoperative assessment and preparation, regional anaesthetic techniques, the choice of sedative and local anaesthetic (LA) drugs, the available evidence comparing regional and general anaesthetic (GA) techniques, perioperative arterial pressure management, and the treatment options for patients developing neurological deficits after carotid cross-clamping.

Methods of achieving regional anaesthesia are detailed, and the authors note the improvements in the equipment available. They note the reduced hospital stay, but the similar rate of stroke, compared with general anaesthesia. Methods of achieving regional anaesthesia are detailed, and the authors note the improvements in the equipment available. The authors review the equipment available. The 2012 National Guidelines for Stroke recommend that carotid intervention for recently symptomatic severe carotid stenosis should be regarded as an emergency procedure in patients who are neurologically stable, and should ideally be performed within 48 h of a transient ischaemic attack or minor stroke and definitely within 1 week,4 as the benefits of carotid surgery decrease rapidly after this. Implementation of these recommendations means that there is less time for preoperative patient preparation, including arterial pressure control, which could predispose to arterial pressure lability. Preoperative hypertension is a risk factor for postoperative stroke and death,2 so patients with uncontrolled hypertension require close attention to perioperative arterial pressure control. Specific figures for preoperative arterial pressure targets have not been defined from controlled trials, but a sensible target is that systolic and diastolic arterial pressures are ≤180 and ≤100 mm Hg, respectively.3

The authors review the equipment available. The 2012 National Guidelines for Stroke recommend that carotid intervention for recently symptomatic severe carotid stenosis should be regarded as an emergency procedure in patients who are neurologically stable, and should ideally be performed within 48 h of a transient ischaemic attack or minor stroke and definitely within 1 week,4 as the benefits of carotid surgery decrease rapidly after this. Implementation of these recommendations means that there is less time for preoperative patient preparation, including arterial pressure control, which could predispose to arterial pressure lability. Preoperative hypertension is a risk factor for postoperative stroke and death,2 so patients with uncontrolled hypertension require close attention to perioperative arterial pressure control. Specific figures for preoperative arterial pressure targets have not been defined from controlled trials, but a sensible target is that systolic and diastolic arterial pressures are ≤180 and ≤100 mm Hg, respectively.3

Summary. Regional anaesthesia is a popular choice for patients undergoing carotid endarterectomy (CEA). Neurological function is easily assessed during carotid cross-clamping; haemodynamic control is predictable; and hospital stay is consistently shorter compared with general anaesthesia (GA). Despite these purported benefits, mortality and stroke rates associated with CEA remain around 5% for both regional anaesthesia and GA. Regional anaesthetic techniques for CEA have improved with improved methods of location of peripheral nerves including nerve stimulators and ultrasound together with a modification in the classification of cervical plexus blocks. There have also been improvements in local anaesthetic, sedative, and arterial pressure-controlling drugs in patients undergoing CEA, together with advances in the management of patients who develop neurological deficit after carotid cross-clamping. In the UK, published national guidelines now require the time between the patient’s presenting neurological event and definitive treatment to 1 week or less. This has implications for the ability of vascular centres to provide specialized vascular anaesthetists familiar with regional anaesthetic techniques for CEA. Providing effective regional anaesthesia for CEA is an important component in the armamentarium of techniques for the vascular anaesthetist in 2014.

Keywords: carotid endarterectomy; outcome; regional anaesthesia

Preoperative assessment

Preoperative hypertension is a risk factor for postoperative stroke and death,2 so patients with uncontrolled hypertension require close attention to perioperative arterial pressure control. Specific figures for preoperative arterial pressure targets have not been defined from controlled trials, but a sensible target is that systolic and diastolic arterial pressures are ≤180 and ≤100 mm Hg, respectively.3

The 2012 National Guidelines for Stroke recommend that carotid intervention for recently symptomatic severe carotid stenosis should be regarded as an emergency procedure in patients who are neurologically stable, and should ideally be performed within 48 h of a transient ischaemic attack or minor stroke and definitely within 1 week,4 as the benefits of carotid surgery decrease rapidly after this. Implementation of these recommendations means that there is less time for preoperative patient preparation, including arterial pressure control, which could predispose to arterial pressure lability.

