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Anesthesiology Clin N Am
22 (2004) 265–287

ANESTHESIOLOGY
CLINICS OF
NORTH AMERICA

Intraoperative management: carotid endarterectomies

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The management of anesthesia for patients undergoing carotid endarterectomy (CEA) is challenging and dynamic. Effective management and good outcome requires the anesthesiologist's understanding of cerebral physiology, knowledge of neck anatomy, and understanding of the rapid pathophysiologic changes that occur during carotid artery manipulations. The anesthesiologist must be flexible in the management of patients, who frequently have underlying multiorgan pathology and cardiovascular compromise. Good communication between the anesthetic and surgical teams is needed to avoid irreversible debilitating consequences for the patient.

Anatomy

Two carotid and two vertebral arteries provide the arterial circulation to the brain. All four arteries are interconnected by the circle of Willis on the base of the brain (Fig. 1). In addition, collateral connections between the respective vascular beds exist throughout the cortex. If circulation through one of the carotid arteries is critically diminished or stopped by disease or by surgical manipulations, blood flow through the circle of Willis maintains perfusion in affected cerebral zones by providing adequate blood and oxygen supply from the other three arteries. Therefore, intraoperative surgical occlusion of the carotid artery will result in ischemia and necrosis if the collateral blood supply is not adequate.

Surgery and local anesthetics can cause temporary or permanent impairment of several nerves, including the vagus nerve, the recurrent laryngeal nerve, the glossopharyngeal nerve, and the phrenic nerve. The vagus nerve usually lies just lateral to the internal and common carotid arteries. The vagus can be injured from surgical manipulation with forceps or retractors or from scalpels or cautery. The recurrent laryngeal nerve branches from the vagus inside the mediastinum and

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0889-8537/04/$ – see front matter
doi:10.1016/j.atc.2004.01.001

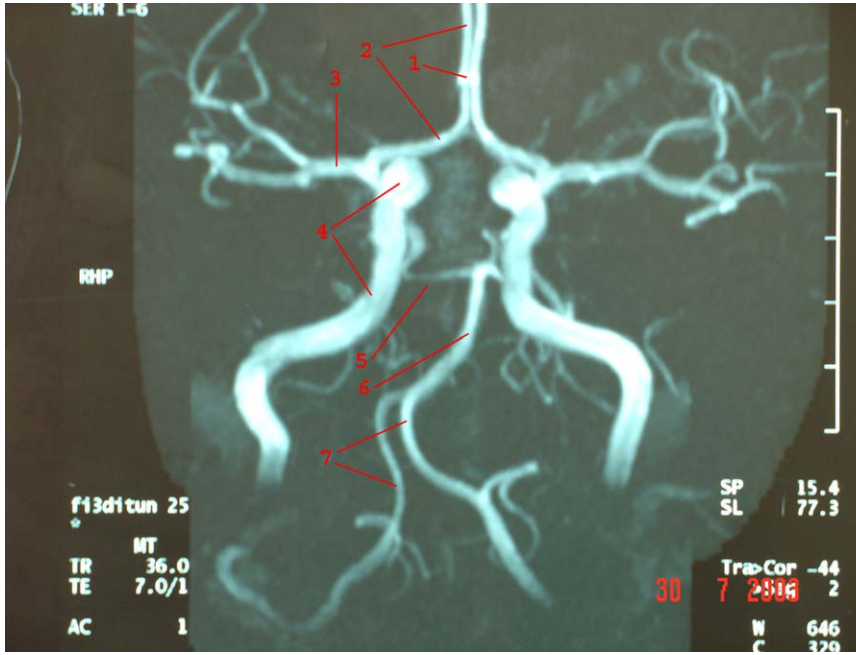

Fig. 1. MRI scan of the circle of Willis. (1) Anterior communicating artery. (2) Anterior cerebral artery. (3) Middle cerebral artery. (4) Internal carotid artery. (5) Posterior communicating artery. (6) Basilar artery. (7) Vertebral arteries.

returns into the neck posterior to the thyroid. It can, at times, originate above the thoracic inlet and lie next to the common carotid artery or carotid bifurcation. Unilateral injury to the vagus or recurrent laryngeal nerve results in paralysis of the ipsilateral vocal cord, which leads to an inadequate cough. Inability to eliminate supraglottic secretions and hoarseness can lead to respiratory and psychological distress, which, if secondary to the effects of local anesthetics, is relatively short-lived.

The glossopharyngeal nerve crosses the carotid artery and can sometimes sustain blunt or sharp surgical injury during dissection. This nerve is responsible for the function of the middle pharyngeal constrictor muscle, which regulates the swallowing mechanism. Unilateral injury of the glossopharyngeal nerve results in an inability to swallow. Significant supraglottic accumulation of the oral and nasal secretions predisposes the patient to potential pulmonary aspiration and respiratory distress.

The phrenic nerve originates from C3 to C5 roots and lies anterior to the scalenus muscle and anterolaterally to the internal jugular vein under the prevertebral fascia. Unilateral phrenic nerve injury or anesthesia results in loss of ipsilateral function of the diaphragm that can potentially lead to respiratory insufficiency, particularly in patients who have preexisting chronic respiratory disease.

Pathophysiology

During CEA, clamping of the carotid artery is followed by incision and opening of the artery with stripping of the diseased intima. It is important to keep in mind that type I and type II baroreceptors located throughout the adventitia and its border with the media are affected by crossclamping. When the clamp has been applied and the artery incised, the pressure registered by these receptors becomes equal to atmospheric. Afferent alarm signals of low pressure then travel by way of myelinated A-type and unmyelinated C-type fibers of the glossopharyngeal nerve to the nucleus tractus solitarius, triggering a central systemic pressure–response. Afferent output further increases following surgical "stripping" of the atherosclerotic rigid intima and exposure of internal elastic lamina with underlying, now "sensitive" media.

Carotid chemoreceptors are also affected. Clamping of the carotid artery leads to a rapid drop in oxygen tension, which is registered by these cells located in the carotid body. Afferent signals then travel by way of the glossopharyngeal nerve into the same area of the nucleus tractus solitarius, activating descending sympathetic pathways. Systemic pressure response is therefore further augmented by the afferent output from these receptors. Augmented autonomic efferent output can lead to onset of tachycardia, severe arterial hypertension, and an increase in afterload and myocardial oxygen demand.

Surgery

Two skin incision techniques are employed most frequently for this surgery. The more frequent approach is to run the incision along the anterior border of sternocleidomastoid muscle (between the mastoid process and sternoclavicular junction). The second option is to cut obliquely starting on the mastoid process, overlapping the projection of the carotid bifurcation and continuing horizontally across the neck. Subsequent dissection of the neck tissues allows the surgeon to mobilize and retract the sternocleidomastoid muscle to expose the carotid sheath. Rigorous surgical retraction of the sternocleidomastoid muscle can cause discomfort in an awake patient, which can be alleviated by injection of 5 to 6 mL of local anesthetic into the muscle sheath.

