Anesthetic Considerations for Carotid Artery Surgery

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Preoperative Considerations

Current indications for carotid endarterectomy (CEA) are based upon prospective randomized trials of symptomatic and asymptomatic patients with carotid artery disease.[1,2] It is well accepted that symptomatic patients (previous transient ischemic attack, reversible ischemic neurologic deficit or mild stroke within 6 months) with ≥ 70% carotid artery stenosis (CAS) are candidates for CEA. In addition, treatment of asymptomatic CAS > 60% with aspirin and CEA reduces 5 year risk of fatal and non-fatal strokes compared to aspirin alone.[2]

Preoperative conditions other than angiographic anatomy have not consistently been identified as predictive of adverse outcome in CEA patients. A multicenter review of nearly 700 CEA found that only angiographic characteristics (ipsilateral carotid occlusion, stenosis near the carotid siphon or intraluminal thrombosis) and age > 75 yr were predictive of perioperative complications.[3] Another multivariate analysis was unable to identify any predictive association of age, sex, indication for surgery, bilaterality of CAS, hypertension or smoking with adverse outcome after CEA.[4] In contrast, a larger multicenter study (n=1,160) identified several clinical predictors of adverse outcome (CVA, MI or death) after CEA, including age > 75 yr, symptom status (ipsilateral symptoms versus asymptomatic or non-ipsilateral symptoms), severe hypertension (diastolic BP>110 mmHg), CEA prior to CABG, hx of angina, evidence of internal carotid artery thrombus as well as internal CAS near the carotid siphon.[5] The presence of ≥ 2 factors was associated with a two-fold increase in adverse events.

Coexistent CAD remains a major cause of morbidity and mortality after CEA and assessment of the severity of CAD is an important aspect of evaluation before CEA. The decision to proceed with preoperative testing to assess cardiac risk should be based on the presence of clinical markers (major, intermediate or minor predictors), functional capacity and the surgical procedure specific cardiac risk.[6] Carotid endarterectomy is typically considered an intermediate risk procedure. Specific preoperative cardiac testing is generally not indicated in this setting in the absence of major clinical predictors of increased cardiac risk (unstable coronary syndromes such as recent MI with evidence of important ischemic risk and unstable or severe angina, decompensated CHF, significant arrhythmias or severe valvular disease).

Patients with internal CAS often have associated impairment of cerebrovascular reactivity (CVR) and reduced ability to further dilate intracerebral arterioles in response to declines in cerebral perfusion pressure. Use of transcranial Doppler (TCD) assessment of changes in middle cerebral artery blood flow velocity as a marker of CVR has been recommended for prediction of cerebral ischemic risk and to identify asymptomatic patients with CAS who are at greatest stroke risk.[7] Patients with impaired CVR to CO_2 demonstrated by TCD preoperatively have not been shown to have increased risk of cerebral ischemia during CEA, as assessed by somatosensory evoked potential recording (SEP).[8] Patients with residual cerebral ischemia after obstructive carotid artery lesions are removed or bypassed may have impaired CVR with an increased risk of stroke[9] and decreases in BP should be meticulously avoided in such patients.

Although adequate preoperative BP control should logically be associated with decreased incidence of cardiac and neurologic morbidity, there are no conclusive prospective data to confirm that delaying CEA to achieve a certain level of preoperative BP actually reduces morbidity and no data to define how long a period of control might be required to realize such a potential benefit. What is known is that poorly controlled hypertensive patients frequently have labile intraoperative BP and are more likely to have both postoperative hypotension and hypertension. Retrospective data indicate that hypertensive patients whose BP is pharmacologically controlled before surgery have a lower incidence of postoperative hypertension (and transient neurologic deficits) than patients with poorly controlled BP (170/95 mmHg).[10] Given the known alterations in cerebral blood flow autoregulation with chronic hypertension, BP reductions should be undertaken gradually and complete normalization of BP is probably not required and may even have detrimental effects.

Intraoperative Considerations

The major goals of management for patients undergoing CEA are modulation of risks for myocardial and cerebral ischemia that are amenable to modification. Maintaining adequacy of cerebral perfusion, continual adjustment of cardiovascular parameters and monitoring the patient appropriately to facilitate prompt intervention to reduce the risk of potential adverse neurologic or cardiovascular events are the essential elements of anesthetic management.