An estimation of the patient’s ‘normal’ arterial pressure should be obtained from several sources including the clinic visit, the preoperative assessment clinic, and the anaesthetic room—this is the minimum arterial pressure accepted during
the period of carotid cross-clamping. Arterial pressure should be measured in both arms using the correct techniques described by the British Hypertension Society.

The patient’s neurological status should be assessed before operation, and neurological deficit(s) documented, as differences detected in the postoperative period potentially require surgical re-exploration. It is worthwhile noting the patient’s presenting neurological complaint, for example, amaurosis fugax, dysphasia, etc. If the patient develops a neurological deficit when the carotid cross-clamp is applied, they commonly present with the same symptoms that they first presented with (M.D.S., unpublished observations).

Anti-hypertensive medications should usually be continued except for angiotensin-converting enzyme inhibitors and angiotensin II receptor antagonists, but reductions in arterial pressure should be avoided in patients with neurological symptoms. β-Blockers protect against perioperative cardiac complications in patients with a history of myocardial ischaemia, but this is offset by an increased risk of stroke in higher dose regimes and in patients who become acutely anaemic (although acute anaemia is rare in patients undergoing CEA). The American College of Cardiology recommendations for perioperative β-block include continuation if already taking but not to start unless specifically indicated. Metoprolol is associated with increased risk of stroke in patients undergoing non-cardiac surgery compared with other selective drugs such as atenolol or esmolol and should probably be avoided. Statins should be continued as there is evidence of up to a 3% reduction in the incidence of stroke after CEA.

Antiplatelet therapy has a theoretical, therapeutic benefit both at the thrombogenic endarterectomy site and in the coronary circulation in high-risk vascular patients. Aspirin is recommended for all vascular patients in the perioperative period, but the situation regarding clopidogrel is less clear. There is certainly evidence in patients undergoing CEA of the benefits of dual antiplatelet therapy (aspirin combined with low-dose clopidogrel) to reduce the rate of micro-embolization after operation, and a Cochrane review of randomized trials found established observations.

Neither the incidence of clinically important neck haematoma nor the morbidity rate from haemorrhagic complications increases in patients undergoing CEA taking clopidogrel with or without aspirin. Surgery may take longer; however, careful consideration should be given to the risks and benefits of performing regional anaesthesia in these patients. There is no evidence available in the literature on the safety of performing CEA under GA or regional anaesthesia techniques in patients receiving newer, faster onset and offset antiplatelet drugs such as ticagrelor or prasugrel or any other of the newer oral anticoagulants.

**Regional anaesthetic techniques**

**Key anatomy**

The cervical plexus is formed by the ventral rami of the first four cervical nerves (C1–4). The nerves pass laterally along the corresponding transverse process immediately posterior to the vertebral artery and vein. The deep branches are entirely motor and supply the neck muscles. The superficial branches are sensory and supply the skin and subcutaneous tissues of the neck and posterior aspect of the head.

It is possible for the anaesthetist or the surgeon to anaesthetize the tissues layer by layer using large volumes of dilute LA along the line of incision, and thereafter at each dissection plane and finally into the carotid sheath. This represents the simplest and, by default, rescue technique for a less than adequate block or when procedures are to be done by anaesthetists less familiar with nerve blocks. However, most clinicians choose to utilize a formal regional anaesthetic technique.

Cervical epidural anaesthesia can provide suitable conditions for carotid surgery. An epidural catheter is sited at C6–7 and a dilute anaesthetic solution such as bupivacaine 0.25% injected. However, bilateral cervical and upper thoracic nerve roots are affected resulting in significant side-effects, including hypotension, bradycardia, and respiratory impairment. Other complications include conversion to GA, dural tap, epidural haematoma, and direct spinal cord damage. Although epidural anaesthesia is used infrequently in the UK, it may be useful for more extensive procedures, for example, carotid cross-overs and combined carotid–subclavian reconstructions. An epidural catheter may be ‘topped up’ for prolonged procedures, although this has also been described with deep cervical block.