When the carotid sheath has been opened, the vagus nerve can frequently be seen, usually positioned posterior to the artery. The common facial vein is then ligated at the crossing point with internal carotid artery. Ligatures are placed around the common carotid artery and the internal and external carotid arteries (Fig. 2). Retraction of the hypoglossal nerve that crosses the artery at the bulb level is frequently required, which can cause nerve damage.

The tongue deviates toward the operative side when extruded from the mouth. Dissection and mobilization of the carotid artery near the bifurcation frequently results in bradycardia, usually caused by vagal stimulation. Bradycardia can be prevented or treated by infiltration around the carotid bulb with a local anesthetic.

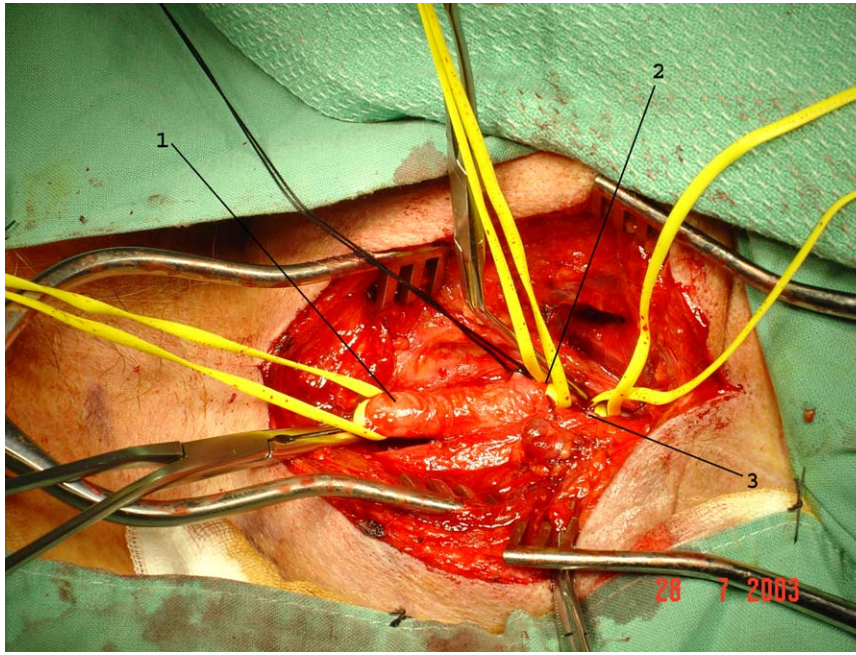

Fig. 2. Ligation of the carotid artery. Yellow elastic bands are seen around the (1) common carotid artery, (2) external carotid artery, and (3) internal carotid artery.

Physical manipulations and excessive handling of the arterial portions filled with atheromatous plaques can lead to the dislodgement of atheromatous debris, a cerebral embolism, and an acute stroke.

When the artery is mobilized and ready for clamping, a decision must be made regarding the need for a carotid shunt. Before carotid clamping, 5000 to 7500 units of heparin are usually administered intravenously. A 3-minute test occlusion technique can be used. Patients are observed for any neurological signs or symptoms of cerebral oxygen deficit that would determine the need for a carotid shunt.

Javid's shunt is one of the most popular devices used to shunt blood during CEA. It is inserted by way of an arteriotomy distally into the internal carotid artery and then proximally into the common carotid artery. Carotid shunting is not a benign procedure; it requires additional surgical time and makes surgical access to the carotid artery more cumbersome. Cerebral embolization from atheromatous debris and microbubbles of air from the tubing can occur, as can bleeding, scuffing of the distal intima, and creation of an intimal distal flap—a potential future source of cerebral embolism.

Endarterectomy is done by dissecting the diseased intima (Fig. 3). The media is left in place. Free edges of the intima are then smoothed carefully, and the incised artery is washed generously with heparinized saline to remove any residual debris.

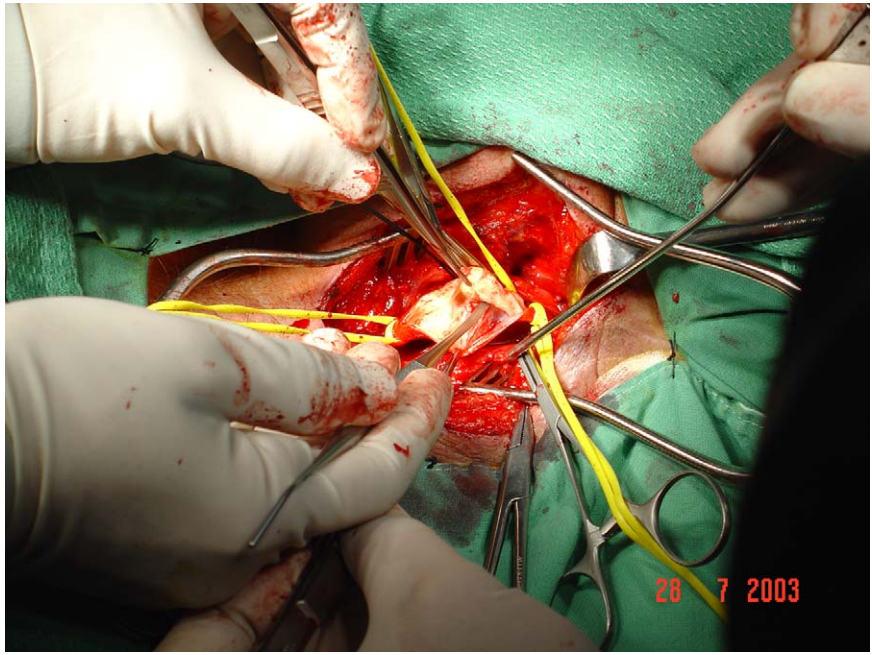

Fig. 3. CEA. Arteriotomy is performed following carotid crossclamping. Diseased intima is carefully dissected, leaving media in place.

Frequent neurological assessment in an awake patient, or monitoring of the electroencephalogram (EEG) in an anesthetized patient, throughout the cross-clamp stage is important. Should any deterioration in neurological status or change in EEG occur, an immediate intervention could save the patient from permanent neurologic damage.

Closure of the artery might, at times, require a synthetic patch to prevent future recurrent stenosis.

The crossclamp is removed when the artery has been closed. Hypertension is commonly seen during crossclamping, but hypotension can develop when the crossclamp is removed.

Closure of the surgical wound should be done only when bleeding is under control. Occasionally protamine might be required to antagonize the action of heparin. A vacuum drain is usually left in place to prevent formation of hematoma on the neck.