Cerebral Monitoring

A large number of methods are available for intraoperative neurologic monitoring, although no single method is infallible, in large part because of the heterogeneity of the causes of cerebral ischemia and the complex sequelae of cellular events along with the variable location of ischemic insults (i.e. lacunar versus cortical). The ideal method of monitoring cerebral perfusion during CEA remains controversial. Available techniques include xenon blood flow, transcranial Doppler ultrasonography, cerebral oximetry, SEP, EEG and continual clinical neurologic exam during regional anesthesia (RA). The latter two methods are the most commonly utilized and they are probably better monitors of the adequacy of cerebral perfusion than carotid stump pressure alone.[11] Although reduced carotid stump pressure is generally associated with a greater risk of ischemic EEG changes, it is generally considered to be neither sufficiently sensitive nor specific to serve as a guide to selective carotid shunting,[12] and it is difficult to define a critical pressure that does not result in an unacceptably high number of false positives or false negatives.

Neurologic testing during CEA in the awake patient with RA is generally accepted as a sensitive monitor of cerebral function and can reveal clinically significant cerebral ischemia even when sensitive EEG monitoring remains unchanged.[13] This can potentially occur when the ischemic insult is located within deeper brain structures and when pre-existing electrophysiologic abnormalities make it difficult to identify superimposed new abnormalities.[14] While processed EEG data are more “user friendly” sensitivity is reduced compared with multichannel analog EEG. For example, density spectral array analysis simplifies interpretation of EEG data, but it may not reliably detect mild analog EEG pattern changes of cerebral ischemia.[15] Compressed spectral array analyses of EEG data, especially declines in the spectral edge frequency are also less sensitive than the raw EEG as a marker for ischemia.[16] One observational, non-interventional study collected EEG data during CEA without shunting and documented that 80% of immediate strokes after awakening from GA for CEA are associated with severe intraoperative EEG changes.[17] However, no data define how severe or how long intraoperative EEG changes must persist to be predictive of stroke after CEA, nor are there prospective data to define whether the EEG is decisively better than alternative methods of assessing the adequacy of cerebral perfusion.

Transcranial Doppler (TCD) ultrasonography applied across the relatively thin temporal bone allows continuous measurement of blood flow velocity in the middle cerebral artery distribution and may be helpful in differentiating between intraoperative hemodynamic versus embolic neurologic events. Failure to obtain interpretable TCD signals occurs in 15-20% of cases because of temporal hyperostosis or other technical difficulties. Unfortunately, values for blood flow velocity and/or pulsatility index which correlate with critical cerebral blood flow reduction have not been identified. Patients with minimal changes in blood flow velocity during carotid clamping (with shunting) have been shown to have stroke rates similar (or even slightly greater) than when flow velocity is unchanged and shunts are not used.[18] Transcranial Doppler detected embolization occurs in more than 90% of patients during CEA.[19] Emboli having TCD characteristics of air (occurring at shunt opening and during restoration of flow) are generally not associated with adverse clinical outcome. However, particulate emboli (> 10) detected by TCD during carotid dissection correlate with significant deterioration in cognitive function after CEA,[19] postoperative ischemic events, as well as new ischemic lesions on magnetic resonance images of the brain.[20] More careful surgical dissection of the artery and more meticulous attention to backbleeding and flushing to avoid embolization may be guided by acoustic evidence for embolism although it is unknown if such an approach alters outcome. Transcranial Doppler monitoring may also indicate which patients should have aggressive hemodynamic interventions and/or be anticoagulated since cerebral embolic events and decreased cerebral blood flow velocity can be differentiated.

Near-infrared spectroscopy (NIRS) can also be used to assess changes in cerebral blood flow by measuring regional cerebral oxygenation. This technique assesses oxygenation of arterial, capillary and venous hemoglobin and predominantly estimates venous oxygenation (the largest of the three cerebral vascular compartments). Carotid artery clamping results in a variable decrease in cerebrovascular hemoglobin oxygen saturation in the majority of patients undergoing CEA.[21] Declines in regional cerebral oxygenation correlate variably with decreases in evoked potential amplitude during CEA.[22,23,24,25] Specific regional cerebral saturation threshold values defining critical cerebral ischemia have not been definitively established and the specific role of this monitor compared to other methods for cerebral ischemia detection remains to be defined.