Single injection posterior cervical paravertebral block at the C4 level using a nerve stimulator is another technique of blocking C2–4 dermatomes, which may reduce the risk of accidental vascular injury or injection.

**Superficial cervical plexus block**

The technique for this block has been described previously. Superficial block is performed superficial to the investing layer of deep cervical fascia. Large volumes, for example, 20–30 ml of LA agents, are typically used. An oft-cited disadvantage of superficial compared with deep block is said to be that it does not provide neck muscle relaxation, although this has not been shown to be important clinically.

**Deep cervical plexus block**

This block is performed as a single (C3 or C4–5) or multiple injection technique (C2, C3, C4). With the patient supine and the head turned towards the opposite side and slightly up, the cervical transverse processes are palpated behind sternocleidomastoid. After skin disinfection and intradermal infusion with lidocaine, a 50 mm, 25 G block needle is introduced aiming slightly caudally and posteriorly until the cervical transverse process is encountered, usually 1–2 cm under the skin.

Single-injection blocks cause less pain during the block, although the number of injections does not appear to affect overall block efficacy. Single injections may be associated with less systemic absorption of LA. The deep block has been implicated with a higher risk of accidental involvement of deep structures, such as the carotid and vertebral arteries, the phrenic nerve, dura mater, and the sympathetic trunk.
Continuous deep block has been described and may be considered in high-risk cases in which the procedure may be prolonged. An 18 G Tuohy needle is directed towards the C3 transverse cervical process using 1 mA current. Once paraesthesia is encountered in the anterior region of the neck, a 20 G catheter is inserted and secured at 4 cm depth (catheter tip to skin distance).22

Intermediate cervical plexus block refers to an injection of LA in the space between the superficial and deep cervical fascia25 (although the existence of the deep fascia has been disputed).29 Intermediate block should theoretically reduce complications of deep block such as intrathecal or intra-arterial injection, although there is no evidence supporting this. The technique is straightforward. A needle is inserted perpendicularly to the skin midpoint and posteriorly to the sternocleidomastoid to a depth of 15 mm, just below the superficial cervical fascia. This depth guide may not be valid in obese patients.30 There may be a perception of ‘loss of resistance’. The efficacy of superficial (subcutaneous) and intermediate cervical blocks appears similar.31

Combined superficial and deep cervical plexus block is commonly practiced and consists of a deep injection plus superficial or intermediate block.32 Superficial and combined blocks appear to be equally efficacious.24 32

Nerve stimulators may be used to identify the deep cervical plexus. A short-bevelled, 50 mm block needle connected to a nerve stimulator is inserted perpendicularly to the skin consecutively at C2, C3, and C4. A current of 0.5 mA may elicit neck muscle contractions; ipsilateral head twitch;28 elevation and internal rotation of the scapula;27 paraesthesia over the upper arm, shoulder;33 neck; or paraesthesia radiating up to the ear. By identifying diaphragmatic muscle response, a nerve stimulator may avoid administration of the LA directly onto the phrenic nerve, thereby avoiding phrenic nerve palsy.34

Additional nerve blocks have also been described to supplement cervical plexus block. Submandibular and referred dental pain commonly occur, which may not respond to administration of supplemental LA.35 This pain may be ameliorated by anesthetizing the mandibular division of the trigeminal nerve via the intraoral approach.36 This may be useful in patients with short necks, where there is a high carotid bifurcation or where the atheromatous plaque extends cranially in the internal carotid artery.37

Ultrasound-guided superficial cervical plexus block

Ultrasound can demonstrate the cutaneous branches of the superficial cervical plexus and their relation to the surrounding anatomy. The advantages over the landmark technique are the ability to visualize the spread of LA in the correct plane and to avoid inadvertent damage to, or accidental puncture of, neighbouring structures. However, ultrasound guidance has not been shown to improve the success of superficial cervical plexus blocks.38

The superficial cervical plexus lies laterally to the posterior border of the sternocleidomastoid muscle. It can be visualized as a linear streak of hypoechoic nodules, which have a honeycomb appearance, below the prevertebral fascia and immediately above the interscalene groove (Fig. 1).