General versus local/regional anesthesia

The fundamental question regarding the best choice of anesthesia for CEA remains unanswered. Overall, three choices are available: general, regional, or

local anesthesia, and any combination thereof. A multitude of studies have attempted to define which technique is safer. A Cochrane review was completed on this subject in 1996 and has undergone several updates [1]. Three randomized trials and 17 nonrandomized trials were included in the most recent review, which concluded that there was insufficient evidence to draw conclusions in comparing general versus regional anesthesia for CEA.

In 1999 Sbarigia and colleagues reported the results of their prospective randomized trial of regional versus general anesthesia and its impact on perioperative myocardial ischemia [2]. They analyzed 107 patients who had significant underlying ischemic heart disease who underwent CEA between 1995 and 1998. Patients operated on under general anesthesia had cardiac complications twice as often than those who underwent surgery under regional anesthesia.

Mellière and colleagues conducted a retrospective monocentric study of 670 patients undergoing CEA [3]. Their results also did not reach statistical significance, although regional anesthesia was associated with a lower rate of neurological morbidity and mortality.

Love and Hollyoak prospectively collected data on 443 patients undergoing CEA and concluded that regional anesthesia significantly decreased the number of major strokes and mortality without affecting the cardiovascular complication rate [4]. They also noted that age greater than 74 years was associated with a greater number of major complications.

In 2000 Bartolucci and colleagues conducted another review of the subject [5]. They examined 17 studies involving 14,776 CEAs that were published between 1990 and 2000. Only 2 of the 17 studies shared lower rates of intraoperative shunt use in the regional anesthesia group, but there was no difference in the incidence of stroke rate and neurological mortality.

In 2001 McCleary and colleagues concluded that there are theoretical arguments and clinical evidence that the outcome from CEA might be better when regional anesthesia is used [6]. They called for another prospective, controlled, randomized trial to confirm their impressions.

Calligaro and colleagues from the University of Pennsylvania Hospital reported a retrospective analysis of 401 CEAs performed between 1992 and 1998 [7]. They concluded that a cervical block anesthetic was as safe as general anesthesia, although cephalad carotid lesions are best treated under general anesthesia.

Sternbach et al reported another retrospective analysis of 550 CEAs performed between 1998 and 2000 [8]. They saw no difference in neurological morbidity or mortality but reported lower major cardiac morbidity and less hemodynamic instability in the cervical block group when compared with the general anesthesia group.

McCarthy and colleagues conducted a prospective, randomized clinical trial of 67 CEAs [9]. Their results suggested better preservation of the ipsilateral cerebral circulation and increased tolerance to the effects of carotid clamping in the regional anesthesia group. The combined death/stroke complications rate, however, remained similar in both groups (regional versus general anesthesia).

Gurer and colleagues retrospectively reviewed their results in 329 patients who underwent CEA over the last 10 years [10]. The stroke rate was significantly lower in the regional anesthetic group. Overall mortality and morbidity were similar, though, with death from myocardial infarction the leading cause of mortality.

The author's experience with regional anesthesia is that it is superior to general anesthesia for monitoring cerebral function following carotid clamping. It allows selective intraoperative carotid shunting and helps to prevent major perioperative cerebral ischemic events. No strokes were detected in a personal series of more than 120 consequent CEAs performed under regional anesthesia and mild to moderate intravenous sedation. Morbidity consisted mostly of intraoperative hypertension; two patients had a myocardial infarction postoperatively, but no patients died.

In conclusion, no recommendations can be made regarding the better choice of anesthesia for CEA. Data available from prospective, randomized, controlled trials are insufficient and too controversial for one to make a recommendation.

Patient positioning/warming

Appropriate positioning of patients is paramount for optimization of the surgical exposure (Fig. 4). Patients are usually positioned supine with minor extension of the head achieved by elevation of the shoulders with a sandbag or a slim pillow under the shoulders. The head is also rotated to the contralateral side. Most patients for CEA are elderly and have a degree of limitation in neck mobility caused by arthritis affecting the cervical vertebrae. Such patients might wake up with significant neck pain if they have general anesthesia with aggressive positioning of the head and neck. If patients have regional anesthesia, they will become uncomfortable soon after the beginning of the surgery, restless, poorly cooperative, and will try to move to correct the discomfort, making surgery difficult. Overextension and overturning the head also will create tension in the neck tissues in ipsilateral side, leading to difficulties in tissue retraction and surgical exposure. Using 10° to 20° reverse Trendelenburg positioning of the patient improves venous and lymphatic drainage from the head, leading to the reduction in stasis of blood and minimizing bleeding.

Intravenous cannulas are better placed in the contralateral radial artery, which allows ready access to the intravenous site. It is, however, better in patients who have regional anesthesia to have the radial artery cannula inserted in the ipsilateral side because movements of the contralateral hand are used intraoperatively to assess neurologic function.

The skin is scrubbed and sterile drapes are applied. Drapes can be placed loosely over the patient's head or fixed on poles, leaving free access to the head from contralateral side from the surgery (Fig. 5). The latter is preferable in cases of general anesthesia and is necessary in cases of regional anesthesia because it allows uninterrupted access to the airways and an ability to communicate with the patient. It also helps avoid problems associated with claustrophobia.

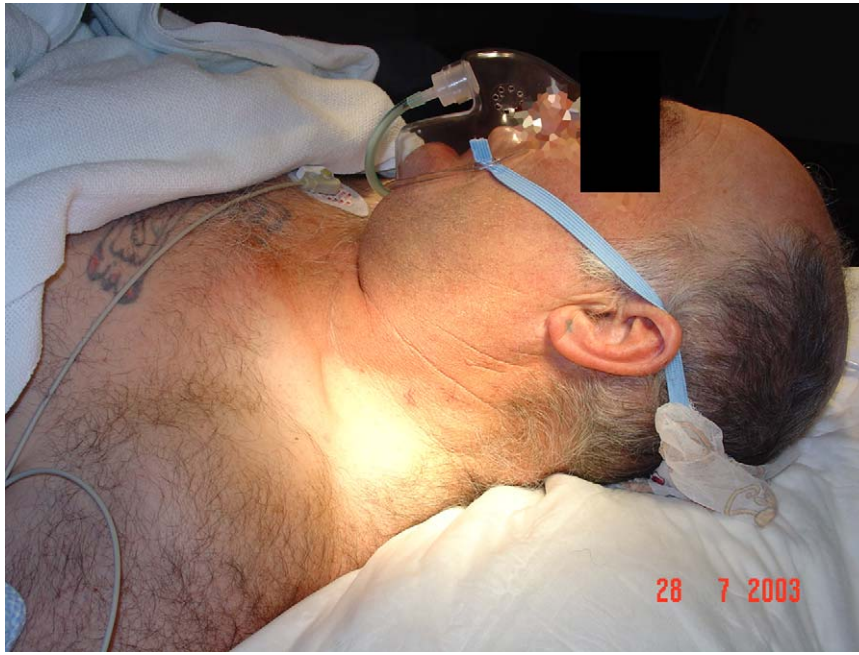

Fig. 4. Patients are usually positioned supine with 10° to 20° reverse Trendelenburg cephalad elevation to improve venous and lymphatic drainage. A slim pillow or sandbag is positioned under the shoulders to allow mild extension of the head and is contralateral from surgery rotation. In patients who have short necks and underlying cervical spondyloarthritis, a more comfortable position must be sought.