Cerebral and Myocardial Ischemia

Hyperventilation has been proposed to redistribute blood flow from normal areas of the brain with preserved CO_2 reactivity to ischemic areas in which CO_2 reactivity has been lost, but controlled studies have not identified any benefit

attributable to an "inverse steal".[26] Available data do not support reduction of $PaCO_2$ as a routine intervention to reduce cerebral injury and normocapnia seems to be most appropriate during CEA in most situations.

Cerebral ischemia during carotid clamping can be reduced with the use of a carotid shunt, although to optimize benefit the shunt must be functional within 2-4 min without dissection or embolization. Even functioning shunts do not guarantee adequacy of cerebral perfusion, and there are markedly variable flow rates for different types of shunts. For example, the flow through a long Inahara-Pruitt shunt is about half that through a Javid shunt under similar conditions.[27] Of course, hypotension and low cardiac output compound such flow discrepancies and may be associated with decreased cerebral perfusion despite shunt patency.

Most practitioners advocate maintenance of BP close to the preoperative level, while some recommend a BP of 10-20% above normal. The rationale for maintaining normal or mildly increased systemic BP during CEA is based upon three concerns: (1) the normally occurring reduction in cerebral perfusion pressure in boundary zones between principle vascular territories, (2) the increased vulnerability of these areas to declines in BP if intracranial occlusive disease or cerebral infarction are present and (3) alteration of normal autoregulation in the presence of volatile anesthetics or chronic hypertension. Definite neurologic benefits of intraoperative "hypertension" have not been documented, although some concern has been raised about potential myocardial risks. Smith et al[28] showed that TEE diagnosed myocardial ischemia (identified as new SWMA) occur frequently during CEA when phenylephrine is administered to support BP with moderately deep levels of inhaled anesthesia. These changes may be related to changes in ventricular loading conditions when a pure alpha-1 agonist is administered in the presence of a volatile anesthetic with negative inotropic effects, resulting in altered regional wall motion and overdiagnosis of ischemia. Mutch et al[29] found no evidence for Holter monitored ischemia when phenylephrine was infused to support MAP at 110 ± 10% of ward values during carotid artery clamping. However, Holter diagnosed myocardial ischemia that is prolonged and occurring during carotid artery clamping or within two hours following declamping is highly predictive of adverse cardiac complications.[30]

During CEA, myocardial ischemia can occur in close association with marked fluctuations in BP partially related to carotid baroreceptor de-activation (during clamping) and reactivation (after declamping).[30] While intraoperative carotid sinus infiltration with local anesthetic has been recommended to reduce such hemodynamic fluctuation, this approach is associated with a greater frequency of intraoperative and postoperative hypertension.[31,32] The extent of surgical or pharmacologic denervation of the carotid sinus during CEA is likely an important determinant of post-clamp hemodynamic responses. Perioperative hypertension has a multifactorial etiology, is dependent on adequacy of preoperative BP control as well as presence of peripheral vascular disease, and may be impacted significantly by choice of anesthesia.[33] Hemodynamic instability with episodes of tachycardia and hypertension upon awakening/ tracheal extubation after CEA are associated with myocardial ischemia.[29]

Choice of Anesthesia

Debate over choice of RA versus general anesthesia (GA) persists because of differing conclusions of various studies of risks and benefits. The main advantage of RA is the ability to predict cerebral ischemia after carotid artery clamping, although various retrospective analyses have not been able to identify a clear difference in stroke or mortality rate between GA and RA.[34,35] Uncontrolled retrospective studies have suggested that carotid artery shunting is required less frequently with RA,[34,36] and that there is a lower incidence of postoperative hemodynamic instability[33,36,37] as well as a shorter duration of postoperative hospital stay with RA.[34,38]

However, other retrospective analyses have found no difference in cardiovascular outcome or hospital stay after CEA regardless of anesthetic technique.[35] Interestingly, one prospective investigation found RA for CEA to be associated with a high incidence of tachycardia.[39] A retrospective review of GA versus RA for CEA found a greater incidence of ventricular arrhythmias with GA but other adverse cardiac and neurologic events occurred with similar frequency between the two techniques.[40] Another retrospective analysis of over 1,000 CEA (2/3 with cervical block) could not identify any difference in cardiac complication rates between GA and RA.[41] The latter retrospective study did however report a lower stroke rate after CEA with RA (1.3%) compared with GA (3.2%).[41] Most studies evaluating the influence of choice of anesthesia on MI after CEA are not prospective and have screened for MI on the basis of clinical symptoms only, so that the question of whether there are differences in true rates of adverse cardiac outcome remains unresolved. No carefully controlled randomized trial has been conducted to identify whether any definite cardiac or neurologic outcome difference exists with RA vs. GA for CEA.