Using aseptic technique, a high-resolution linear transducer is placed transversely over the midpoint of the sternocleidomastoid muscle (usually at the level of the cricoid cartilage, where the external jugular vein crosses sternocleidomastoid). With an in-plane or out-of-plane technique, a 50 mm nerve block needle is advanced adjacent to the superficial cervical plexus in the plane deep to sternocleidomastoid, underneath the prevertebral fascia and immediately above the interscalene groove (Fig. 2). After negative aspiration, 10–15 ml of LA is placed in this fascial plane while visualizing the spread of the LA with ultrasound. High concentrations of LA agent are not required since the superficial cervical plexus comprises purely sensory nerves.39
Ultrasound-guided deep cervical plexus block
The transverse processes are easily visualized subcutaneously with ultrasound. The vertebral artery makes a prominent loop between C1 and C2, which may be visualized and is an accurate landmark for the transverse process of C2. The C2 spinal nerve lies inferior and posteriorly to the vertebral artery at this level. The vertebral artery is most susceptible to inadvertent puncture at this point but avoided by visualization with ultrasound.

The C3 and C4 spinal nerves exit in the gutters of the transverse processes and posterior to the vertebral artery. Vertebral artery injection with the needle positioned over the C3 and C4 transverse processes is virtually impossible. The neck is scanned from the mastoid process to Chassaignac's tubercle allowing identification of the relevant anatomy. The transverse processes appear as a hyperechoic formation with posterior acoustic dropout on ultrasound, which is lost when the probe is moved further caudally. With strict asepsis under ultrasound guidance, a 50 mm nerve block needle is advanced until contact is made with the transverse process (Fig. 3). After negative aspiration, 5 ml of LA is injected, which may be repeated at each level.40

Ultrasound-guided intermediate cervical plexus block
Fifteen to 20 ml of ropivacaine is deposited, with ultrasound guidance, in the fascial band between the sternocleidomastoid and levator scapulae muscle.61 This is effective when combined with a perivascular carotid sheath block using 3–5 ml 0.5% ropivacaine and requires little LA supplementation during carotid surgery.42

Pharmacology of cervical plexus blocks
Most LA agents have been used for cervical plexus block. Ropivacaine and levobupivacaine are less potent than bupivacaine, but have a safer profile with regard to LA toxicity.72 Ropivacaine has the advantage of offering longer postoperative pain relief compared with mepivacaine64 and causing less vasodilatation than levobupivacaine.45 LA supplementation by the surgeon in the form of lidocaine 0.5% or 1% is commonly required. Adjuncts to LA agents which may be used to improve the block or reduce toxicity are shown in Table 1.

Assessment of the block
The loss of pinprick sensation in the distribution of the C2–4 dermatomes, together with complete sensory loss46 or altered temperature sensation,47 may be used to assess the effectiveness of the block—which should extend over the shoulder and down to the clavicle (C4 dermatome), and up to and including the neck up to the earlobe (C2). On occasions, the block may spread down to C5 and other lower cervical roots50 after which the patient may complain of transient ipsilateral arm weakness, thus differentiating it from an intraoperative neurological event that would be contralateral.

Intraoperative management
Good communication at all times is important for patient reassurance and cooperation. The patient is positioned in a ‘deck-chair’ position with the head on a soft head-ring, extended (if tolerated), and rotated contralaterally. Patient comfort is vital to allow them to tolerate 2–3 h in this position. Other potential comfort measures include: the use of a non-heated mattress, padding and support under all pressure areas, a pillow placed under the patient’s knees, ensuring the patient’s bladder is emptied before the operation, and keeping surgical drapes off the patient’s face. Clear plastic drapes may be used to reduce the sensation of claustrophobia. Blood loss is rarely a problem, so fluids should be kept to a minimum to avoid the need to void intraoperatively. Water (e.g. from a 20 ml water for injection ampoule) may be administered to ‘wet the lips’ during the operation. Avoid allowing the patient to drink, however, as the combination of lying supine, the possibility of recurrent laryngeal nerve block, and residual sedation effects make accidental aspiration of water possible, resulting in choking, coughing, hypertension, and potential venous bleeding.

