ANESTHETIC MANAGEMENT FOR CAROTID ENDARTERECTOMY

A Review

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INTRODUCTION

Stroke is a major healthcare problem and is the third leading cause of death in the United States. Over 500,00 strokes occurring annually have a mortality of 40%. Atherosclerosis involving the carotid bifurcation with thromboembolism causes ischemic infarcts.

Carotid endarterectomy (CEA) have shown to reduce the incidence of stroke from 75% to 85% when performed in symptomatic patients. A recent study of North American Symptomatic Carotid Endarterectomy trial showed a reduction of 50% in selected patients.

About 100,000 CEA are performed in the United States. The anesthetic management contributes to the success of this operation. This review discusses the pathophysiology, diagnosis and anesthetic management of a patient for CEA.

Carotid endarterectomy (CEA) is one of the most commonly performed surgical procedures worldwide. Since 1954, when the carotid endarterectomy was reported in the literature for the first time the number of these operations are increasing each year. It is evident that as the survival rate in the population increases, the disease will be diagnosed in even greater numbers and thus requiring surgical treatment. As the disease has preoperative and postoperative morbidity and mortality it is important to understand the pathophysiology of carotid artery stenosis. One of the risk factors for developing the disease is age of older than 70 years[1]. Although women may have severe disease, men are predominantly afflicted. History of smoking, hypertension and diabetes mellitis is prevalent in patients with carotid artery disease. These patients also are likely to be suffering from peripheral vascular disease and coronary artery disease (CAD) especially of the left main coronary artery.[2] The patients, with carotid artery disease, may present with asymptomatic bruit in the neck on a routine physical examination. Asymptomatic carotid bruits are more common in patients with vascular disease than in the general population over the age of forty years and 50% of these bruits are associated with underlying CAD[3]. These patients have a higher rate of future neurological events than the general population[4].

Transient Ischemic attacks (TIA) causing episodes of weakness of extremities with or without paraesthesia and speech defects are another common presentations. These attacks are reversible usually within 24 to 48 hours. Complete strokes also is a presentation in which neurologic deficit does not reverse in 24-48 hours but becomes stable and may never resolve. The causes of the symptoms are emboli causing cerebral hypoperfusion. These emboli originate from the atheromatous plaques and are lodged in the cerebral circulation.

Indications for CEA are well accepted and include symptomatic patients with previous history of TIA, reversible ischemic neurologic defect (RIND), or mild stroke with in the past six months and carotid artery stenosis (CAS) of more than 70%. A randomized clinical trial show that patients with asymptomatic carotid artery stenosis of more than 60% treated with aspirin and CEA have a reduced five year risk of fatal and nonfatal strokes compared to aspirin alone[5]. The operative procedure of CEA has a combined stroke-mortality rate of less than 3% in a neurologically asymptomatic patient and less than 5% for symptomatic patients and less then 7% for patient with prior strokes. Several factors appear to be associated with increase in morbidity and

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mortality after CEA. McCrory et al. Described that the presence of one or more risk factors were associated with nearly two fold increase in postoperative strokes, myocardial infarction or death. The risk factor includes age of 75 or older, preoperative neurological symptoms, severe hypertension (diastolic blood pressure of greater than 110mm/Hg), angina, CEA prior to coronary artery bypass, evidence of internal carotid thrombus and internal CAS near the Carotid Siphon[7]. Adverse outcomes were also more common in patients with complete contralateral carotid occlusion, ipsilateral intraluminal thrombus, or ipsilateral carotid siphon stenosis and the experience of the surgeon performing the procedure[8]. This review will describe the anesthetic management of patient presenting for CEA.

PREOPERATIVE EVALUATION

There are many methods to evaluate the extent of carotid artery disease. They are as follows:

1. Auscultation
2. Carotid Angiography
3. Orbital direction doppler ultrasonography
4. Oculoplethysmography
5. Phonoangiography
6. Continuous wave doppler ultrasonography
7. B-mode ultrasonography
8. Duplex scan
9. CT
10. MR-contrasted enhanced carotid magnetic resonance angiography