Opponents of RA for CEA are often concerned about its finite "failure rate", defined as need for conversion to GA. This may be reduced by supplemental infiltration with local anesthetic by the surgeon. A major factor for success of RA for CEA is gentle handling of tissues by the surgeon, as well as appropriate and frequent patient communication during the procedure. Success of RA is also improved with infiltration of local anesthetic at the ramus and lower border of the mandible. Even with these qualifiers, RA is not ideal for patients with expected long operative time or difficult vascular anatomy, especially a more cephalad carotid

bifurcation or high carotid plaque requiring vigorous submandibular retraction. In addition, unsatisfactory conditions may become manifest with RA in patients with short necks presenting difficulty in surgical exposure. Intraoperative mandibular nerve block can relieve discomfort associated with forceful or prolonged retraction on the mandible. Patients who become uncomfortable or restless may require airway intervention under physically awkward conditions, and the clinician must be ready to deal with this circumstance whenever embarking on RA for CEA. RA and GA are both acceptable options for CEA, and the decision to use one or the other technique should depend on the combined desires and experience of the anesthesiologist and surgeon as well as patient preference. With regard to RA techniques, a prospective, randomized comparison of deep vs superficial cervical plexus block (CPB) for CEA found no differences in patient satisfaction or intraoperative conditions, although deep CPB resulted in later onset of postoperative discomfort and less likelihood of requiring analgesia in the first 24 hr after CEA.[42]

Most anesthetic agents commonly used for induction and/or maintenance of GA decrease cerebral metabolism, although it is likely that any neuroprotective effect of anesthetics is more related to complex biochemical effects on ischemic brain tissue than simply to reduction of cerebral metabolism. While isoflurane has been associated with fewer EEG changes during carotid clamping[43] and with a lower critical regional cerebral blood flow[44] when compared with older volatile agents such as halothane or enflurane, retrospective comparison of these three anesthetics could not identify any difference either in neurologic outcome[43] or in cardiac outcome after CEA.[45]

Hemodynamic stability during GA for CEA can be enhanced with moderate amounts of opioids such as fentanyl or its derivatives, although care must be exercised to avoid doses that compromise rapid emergence at the end of the procedure. Judicious administration of beta blockers is also useful to minimize surges in heart rate and BP during stressful intraoperative periods and perioperative beta-blockade may have beneficial effects on cardiac outcome. Alpha-2 agonists are also useful to attenuate adverse hemodynamic responses during CEA.

"Minimally Invasive" Carotid Artery Revascularization

"Minimally invasive" techniques for management of carotid artery stenosis present considerable challenges for the anesthesiologist. Percutaneous interventions for carotid angioplasty and stenting may be performed either via the femoral artery approach or (less commonly) via direct puncture of the common carotid artery. Sedation is usually sufficient for groin cannulation, with the patient awake during carotid balloon inflation. Anticholinergic agents (atropine or glycopyrrolate) are administered to attenuate the baroreceptor response during balloon dilation or stent deployment. General anesthesia with short acting agents is more commonly performed when direct carotid artery puncture is utilized, with prompt awakening at the end of the procedure being desirable to facilitate early neurologic evaluation. Particular vigilance to monitoring hemodynamic and neurologic status is required during these procedures, especially during balloon inflations and after sheath removal for the cervical approach. Control of hypertension is particularly important since it increases the risk of hematoma formation, a potential catastrophic event, especially if residual anticoagulation is present. Maintenance of adequate perfusion pressure is particularly important to facilitate collateral blood flow during balloon dilation. Although CEA is currently the "gold standard" therapy for carotid artery stenosis, experience with endovascular techniques suggests complication rates comparable to CEA, with carotid patency even after six year follow-up.[46]