If the surgery is conducted under general anesthesia, temperature monitoring is required. Several centers induce a mild degree of passive hypothermia to approximately 34°C to increase the safe ischemic time. Active warming with a warming blanket might be required in some cases, particularly in elderly patents. If the surgery is conducted under regional anesthesia, in most instances active warming is not required; it might cause overheating with associated discomfort and restlessness.

Monitoring of cerebral function

CEA requires complete temporary unilateral occlusion of the carotid artery. Timely identification of inadequacy of collateral cerebral circulation with a critical focal oxygen deficiency provides a window of opportunity for immediate intervention and correction of such deficit. A range of practical and experimental techniques exists for monitoring cerebral function during carotid clamping, but all of them have drawbacks.

Continuous assessment of global and focal neurologic status is possible in awake patients undergoing a regional anesthesia. Carotid occlusion can lead

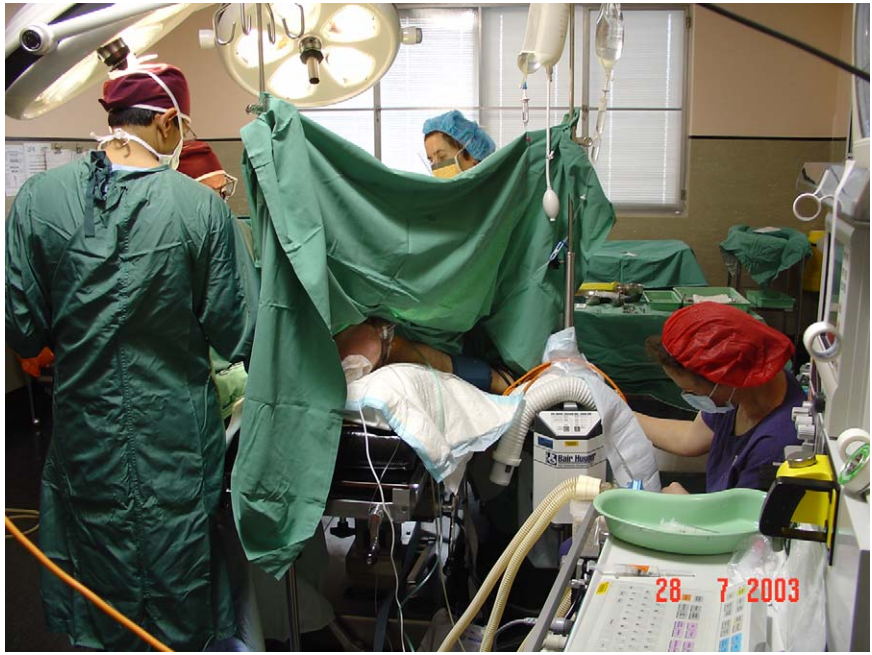

Fig. 5. When CEA is performed under regional anesthesia, free access to the airway is paramount for safety. Drapes are positioned so the patient's face can be seen at all times and free communication with the patient is available. The contralateral hand is extended on an arm support to allow assessment of the grip by the medical staff during crossclamping and reperfusion. The anesthetic machine and all other equipment should be positioned so that the anesthesiologist has uninterrupted access to the head of the patient and ventilation can be initiated at any stage if required.

to critical ischemia of the ipsilateral hemisphere; therefore, contralateral motor function of the extremities needs to be observed. Practical assessment consists of frequent (every 2–3 minutes) examination of the strength and contralateral handgrip and maintenance of constant verbal contact with the patient to assess the level of consciousness, which requires the patient's cooperation, reasonable comprehension, and presence of the same observer throughout the surgery to enable adequate comparison of the strength of handgrip before and during the carotid occlusion.

Such a technique has provided the main argument for regional anesthesia in vascular surgery since Matas described a challenge occlusion test in 1911 for the assessment of collateral circulation [11]. Usually the first clinical signs of cerebral ischemia appear after 2 to 3 minutes of the carotid occlusion. If any neurological problems are observed, shunting can be employed immediately.

Monitoring of awake patients is a gold standard for neurological assessment. In patients under general anesthetic, indirect cerebral monitoring techniques had to be found to identify neurologic deficits during crossclamping to allow intervention (shunting) to decrease the incidence of postoperative deficit. The many monitoring techniques that have been advocated include internal carotid artery

stump bleeding, stump pressure, jugular venous oxygen saturation, EEG, processed EEG (bispectral index [BIS]), somatosensory-evoked potentials (SSEPs), transcranial Doppler (TCD), arteriography, and some other experimental techniques that at present have no practical applications.

Backbleeding of the internal carotid artery suggests reasonable collateral circulation above the clamp. It is, however, a subjective and nonquantitative technique. This method has not been supported by randomized, prospective studies and could not be recommended for routine assessment of the adequacy of cerebral collateral circulation.

Backpressure measurements can be used to quantify carotid stump bleeding. Moore and colleagues suggest that a temporary shunt should be used routinely in all patients who had a previous cerebral infarction independent of backpressure and for all patients who have a stump pressure of less than 25 mmHg [12]. They further concluded that patients undergoing CEAs for transient ischemic attacks or asymptomatic carotid stenosis do not require shunting unless their carotid stump pressure is below 25 to 40 mmHg. At present, many clinicians use the level of 50 mmHg as a cutoff value. A recent study of 147 patients who underwent CEA under regional anesthesia with awake neurological monitoring suggested that the level of stump pressure is not a reliable predictor for the need of a shunt [13].

EEG is the gold standard for monitoring patients undergoing CEA under general anesthesia. A recent retrospective study from the Mayo Clinic conducted on 135 patients undergoing CEA under regional anesthesia demonstrated that 7.4% of patients had hemispheric EEG changes with none exhibiting global EEG changes [14]. These results were compared with 288 patients who had CEAs under general anesthesia; 15.3% of those patients had some EEG changes and 3.5% of the 228 patients had global-type EEG changes. Another group of investigators recently reported that the routine use of EEG during CEA performed under general anesthesia in 564 patients resulted in complete elimination of intraoperative strokes [15].