Oxygen is administered via a Hudson facemask continuously throughout the operation. Perioperative monitoring should be placed before sedation and performance of the regional
Carotid cross-clamping

The period of carotid cross-clamping may be up to or even longer than 1 h, although there is considerable variation. Cerebral perfusion is most at risk during this period. Under GA, augmentation of arterial pressure to maintain cerebral perfusion is used by some, and augmentation of arterial pressure to 20% above baseline has been recently shown to reduce early postoperative cognitive dysfunction; however, this can precipitate myocardial ischaemia so must be done cautiously. In patients with poor cardiac function or cardiac ischaemia, more invasive haemodynamic monitoring may be considered.

Sedation

Sedation is commonly administered to supplement the regional block during awake CEA, as inadequate analgesia or anxiety may increase the stress response and could precipitate adverse cardiovascular effects. Judicious use of conscious sedation (communication maintained with the patient at all times) ensures patient comfort throughout the procedure while minimizing risks.

Several classes of sedative agents have been used including: opioids (remifentanil, fentanyl), α2-agonists (clonidine, dexmedetomidine), propofol, and, historically, butyrophenones (droperidol, haloperidol). The ideal sedative agent should reduce anxiety without causing respiratory depression, airway compromise, or haemodynamic instability, while the depth of sedation is altered rapidly.

Propofol target-controlled infusion produces effective, easily controlled sedation, easily titrated to an optimum level, while rapidly reducible to allow neurological monitoring during carotid cross-clamping.

α2-agonists are ideal drugs as in addition to sedation, they reduce analgesic requirements and are hypotensive agents with cardioprotective properties. Clonidine (1 μg kg−1 loading..
dose plus 1 μg kg⁻¹ h⁻¹ infusion) has been safely used in patients undergoing CEA without impairment of neurological monitoring, while decreasing the incidence of postoperative pain, hypertension, and neurological complications.71

Dexmedetomidine is licensed in the USA as a sedative. It produces easily rousable sedation with no respiratory depression, reduces opioid administration,72 and has a short half-life allowing rapid titration to effect.71 Compared with other conventional sedatives, dexmedetomidine is associated with less intraoperative and immediate postoperative hypertension, fewer interventions for the treatment for hypertension,74 and less pain after operation.75 Suitable doses of dexmedetomidine for sedation during CEA are 0.2 μg kg⁻¹ bolus followed by 0.2 μg kg⁻¹ h⁻¹ infusion.72

Remifentanil is the preferred sedative agent of the authors. It causes hypventilation and therefore may cause hypercarbia, but this effect is minimal if the dose is titrated to effect.69 Remifentanil at doses up to 3 μg kg⁻¹ h⁻¹ produces a rapidly reversible and predictable sedo-analgesia while at the same time reducing LA supplementation.76

Sedation must be minimal during cross-clamping to allow frequent neurological assessment. It is the authors’ practice to turn off the remifentanil sedation when the surgeon asks for heparin to be administered before cross-clamping, such that the patients are fully conscious when the period of carotid cross-clamping commences.

Haemodynamic management3

Patients who require perioperative pharmacological treatment for hypotension and hypertension have >1 yr morbidity and mortality.77 The cardiovascular ‘profiles’ associated with carotid surgery performed under GA and regional anaesthesia differ considerably. Under GA, patients tend to be relatively hypotensive intraoperatively, commonly requiring vasopressor support.53 After operation, GA patients tend to be hypertensive, due to emergence from anaesthesia and, perhaps, in pain. On the other hand, patients under regional anaesthesia are often relatively hypertensive intraoperatively, particularly during the cross-clamp period, but then relatively hypotensive after operation. These observations may be explained by cerebral autoregulation, which is probably still functioning in awake patients.