Postoperative Considerations

Postoperative Neurologic Dysfunction

Perioperative stroke after CEA has many causes, with more than half of surgical etiology (ischemia during carotid clamping, postoperative thrombosis and embolism), and the remainder due to other factors such as reperfusion injury, intracranial hemorrhage or other postoperative events. Embolization is the most common cause of strokes developing intraoperatively and is invariably associated with surgical events. It is estimated that only approximately 20% of strokes due to intraoperative events are hemodynamic in origin (related to carotid clamping, intracranial occlusive disease, shunt problems). Nonetheless hemodynamic strokes may follow a critical reduction in boundary zone perfusion secondary to intracranial occlusive disease or in areas around old cerebral infarcts, where apparently innocuous reductions in cerebral perfusion pressure may result in adverse outcome. After CEA, strokes tend to be related to embolization and/or thrombosis although intracerebral hemorrhage may also occur. Early thrombosis may be related to intimal injury and/or flap formation as well as enhanced platelet activation/deposition at the operative site. Neurologic events occurring during CEA are not necessarily predictive of postoperative complications, which may explain the variable findings of studies examining the effects of intraoperative interventions on overall stroke incidence.

Postoperative Hyperperfusion Syndrome

Postoperative hyperperfusion syndrome describes an abrupt increase in blood flow with loss of autoregulation in surgically reperfused brain. Patients with severe hypertension after CEA are at increased risk of developing this syndrome which may present with a spectrum of findings including headache, signs of transient cerebral ischemia, seizures, brain edema and even intracerebral hemorrhage.[47] Middle cerebral artery blood flow has been shown to be

pressure dependent in patients with post-CEA hyperperfusion (consistent with defective autoregulation) and systemic BP should therefore be controlled meticulously in the immediate recovery period after CEA, especially when there was a large pressure gradient across a severe CAS preoperatively.[48]

Blood Pressure Lability

Before CEA, carotid sinus baroreceptors may reset secondary to proximal arterial occlusion. After CEA, the reset baroreceptor may sense sudden increases in BP, triggering subsequent baroreceptor mediated hypotension. As noted above, although anesthetizing the carotid sinus nerve can improve hemodynamic stability during CEA, this practice as well as surgical denervation of the carotid sinus compounds the risk of postoperative hypertension, especially in patients with significant preoperative hypertension.

Cranial Nerve and Carotid Body Dysfunction

Transient dysfunction of adjacent cranial nerves and their branches may occur despite gentle dissection and retraction during CEA. Injury to the superior laryngeal nerve can occur and primarily results in mild relaxation of the ipsilateral vocal cord manifested by early fatigability of the voice and impairment of high-pitched phonation. Recurrent laryngeal nerve (RLN) dysfunction after CEA may result in paralysis of the ipsilateral vocal cord in the paramedian position, hoarseness and impairment of the cough mechanism. If RLN injury occurs and contralateral CEA is planned, consideration should be given to postponing the subsequent operation until satisfactory RLN function returns, or at least precautions for postoperative airway management should be planned if operation cannot be delayed. Although bilateral CEA is known to result in carotid body dysfunction and increases in resting $PaCO_2$, carotid body function can be abnormal even after unilateral CEA, with impaired ventilatory response to mild hypoxemia.

Airway and Ventilation Problems

Upper airway obstruction after CEA is a rare but potentially fatal complication which may occur not only because of hematoma formation but more commonly because of tissue edema, secondary to venous and lymphatic congestion. This diffuse type of neck edema (lateral and retropharyngeal) may be associated with markedly edematous supraglottic mucosal folds. Although such edema has also been postulated to be the effect of tissue trauma with increased capillary permeability induced by release of vasoactive mediators, steroid administration immediately prior to CEA does not reduce edema formation.[49] The presence of supraglottic edema after CEA may make intubation and mask ventilation difficult. Awake intubation may be necessary and opening the neck wound may not be of benefit in this situation.

Phrenic nerve paresis is common after cervical plexus block. While this normally has little clinical consequence (except for mildly increased $PaCO_2$), it is a potentially more serious problem in some patients with severe pulmonary disease or with preexisting contralateral diaphragm dysfunction.

Fast Track Recovery

Most patients undergoing CEA have historically been hospitalized for a few days, commonly with overnight observation in an ICU to treat BP and monitor for neurologic deficits or cervical swelling. As with other surgical procedures, fast-track recovery without routine ICU admission and with early home discharge is being applied to patients after CEA. The feasibility and safety of fast-tracking protocols has been examined and the majority of patients do not require ICU admission and may be safely discharged home 1-2 days after CEA.[50] Acute postoperative neurologic events usually occur early in the postoperative course,[51] which allows timely identification of patients who specifically require overnight ICU admission.

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