If using EEG to monitor patients undergoing CEAs, one needs to be aware that the general anesthetic can influence the EEG [16]. Laman et al analyzed 152‘ patients and found that isoflurane compared with propofol resulted in different EEG patterns during crossclamping [16]. They recommended differential derivation of the EEG depending on the general anesthetic used; however, analyzing the EEG is cumbersome in practice and requires special expertise for interpretation of the data.

Processed EEG is easier to monitor and interpret. BIS, which is increasingly being used in routine practice to monitor the depth of general anesthesia and sedation, has been found to identify severe cerebral ischemia; however, BIS is a global hemispheric monitor and cannot be used to detect focal cerebral ischemia reliably.

Jugular oxygen venous saturation has been used in an attempt to detect the sudden onset of cerebral ischemia. It is, however, too global an assessment that does not reflect regional or especially focal cerebral ischemia, and it cannot be recommended for routine clinical practice for CEA.

The use of SSEP monitoring during CEA is still inconclusive. A group from Toronto reported a retrospective review of 204 patients who underwent CEA under general anesthesia and concluded that the technique could be useful; even patients who had preoperative strokes had baseline asymmetry in their SSEP [17]. A prospective study of 50 consequent patients undergoing CEA under regional anesthesia concluded that SSEP was associated with a 2% false-negative rate [18]. The threshold for detecting cerebral ischemia was lower than the currently reported value for patients under general anesthesia. They further concluded that the SSEP has limited value in the detection of cerebral ischemia and in assessment of the effectiveness of the shunting because of hysteresis.

TCD ultrasonography provides noninvasive assessment of the velocity of blood flow in the middle cerebral artery. Though the idea of continuous monitoring of blood flow inside the brain sounds good, the change in the velocity might reflect not only a decrease in flow but also changes in the diameter of the artery. Belardi and colleagues concluded that TCD is not sufficiently reliable for the prediction of shunt requirements [13]. Doppler is useful for detection of intraoperative cerebral emboli. Lennard and colleagues conducted a prospective study of 252 patients combining TCD and angioscopy for identification of intimal flaps [19]. They had a postoperative stroke rate of 2.8% and concluded that despite the reduction in the rate of intraoperative stroke, perioperative neurological problems were not necessarily avoided.

There are several drawbacks to the procedure. The ultrasound probes are positioned in the temporal areas and limit surgical and anesthetic access to the head. Technical difficulties make the TCD difficult to interpret 20% of the time.

Meticulous preoperative assessment of patients to select those who might require shunting has been suggested as a way to predict who will require intervention in addition to intraoperative monitoring. Detailed preoperative evaluation of the anatomy of circle of Willis [20] and stress brain–perfusion SPECT imaging [21] has been suggested as effective techniques for preoperative prediction of the need for a shunt.

In conclusion, it is the author's opinion that an awake patient is preferable to using general anesthesia and intraoperative monitoring to predict the need for shunting. Despite technological advances and a multitude of techniques available for the cerebral monitoring during general anesthesia, none of those techniques is ideal and all of them have practical limitations. The EEG assessment offers the most reliable results.

Cardiovascular monitoring

Intraoperative mortality in patients undergoing CEA is largely caused by myocardial infarction. Underlying coronary artery disease (CAD) has been reported to have 30% to 85% association with carotid disease. Up to 30% of patients undergoing CEA might develop an intraoperative myocardial ischemia. CEA is associated with rapid, unpredictable, significant shifts in heart rate and blood

pressure. Stress, coronary angiospasm, tachycardia, and increased afterload are the most frequent triggers of myocardial oxygen debt. It is imperative to conduct close cardiovascular monitoring throughout the anesthetic and early postoperative period.

Standard cardiovascular monitoring should include continuous ECG (usually leads II and V5) with ST segment analysis and invasive arterial pressure monitoring. Optional techniques might include 12-lead ECG and transesophageal echocardiography during general anesthesia for patients who have severe CAD.

The pulmonary arterial catheter is not a sensitive tool for detecting myocardial ischemia. CEA is rarely associated with significant loss of blood and shifts of fluids. Intravenous fluid management is rarely a problem. Routine use of a pulmonary arterial catheter cannot be recommended in CEA.

Maintenance of cardiovascular status

A labile hemodynamic state is characteristic for patients undergoing CEA. All types of hemodynamic changes can take place.

Bradycardia

The heart rate slows during surgical manipulations of the carotid sinus. Direct stimulation of the vagus nerve during dissection also can contribute to the onset of bradycardia. The presence of hypovolemia might exacerbate this phenomenon. Maintenance of adequate volemic status is required. Prophylactic injection of 1 to 2 mL of a local anesthetic between the internal and external carotid arteries before manipulation of these vessels can attenuate the bradycardia. Other treatment is usually not needed. Administration of anticholinergic drugs can result in tachycardia, excessive hypertension, and increased myocardial oxygen requirements, so it is better avoided.

Tachycardia

Tachycardia often occurs as a result of stress, pain, or as a direct result of catecholamine release following manipulation of the carotid sinus. Tachycardia is undesirable because the majority of patients have underlying CAD. Because the final common pathway is the same (increased catecholamines), treatment with short-acting β-blockers (eg, esmolol) might be helpful, but only after analgesic requirements have been met.

Hypotension

Hypotension is more frequent under general anesthesia because of the direct vasodilating and negative inotropic effects of several anesthetics, but it can also occur because of hypovolemia and bradycardia. Hypotension following carotid unclamping and cerebral reperfusion is common [22], particularly in patients who have severe carotid stenosis. The cause is not clear, but it could possibly be a

cerebral protective process. Cerebral autoregulation protects the brain from reperfusion injury by reducing cerebral production of rennin, vasopressin, and norepinephrine, leading to hypotension.

Severe hypotension can be prevented in many instances by adequate intraoperative intravascular hydration [23]. Blood pressure less than the preoperative level should be avoided during crossclamping of the carotid artery, especially if a shunt is not used. Low blood pressure during the carotid crossclamping time should be managed with intravenous fluids and titration of vasopressors to preserve collateral cerebral perfusion.

Untreated severe hypotension after unclamping of the carotid artery can potentially lead to cerebral hypoperfusion, thrombosis of the carotid artery, myocardial ischemia, and third organ damage. Mild hypotension might be tolerated well by patients whose danger of cerebral reperfusion injury and decreased myocardial oxygen consumption is low. In such cases treatment is not required.

No strong recommendation can be made at present regarding the correction of hypotension after unclamping the carotid artery. The decision to treat low blood pressure should be made by clinicians on individual basis, considering the needs of each patient.

Hypertension

Many patients undergoing CEA have underlying hypertensive disease that is further exacerbated by the surgical stress and surgical manipulation of the carotid body, causing sympathetic stimulation and the release of catecholamines.