Arterial pressure during carotid cross-clamping should not be allowed to decrease below the patient’s ‘baseline’ and should be kept at or up to 20% above this. However, after carotid artery unclamping, hypertension should be avoided to reduce the likelihood of hyperperfusion syndrome.78 The choice of specific vasoactive drugs and reported thresholds for arterial pressure control depends on a variety of factors that include the patient’s history, comorbidity, underlying heart rate, and drug history.

Metaraminol, ephedrine, and phenylephrine are commonly used in the UK to augment arterial pressure. A wide variety of drugs including hydralazine, glyceryl trinitrate (GTN), calcium channel antagonists, β-blockers (labetalol, esmolol, atenolol), and α-2 agonists, such as clonidine, may be used to treat perioperative hypertension. Metoprolol should probably be avoided, due to pharmacogenetic variation in metabolism8 and the associated increased incidence of perioperative stroke.12 Calcium channel blockers and vasodilators may be beneficial in patients with ischaemic heart disease. GTN can increase cerebral perfusion pressure, despite a decrease in mean arterial pressure79 and nifedipine has caused precipitous decreases in arterial pressure80-81 and should be avoided in the acute management of hypertension in patients undergoing CEA.

Complications of regional anaesthesia

Seizures occurring during CEA can be life-threatening, because of possible airway compromise and cerebral ischaemia due to increased cerebral oxygen consumption.82 The cause of seizures during CEA is multifactorial and includes: LA overdose, direct injection of LA into the artery (either by the surgeon82 or by the anaesthetist,83) or cerebral ischaemia. After operation, seizures may be a manifestation of hyperperfusion syndrome,78 which generally requires aggressive anti-hypertensive treatment. Seizures occurring intraoperatively due to intravascular injection of LA may be short in duration, with rapid subsequent recovery82 and do not necessarily preclude safely completing the operation.

Cervical block complications

More complications have been reported in the literature from patients undergoing deep and combined deep and superficial cervical plexus block compared with superficial block alone. In a systematic review of complications reported from 69 papers describing a total of 7558 deep/combined blocks and 2533 superficial/intermediate blocks reported in the literature,84 deep/combined block was associated with a higher complication rate compared with the superficial/intermediate block (odds ratio 2.13, P = 0.006). The conversion rate to GA was also higher with patients receiving the deep/combined block (odds ratio 5.15, P < 0.0001). Only two randomized controlled trials were included in the review and none of the studies specifically compared safety aspects of different cervical plexus blocks, so while this review illustrates the potential dangers of deep block, it would be imprudent to abandon the deep block without formal evidence of increased risk. In addition, performing regional blocks using ultrasound has been shown to be safer in terms of less LA toxicity85 and higher success rates than nerve-stimulator-guided blocks,86 87 but this has not yet been shown for cervical plexus blocks.

Cardiovascular complications

Myocardial infarction is a major cause of perioperative and long-term mortality after CEA. Only nine out of 3523 (0.25%) patients in the GALA trial suffered myocardial infarction, with no difference between the GA and LA groups.88 Cardiac complications are relatively common in arteriopaths presenting for any type of surgery, so the development of cardiac complications after administration of cervical plexus block may not necessarily imply a causal relationship.89 Accidental surgical manipulation of the vagus nerve, which lies within the carotid sheath, can lead to profound
haemodynamic disturbance, including nausea and vomiting, bradycardia, hypotension, and even cardiovascular collapse. Treatment involves alerting the surgeons to their potential vagal stimulation, less retraction, and administration of anti-cholinergic drugs such as atropine.

**Airway complications**

Respiratory distress, secondary to diaphragmatic or vocal cord paralysis, may occur. Phrenic nerve block is common after deep cervical block occurring in up to half of the patients, although this is commonly tolerated without major sequelae. However, in patients with unrecognized contralateral phrenic/recurrent laryngeal nerve damage, due to previous cardiac or neck surgery, cervical plexus block may result in respiratory distress or obstruction. Preoperative examination of vocal cords in patients at risk of contralateral nerve damage has been recommended.