As the limitations of simple auscultation are well known, carotid angiography is performed on all surgical candidates. The next three methods in the list above, measure pressure and flow in branches of internal carotid artery such as the ophthalmic artery. These methods have been largely replaced by simpler techniques. Ultrasonography and duplex scans have become very popular and easy to perform. Contrast enhanced magnetic resonance angiography is less expensive and has fewer risks than conventional angiography. This method will replace conventional angiography before an endarterectomy. Most patients undergoing CEA are elderly and hypertensive with generalised arteriosclerosis. The list of commonly associated medical problems in patients with carotid stenosis include, male sex, coronary artery disease (CAD), abnormal EKG (71%), angina or myocardial infarction (24%), congestive heart failure (12%), coronary stenosis by angiography (40-70%), hypertension (50-70), smoking (90%), diabetes mellitus (20-40%), renal disease and hyperlipidemia (9%). Coexistent coronary artery disease (CAD) remains a major cause of morbidity and mortality after CEA and assessment of the severity of CAD is an important aspect of preoperative evaluation before CEA. It has been found that silent CAD did not effect preoperative outcome but strongly influenced long term prognosis. [10,11] Preoperative testing to assess coronary risk should be based on the presence of clinical markers (major, intermediate or minor predictors), functional capacity and the surgical procedure specific cardiac risk (stratified as high, intermediate and low risk). CEA is typically considered an intermediate risk procedure. [12] Specific preoperative cardiac testing is generally not indicated in these setting in the absence of major clinical predictors of increased preoperative cardiovascular risk (unstable coronary syndromes such as recent MI with evidence of important ischemic risk and unstable or severe angina, decompensated congestive heart failure, significant arrhythmias or severe valvular disease).

There is considerable debate on how to approach patients with combined significant coronary and carotid artery disease. Because the risk of stroke is significant in patients undergoing CABG with coexistent carotid stenosis, a staged or combined CEA and CABG has been advocated. It is now a common recommendation that sequential operation be performed when one lesion is more problematic. For example perform CEA first in patients with more stable CAD but in patients, with symptomatic carotid artery stenosis CEA be done first followed by CABG. Patients with symptomatic coronary artery disease require CABG first followed by CEA and combine CABG/CEA for patients with both symptomatic severe CAD and CAS.

INTRAOPERATIVE MANAGEMENT

The two major goals of intraoperative management for CEA are, to protect the brain and the heart. Maintaining adequacy of cerebral perfusion pressure, continual adjustments of cardiovascular parameters and monitoring the patient appropriately to facilitate prompt interventions to reduce the risk of potential adverse neurologic or cardiovascular events are the essentials of anesthetic management.

CEREBRAL MONITORING

There are number of methods available for intraoperative neurological monitoring, however the ideal methods of monitoring cerebral perfusion pressure during CEA remains controversial. Available method to assess the need for carotid artery shunting during CEA include, xenon blood flow study, transcranial doppler ultrasonography (TCD), somatosensory evoked potencies (SSEP), electroencephalography (EEG) and continuous clinical neurological examination under regional anesthesia.

EEG and continuous clinical neurological examination under regional anesthesia are the most commonly utilized and the review of the current literature would indicate that they are probably the better monitors of the adequacy of cerebral perfusion than carotid artery stump pressure alone.[13] Although lower carotid artery stump pressure is generally associated with a greater risk of ischemic EEG changes, it is neither sensitive nor specific to serve as a guide to selective carotid shunting during CEA.[14]

Neurologic testing during CEA in the awake patient under regional anesthesia is generally accepted as one of the most sensitive monitors of cerebral function and can reveal clinically significant cerebral ischemia despite unchanged EEG. This can potentially occur when the ischemic insult is located within the deeper brain structures and when preexisting electrophysiological abnormalities due to previous cerebral infarct make it difficult to identify superimposed new abnormalities.[15,16] Xenon washout is a well established technique of measuring regional corrected blood flow (CBF). Radiolabeled xenon gas is injected into the internal carotid artery by the surgeon and probes on the scalp measure xenon washout. Measurements are made before, during and after carotid artery cross clamping. Inadequate regional blood flow provides inducement for shunt placement. Evoked potential have also been used to assess the adequacy of cerebral perfusion during cross clamping. Significant changes are assessed by the magnitude of decrease in amplitude or increase in latency of the waveforms.[17]

The 16 to 20 lead unprocessed EEG is still the gold standard for monitoring cerebral perfusion during CEA. A number of Mayo Clinic studies performed have shown good correlation between EEG changes of ischemia and cerebral blood flow measurements, and they conclude that all new neurologic deficits during CEA can be identified. [18] Other have questioned the sensitivity of EEG monitoring in detecting intraoperative cerebral ischemia in patients who have a preexisting neurologic deficit. [19] Furthermore, another retrospective review of 458 CEA's found that 7 out of 10 patients who developed immediate postoperative TIA's failed to show EEG changes from baseline at completion of the procedure. These findings suggest that EEG may not be sensitive to all episodes of focal cerebral ischemia accompanying CEA.