Crossclamping of the carotid artery is usually accompanied by an increase in systemic blood pressure, which helps to preserve collateral cerebral perfusion. Stimulation of baroreceptors in the carotid body and intracranial release of rennin, vasopressin, and norepinephrine are responsible for the increase. The hypertension is usually transient. Severe and persistent hypertension can lead to myocardial ischemia and myocardial infarction or cerebral hemorrhage.

Some increase in arterial blood pressure during crossclamping is desirable, but systolic pressure over 160 mmHg, and certainly over 180 mmHg, requires therapy. Hypertension following unclamping of the artery should also be treated, trying to achieve preoperative levels of blood pressure control. Persistent hypertension can lead to cerebral reperfusion injury, cerebral hemorrhage, surgical bleeding, wound hematoma, and anemia. Nitroglycerin and sodium nitroprusside are agents that are commonly used in this situation. The choice of drugs is probably not critical as long as their half-life is short and does not exacerbate hypotension following carotid unclamping.

Heparin and protamine

Heparin is usually administered as a bolus immediately before carotid clamping. The dose differs from center to center and ranges widely (45–140 u/kg).

Many patients receive antiplatelet agents (eg, aspirin, clopidogrel, ticlopidine) preoperatively for preexisting cerebrovascular and CAD. These drugs are frequently continued until the day of surgery to decrease the risk of stroke and coronary events.

Hartung and colleagues recently reported on the use of low molecular weight heparin (LMWH) for CEA [24]. They administered 70 UI/kg of nadroparin before carotid artery clamping then 2850 to 3800 UI every 12 hours in patients who had CEA between 1995 and 1999. All patients received general anesthesia, and shunting was used in 40% of cases. Seven patients developed perioperative strokes and none developed hemorrhagic stroke. Four patients developed a cervical hematoma requiring re-exploration. One patient died because of duodenal bleeding. No patients developed thrombocytopenia. The authors concluded that LMWH was as efficacious as and could be used with heparin, but it had fewer side effects.

Concurrent administration of antiplatelet agents and heparin might result in persistent bleeding following CEA. Meticulous hemostasis and patience is important for surgical management.

The use of protamine to reverse heparin is controversial. Decisions on its use need to be individualized. The risk of bleeding must be weighed against the potential risk of postoperative thrombosis and hypotension following unclamping of the artery. Sometimes a half-dose of protamine might be sufficient.

Antibiotics

Routine use of antibiotic prophylaxis is not required for CEA; however, single doses of wide-spectrum antibiotics should be given intraoperatively when a synthetic carotid patch is used.

General anesthesia

The major advantage of general anesthesia is an ability to offer ideal operating conditions for the surgical team. Many vascular surgeons still refuse to operate under regional anesthesia, fearing what will happen if the patient strokes intraoperatively. Patients might also express uneasiness about being awake during the surgery and ask for a general anesthetic. The major disadvantage of general anesthesia is the inability to provide precise monitoring of cerebral function.

The major tasks during general anesthesia for CEA are to preserve cerebral circulation and oxygen delivery at all stages of surgery, to provide cerebral protection, to prevent myocardial ischemia, and to prevent patients from coughing and straining.

Because many patients are elderly and have CAD, more care than normal should be taken to avoid hypotension and hypertension on induction of anesthesia. Sodium thiopental, propofol, and etomidate are the induction drugs used most frequently in recognition of their cerebroprotective effects.

Neuromuscular blocking agents (NMBAs) are usually used to ensure patients' immobility. The choice of NMBA is not important as long as it will not induce

hemodynamic disturbances, such as could be seen with a vagolytic agent such as pancuronium.

Tracheal tubes and laryngeal mask airways (LMA) have been used to protect the airway; both have their positive and negative sides. On the positive side, a tracheal tube will ensure adequate airway protection, allowing mechanical ventilation; however, a tracheal tube can induce tachycardia and hypertension following intubation. With extubation, coughing and straining can occur. LMAs help avoid the sympathetic response associated with tracheal intubation. They do not, however, provide complete protection of the airways (this includes ProSeal LMA The Laryngeal Mask Company Limited, Henley on Thames, United Kingdom, as well as the standard LMA), and in some cases they can be a problem for adequate ventilation, especially in rare cases in which glottic or supraglottic edema occurs. The choice of the LMA should therefore be made on individual basis. Should a tracheal tube be used, prior administration of an opioid (ie, alfentanil) might help prevent the sympathetic response to manipulation of the trachea. LMAs should not be used in patients who have an increased risk for aspiration (ie, those who have hiatus hernia).

Total intravenous anesthesia and inhaled anesthetics have been used for CEA and have been compared against one another in several studies. Maintenance of anesthesia should be with drugs that the anesthesiologist knows will provide good cerebral perfusion with maximum cerebral and myocardial protection, avoiding short-acting drugs that prolong recovery.

Nitrous oxide has been associated with an increase incidence of postoperative myocardial ischemia, so it should probably be avoided [25].

Doyle and colleagues compared fentanyl and remifentanil as supplements to isoflurane/nitrous oxide anesthesia. They found no difference in the incidence of hemodynamic events, postoperative pain, and nausea or vomiting [26]. Wilhelm and colleagues compared fentanyl and remifentanil as supplements to desflurane anesthesia [27] and found that remifentanil was superior to fentanyl for rapid recovery and early neurologic examination.

De Castro et al further investigated the use of remifentanil for CEA [28] and concluded that target-controlled infusion of remifentanil allowed better hemodynamic stability when compared with a continuous weight-based infusion. Mouren et al compared remifentanil and sufentanil for CEA [29] and concluded that both drugs provided equally good hemodynamic intraoperative and postoperative stability. Remifentanil was superior in suppressing the sympathetic response of intubation.

General anesthesia does not prevent the hemodynamic responses to manipulation of the carotid sinus; therefore it is advisable to inject a small amount of local anesthetic (1–2 mL of 1–2% lidocaine) in the tissue between the internal and external carotid arteries before surgical manipulation. A recent case report from South Korea reaffirms that carotid sinus stimulation—even under general anesthesia—might precipitate severe hemodynamic response, leading to coronary artery spasm [30].

Hemodynamic abnormalities have a rapid onset and can be extreme, but they are usually short-lived. Bradycardia can be followed quickly by tachycardia, and severe hypotension can be replaced rapidly by severe hypertension depending on the pathophysiologic stimulus. Such labile conditions require the utmost attention throughout the anesthetic procedure with rapid pharmacological response using short-acting drugs when deemed necessary. The use of local anesthetics by the surgeon often can help to prevent extreme changes in hemodynamics.