Airway compromise after CEA may be life-threatening. Airway oedema is demonstrable in all patients undergoing CEA and may be due to local trauma and interference with venous and lymphatic drainage. Haematoma formation (due to arteriotomy dehiscence or wound haematoma) further compresses the airway anatomy.

It may be possible to return the patient to theatre and open the wound with the LA block still working, thus avoiding the need for emergent control of the airway. However, when required, various airway techniques have been described including fibreoptic intubation, direct laryngoscopy using LA with or without mild sedation before induction of anaesthesia, laryngeal mask airway insertion, and tracheostomy. Visualization of the glottis with a fibreoptic scope can itself be difficult due to venous congestion and distortion of the anatomy. In cases of poor direct visualization of the glottis, either before or after induction, decompression of the airway by opening the surgical incision in recovery may facilitate intubation of the trachea, even if a previous fibreoptic intubation attempt has failed. In the presence of acute neck swelling, changes in the voice such as hoarseness are considered harbingers of impending airway obstruction and managed accordingly with appropriate anaesthetic and surgical levels of expertise. Any patient who has been reintubated for airway compromise or surgical evacuation of haematoma will usually require overnight elective ventilation to allow the airway oedema to settle.

Pain may be caused by inadequate regional anaesthesia, variable afferent sympathetic nerve supply to the carotid artery and sheath, and may occur in patients with a high carotid bifurcation where the surgical field is supplied by cranial rather than spinal nerves. Supplementation of the block is easily achieved directly by judicial LA infiltration by the surgeon. The authors have used lidocaine spray (Xylocaine 10% Pump spray, AstraZeneca, Luton, UK), sprayed directly onto the carotid artery or sheath to effect immediate pain relief in particular for referred pain from the carotid sheath.

Intra-arterial injection of LA (despite negative aspiration test), subarachnoid injection (resulting in brain stem anaesthesia), and anaesthesia of the recurrent laryngeal, vagus, hypoglossal, and phrenic nerve have all been described with deep cervical plexus block.

Conversion to GA is rarely required. An incidence of 2.5% is quoted and may result from inadequate anaesthesia, patient agitation, poor patient compliance, severe respiratory compromise, or accidental intravascular injection. Deep combined block has historically been more commonly associated with conversion to GA than superficial block. If conversion to GA is required, this may usually be accomplished by administration of suitable analgesics (e.g. remifentanil) and propofol followed by airway control with a laryngeal mask airway and subsequent ventilation if required. This may arguably be safer and a better plan than a rapid sequence induction due to difficulties with intubation and potential haemodynamic consequences of intubation.

Nerve injuries can occur as a result of the regional block or the surgery itself and may be asymptomatic. The nerves at risk of injury are the marginal mandibular branch of the facial, laryngeal, accessory, hypoglossal, the sympathetic (Horner’s syndrome), and the radial nerve. The cutaneous sensory nerves supplying the second, third, and fourth dermatomes can also be damaged. Most cranial nerve injuries probably result from stretching, retraction, clamping, or improper use of diathermy and resolve within 4 months. Dexamethasone has been shown to be effective at decreasing the incidence of temporary post-CEA cranial nerve dysfunction. Facial nerve block has also been described after superficial cervical plexus block and must be distinguished from a cerebrovascular event.

**Regional anaesthesia vs GA**

Proponents of GA cite neurological protection afforded by thiopental and volatile anaesthetic agents; absolute peri-operative control of ventilation (allowing control of arterial carbon dioxide concentration and its effects on the cerebral vasculature); and finally, the individual preferences of surgeon, anaesthetist, and patient.

The main advantage of regional anaesthesia is the ease of assessment of neurological status during carotid cross-clamping compared with the alternatives available under GA, such as TCD, somatosensory-evoked potentials, processed EEG, and near-infrared spectroscopy. Other benefits of regional techniques include: immediate postoperative neurological assessment, greater cardiovascular stability, better postoperative analgesia, and shorter hospital stay (with associated financial benefits).