Cerebral intraoperative protection

First of all, a decision needs to be made regarding the use of the carotid shunt. Carotid shunting improves cerebral oxygen delivery and offers the best cerebral protection, but it does not guarantee protection against a stroke. Even when a surgeon works fast, 3 to 4 minutes of complete clamping are often needed following arteriotomy for shunt insertion. Another 2 to 3 minutes of full clamping are required when the shunt is removed and the last sutures are placed on the artery, which might, potentially, be enough to sustain a stroke. Displacement of atheromatous debris, embolism of air bubbles, and thrombosis of the shunt are also potential sources of ischemic cerebral events. Total surgical time is increased because additional time is required for the placement of the shunt and because the presence of the shunt makes surgical conditions less optimal, with the tubing obscuring part of the surgical field. Possible surgical complications of the shunt include creation of a distal intimal flap and bleeding. Considering these complications, selective use of shunting should be employed. The presence of severe contralateral internal carotid stenosis does not indicate, per se, the need for a shunt [31].

External carotid artery shunting can be employed instead of internal carotid artery shunting. It can often provide about 20% of the collateral blood supply. Belardi and colleagues prospectively studied 137 patients undergoing CEA under regional anesthesia with TCD monitoring [32]. They concluded that a positive external carotid artery test (mean velocity measured in the middle cerebral artery following crossclamping of the internal carotid artery, similar to the preoperative values) was deemed suitable for selection of the candidates for the effective external carotid artery shunting.

Maintenance of an adequate collateral blood flow requires maintenance of an adequate arterial pressure at all times during crossclamping, even when a shunt is in place. Current recommendations are that arterial blood pressure should be maintained at the preoperative level or slightly higher, which usually occurs without medical intervention because of the factors discussed previously. There are cases, however, when hypotension occurs and rapid pharmacologic response is required. A bolus of intravenous fluids or titration of vasoconstrictors or inotropes might be needed. No evidence is available to help choose intravenous fluids or vasoconstrictors in carotid surgery. If an ionotrope is chosen over a

vasoconstrictor, myocardial oxygen demand use will increase, but the same can occur with a vasoconstrictor because of an increase in afterload.

Induced hypercapnia has been used to achieve cerebral vasodilation to improve blood supply to the affected areas of the brain; however, it has been suggested that a steal effect might develop as a result of cerebral vasodilation in the opposite hemisphere. Induced hypocapnia has been offered as a solution in an attempt to vasoconstrict the contralateral hemisphere. Such a maneuver can result in nondiscriminatory bilateral vasoconstriction and therefore might increase ischemia of the affected areas of the brain. Normocapnia should be maintained during general anesthesia for CEA.

Volatile anesthetics and barbiturates provide some cerebral protection against ischemia, but no controlled, randomized study has been able to demonstrate that any particular pharmacologic agent can reduce the incidence of postoperative neurologic damage.

Extubation and emergence from general anesthesia require careful management to avoid major swings in blood pressure and to avoid coughing. Intraoperative damage to the ipsilateral recurrent laryngeal nerve can result in impaired ability to cough and poor sputum clearance. Voice hoarseness is observed, but it is unlikely to cause any major respiratory difficulties. Tracheal extubation of reasonably deeply anesthetized patients might be helpful to prevent undesirable effects such as coughing or bucking on the table.

Regional anesthesia

The major advantage of regional anesthesia is an ability to ideally monitor cerebral function during the crossclamp stage. Awake patients offer ideal conditions for focal neurologic assessment, which is the basis for selective intraoperative carotid shunting. A regional anesthetic technique avoids unnecessary ventilatory support with all the associated potential complications. The downside of regional anesthesia is the psychological stress of the patient being awake, the possibility of inadequate analgesia, movement of the patient resulting in a less controlled environment for the surgical team, and limitation in administration of reputed cerebral protective drugs such as barbiturates. There is also a concern about access to the airway in the case of unexpected emergencies.

Several techniques can be used to provide regional anesthesia for CEA: deep cervical block, superficial cervical block, and cervical epidural anesthesia.

A deep cervical block anesthetizes the cervical plexus that is formed from C2 to C4 anterior rami of the ipsilateral spinal roots. This plexus is mostly responsible for sensory innervation of the deep cervical structures.

Several techniques have been described for a deep cervical block. Patients are usually positioned supine in reverse Trendelenburg position (10–20°) with the head slightly rotated to the contralateral side and extended posteriorly. After identifying the mastoid process and transverse process of C6, an imaginary line is drawn that is usually 1 cm posterior to and parallel to the sternocleidomastoid

muscle. The C4 process can often be palpated one-third of the distance from C6 to the mastoid process. Alternatively, the intersection of the line with a line drawn across the neck at the level of the midthyroid cartridge or the point of the external jugular vein crossing the sternocleidomastoid muscle can be used as a landmark. A ruler can be helpful for measuring the distance and approximating the position of the transverse cervical processes. The transverse process of C2 can be found about 1.5 to 2 cm below the mastoid and along the same line. The C3 is usually exactly between them. The author usually uses a 25-gauge, 2-inch-long needle to inject 0.5 mL of local anesthetic into the skin. The needle is advanced perpendicular to the neck with 10° to 15° of posterior tilt (Fig. 6). Constant aspiration is used to identify potential penetration through the blood vessels. The C4 process is frequently found 1.5 to 2.5 cm under the skin. The size of the neck plays a major role in detecting the depth of the transverse processes. When the needle has reached the transverse process, it is withdrawn 1 to 2 mm and 5 to 6 mL of local anesthetic is injected slowly. The technique is then repeated at the