The GALA trial (a randomized comparison of GA and LA for patients undergoing CEA) was devised to attempt to determine whether GA or regional anaesthesia was the safer technique. GALA is the largest ever published comparison of two anaesthetic techniques. Over 8 yr, 3526 patients were recruited and randomized to receive GA or regional anaesthesia for CEA. A primary outcome (myocardial infarction, stroke, or...
death within 30 days of surgery) occurred in 4.8% patients assigned to GA and 4.5% of those assigned to LA.

Despite the equivocal results of GALA, the debate as to which anaesthetic technique is associated with the best outcome has not abated. GALA was criticized for being underpowered, despite the large number of patients recruited. Furthermore, GALA trial critics (mostly proponents of regional anaesthesia) cite the wide variability in trial methodology which allowed any GA technique to be compared with any regional anaesthesia technique; the slow recruitment and changing clinical practice over 8 yr; and the wide variability in practice between participating countries. On the other hand, proponents of GA have used GALA trial results to validate their practice.

Since the GALA trial, several other papers have been published showing further subtle differences between GA and regional anaesthesia for CEA. The GALA collaborators published a study showing that patients receiving GA had higher jugular venous concentrations of a marker of cerebral ischaemia (neuronal-specific enolase) compared with those patients receiving LA.

One possible explanation for the equivocal results of GALA is that the choice of anaesthetic technique used is less important than the vascular ‘team’ looking after the patient. Clinical staff who are used to working with each other use protocol-driven methods and techniques to achieve good clinical results.

There are large published case series attesting to the relative safety of both GA and regional anaesthetic techniques with 30 day mortality rates considerably less than the accepted ‘normal’ rates. These case series support this concept of a team approach to major vascular surgery.

Conclusions
Regional anaesthesia for carotid surgery has evolved over the last 15 yr with new regional techniques, novel methods of locating the cervical plexus, new drugs, and better management of the patient during the carotid cross-clamping. While regional anaesthesia has not been shown to be associated with better outcome than GA, there are differences in, including in haemodynamic stability, the ease of neurological monitoring and hospital stay. Our challenges for the future will include: management of the ‘urgent carotid’, optimizing arterial pressure control, and developing clinical protocols to avoid perioperative complications such that overall perioperative morbidity and mortality may be reduced.

Authors’ contributions
M.D.S. had the idea for this review article and wrote the second and subsequent drafts. D.S. did the literature review and wrote a first draft. J.M. obtained the ultrasound images of the cervical plexus and wrote the section on ultrasound-guided cervical plexus blocks.

Declaration of interest
None declared.

Funding
No funding was required for this review article.

References
1 Stoneham MD, Knighton JD. Regional anaesthesia for carotid endarterectomy. Br J Anaesth 1999; 82: 910–9


Pandit JJ, Dorje P, Satya-Krishna R. Investing layer of the cervical fascia of the neck may not exist. Anesthesiology 2006; 104: 1344


Foster RH, Markham A. Levobupivacaine: a review of its pharmacology and use as a local anaesthetic. Drugs 2000; 59: 551–79


56 Stoneham MD, Warner O. Blood pressure manipulation during awake carotid surgery to reverse neurological deficit after carotid cross-clamping. Br J Anaesth 2001; 87: 641–4

57 Stoneham MD, Martin T. Increased oxygen administration during awake carotid surgery can reverse neurological deficit following carotid cross-clamping. Br J Anaesth 2005; 94: 582–5


68 Pennekamp CW, Moll FL, de Borst GJ. The potential benefits and the role of cerebral monitoring in carotid endarterectomy. Curr Opin Anesthesiol 2011; 24: 693–7


82 Stoneham MD, Bree SE. Epileptic seizure during awake carotid endarterectomy. Anesth Analg 1999; 89: 885–6


108 Wijeyaratne SM, Collins MA, Barth JH, Gough MJ. Jugular venous neurone specific enolase (NSE) increases following carotid endarterectomy under general, but not local, anaesthesia. *Eur J Vasc Endovasc Surg* 2009; 38: 262–6


Handling editor: J. G. Hardman