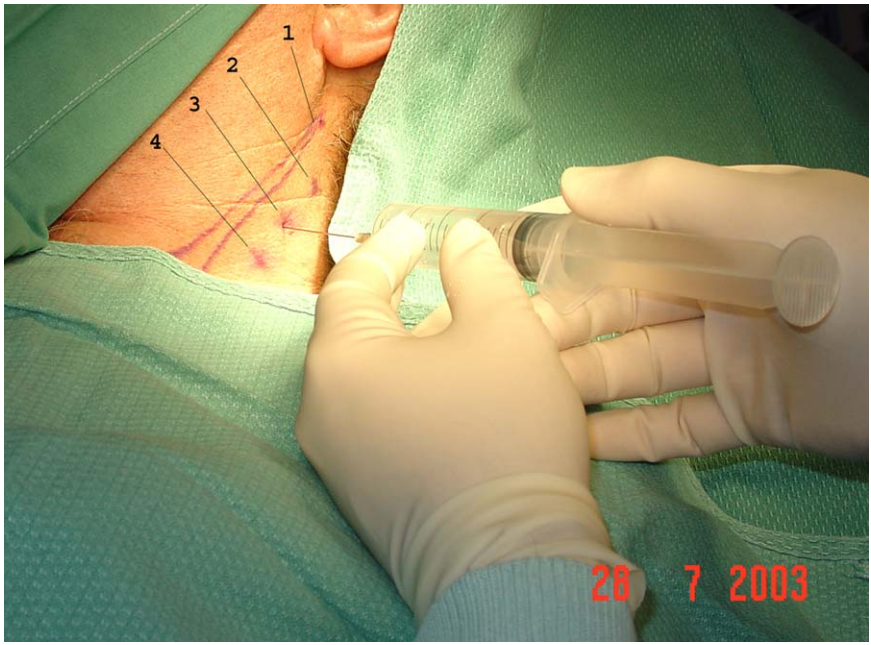

Fig. 6. Deep cervical plexus block. (1) A line is drawn along the sternocleidomastoid muscle. A parallel line is drawn connecting the posterior border of the mastoid process and the C6 lateral process. (2) C2 is usually located approximately 1.5 to 2 cm below the mastoid on this line. (3) C3 is between C2 and C4. (4) C4 can be palpated approximately two-thirds from mastoid to C6 or found on the intersection of this line and midthyroid cartridge. The needle is advanced perpendicular to the neck with a 10° to 15° posterior tilt. Transverse processes are found on average 1.5 to 2.5 cm under the skin. The needle is withdrawn 2 mm and 5 to 6 mL of local anesthetic are injected slowly at each level. Aspiration is required at all stages to prevent arteriovenous or dural penetration and inadvertent intravascular or intrathecal local anesthetic injection.

C2 level with the reduced posterior tilt of the needle on insertion. The C3 root is last, but it might be unnecessary sometimes.

The use of nerve stimulator for identification of the cervical plexus is controversial. Merle and colleagues compared deep cervical block (three roots) against a single injection conducted with nerve stimulator-guided identification of the cervical plexus [33]. They concluded that the latter procedure is as effective and might be associated with less systemic absorption of the local anesthetic than the former procedure.

The major potential risks associated with deep cervical block are related to the proximity of injection to structures such as the carotid artery, internal jugular vein, vertebral arteries, spinal canal, and phrenic nerve. Inadvertent intravascular or intrathecal injections and paralysis of the ipsilateral diaphragm are potential serious side effects.

Superficial cervical block is aimed at anesthetizing the C2 to C4 branches responsible for sensation of the superficial neck tissues. A line is drawn between the mastoid and C6 in a process that is similar to that of the deep cervical block technique. The injection point is in the middle of this line. Alternatively, the intersection between this line and the upper border of the thyroid cartridge, or just above the point of external jugular vein crossing the sternocleidomastoid muscle, can be used. A patient can be asked to lift his or her head to help identify the posterior border of the sternocleidomastoid muscle. The needle is inserted just under the muscle without perforating deep cervical fascia. Three to 5 mL of a local anesthetic is injected, then the needle is reinserted from the same point aiming cephalad then caudal (fan-shaped injection), each time introducing 3 to 5 mL of anesthetic. Aspiration is required every time before the administration of local anesthetic to avoid intravascular injection.

Several studies have been completed comparing deep and superficial cervical blocks. Stoneham and colleagues prospectively randomized 40 patients undergoing CEA to receive deep or superficial cervical plexus block [34]. They concluded that the technique made no difference in the operative conditions or the need for supplemental intraoperative local anesthesia. Pandit and colleagues (from the same center) conducted a randomized study of 40 patients receiving a superficial versus a combined (deep and superficial) block [35]. They also found no significant difference between the techniques.

The major risk associated with superficial cervical block is inadvertent intravascular injection, particularly into the external jugular vein. Rarely, phrenic nerve blockade might occur.

Several authors suggest the use of cervical epidural blockade for carotid surgery [36,37]. In this technique an epidural catheter is introduced 4 to 5 cm cephalad from the C6 to C7 level. A prospective, randomized study of 90 patients suggests that there is no difference in efficacy between cervical epidural blocks and cervical plexus blocks [36]. There was one case of intradural and one case of intravascular injection, neither of which resulted in significant complications. More cases of arterial hypotension were reported in the epidural group. Cervical epidural anesthesia carries more potential risks when compared with the cervical

plexus block, and if there is no difference in efficacy it could not be recommended for routine use in CEA.

The choice of anesthetic agent used during regional anesthesia for carotid surgery should be influenced by the potential length of the operation and by the systemic toxicity of the drug. Bupivacaine seems to produce the longest and strongest block, but it is associated with the most toxicity. Levobupivacaine should have efficacy similar to bupivacaine with less potential systemic effects. Ropivacaine can be used with similar intraoperative results, but postoperative analgesia might be shorter [38]. Leoni and colleagues conducted a prospective, randomized, double-blind study on 60 patients undergoing CEA under a combination of deep and superficial cervical blocks with ropivacaine or mepivacaine [39]. They concluded that both drugs provided good anesthesia, but the ropivacaine group had longer postoperative pain relief.

The difficulties associated with cervical block are experienced more frequently in patients who have short, large necks and in patients who have high carotid stenosis. A combination of deep and superficial cervical blocks using levobupivacaine or ropivacaine and local infiltration of the skin with 1% lidocaine works best. An injection of 2 to 3 mL of local anesthetic into the sternocleidomastoid muscle sheath is also beneficial, as is skin infiltration, which helps cover the most cephalad and caudal parts of the incision, where collateral innervation is often not affected by the cervical plexus blockade.

For regional anesthetic techniques, patients often receive a combination of sedatives and small doses of opioids, which provide supplemental analgesia and serve as anxiolytic and amnesic agents. The main advantage of using regional anesthesia is to maintain a reasonable level of consciousness for neurologic monitoring. Individual titration of sedatives is the best way to maintain the required depth of sedation. Midazolam, propofol, dexmedetomidine, fentanyl, morphine, remifentanil, and other drugs can potentially be used. Remifentanil has a higher incidence of adverse respiratory effects [40], so it should be used with caution.

Return to the operating room

Two major reasons that prompt early return to the operating room following CEA are thrombosis of the carotid artery and bleeding leading to neck hematoma.

The sudden onset of a major ischemic neurologic event signals a possible thrombosis. Doppler ultrasonography or emergency angiography is usually used to confirm the diagnosis. Because of the urgency and type of surgery and because of concerns about the airway, general anesthesia with tracheal intubation should be used.

Bleeding and neck hematoma are more challenging to the anesthesiologist. Urgent removal of the compressing hematoma will frequently not resolve the threat to the airway. Patients can develop stridor and become progressively agitated. Development of supraglottic edema and induration of neck tissues with blood require tracheal intubation to secure the airway before complete obstruc-

tion. If airways are not compromised and regional anesthesia is used during initial endarterectomy, then no additional anesthesia is usually required because cervical plexus blocks last, on average, 6 to 8 hours. The patient might only need mild additional sedation and reassurance.

Summary

Intraoperative management of CEA is highly dynamic and challenging. Further studies are required to establish the best anesthetic technique. Regional anesthesia in an awake patient allows the best cerebral monitoring. Good management is key for cerebral and cardiac protection.

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