Typical changes in the EEG during CEA, which portray a developing neurologic deficit, include unilateral loss of high frequency activity and the appearance of high amplitude delta waves. These changes correlate well with cerebral blood flow (CBF) measurements. Flow of less than 18ml/100g/min (critical CBF) as measured experimentally with the xenon washout technique.[17] As cerebral hypoxia worsens, both frequency and amplitude may diminishes and ultimately electrical silence may ensure.[17] The unprocessed EEG requires close observation and expertise in its interpretation. The processed EEG is simplified for interpretation by the use of different colors, peaks and valleys, lines or dots. The density spectral array (DSA) is easily utilized to assess cerebral perfusion during CEA. Rampil, et al. found that a decrease in EEG frequency of greater than 50% lasting longer than 10 minutes carried a 100% predictive value for postoperative strokes in patients without previous strokes.[20] Postoperative neurologic deficits

could not be predicted with the DSA monitoring in patients who had sustained a previous stroke. These monitors are of limited value in patients with previous CVA unless dramatic and long lasting ischemic events occur. Processed EEGs should be used as adjunct to other monitoring techniques and should not be relied upon exclusively for determination of adequate cerebral perfusion. These monitors are fairly insensitive to ischemic changes of small to moderate size.

Transcranial Doppler (TCD) ultrasonography applied across the 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. TCD may indicate which patients should have aggressive hemodynamic interventions and/or be anticoagulated as cerebral embolic events and decreased cerebral blood flow velocity can be differentiated.

CEREBRAL PROTECTION

Ischemia during the cross clamping of the carotid artery can be reduced with the use of a carotid artery shunt. During carotid occlusion the blood flow to the isolated area will depend on blood flow through the Circle of Willis if shunt is not used. If the carotid stenosis has increased gradually before performing the CEA, collateral from the Circle of Willis will have had time to develop and cerebral circulation may not be compromised by carotid cross clamping. However, if collateral flow is compromised because of occlusive disease of the contralateral carotid artery and/or the vertebral arteries the chances that marked hypoperfusion of the brain may occur during carotid clamping are even greater. Patients with additional contralateral disease have higher risk of perioperative stroke than the patients with ipsilateral disease. Even functioning shunts do not guarantee of adequacy of cerebral perfusion.

Blood pressure management is important and it is advisable to maintain blood pressure to as close to patient's preoperative BP as possible or mildly increased up to 20%. The rationale is based upon the following;

1. The normally occurring reduction in cerebral perfusion pressure in boundary zones between principle vascular territories.
2. The increase vulnerability of these areas to decline in BP in the presence of intracerebral occlusive disease or cerebral infarct.
3. Alteration of normal autoregulation secondary to the effects of volatile anesthetic agent or chronic hypertension. Flow in these areas is believed to be pressure dependent [21,22,23] Under these circumstances prolonged severe hypotension may jeopardize cerebral function. Pharmacological augmentation of BP with sympathomimetic drugs is not with out risk to the heart. Smith et al. showed that indiscriminate use of phenylephrine to increase BP during deep general anesthesia with a volatile agent increases the incidence of intraoperative segmental wall motion abnormality (SWMA) detected by transesophegeal echocardiography as compared with light anesthesia with out the use of phenylephrine.[24] More recently Mutch et al. found no evidence for holter monitored ischemia when phenylephrine was used to support mean arterial pressure at 110 + 10% of preoperative values during cross clamping of the internal carotid artery.[25] During CEA, episodes of ischemia have close association with marked fluctuation of BP and surgical manipulation of the carotid sinus.[26] Such alterations in BP are likely related to deactivation (clamping) and reactivation (declamping) of the carotid sinus baroreceptors.[27, 28] Carotid sinus infiltration with lidocaine is recommended to reduce these hemodynamic fluctuations.[29,30] However, carotid sinus area infiltration with bupivacaine after CEA does not reduce the incidence of postoperative hypotension but in fact may be associated with a greater frequency of postoperative hypertension.

Intraoperative and post operative hypertension has a multifactorial etiology and is also dependent on adequacy of preoperative blood pressure control and presence of peripheral vascular disease

and may also be influenced by the choice of anesthesia, [30, 31, 32] Hemodynamic instability during emergence from general anesthesia after CEA may be associated with myocardial ischemia. [25] Episodes of tachycardia and hypertension upon awakening and tracheal extubation after CEA are also associated with onset of myocardial ischemia.

VENTILATORY MANAGEMENT

While hyperventilation redistributes blood flow from normal areas of the brain with preserved CO_2 reactivity to ischemic areas in which CO_2 reactivity has been lost, controlled studies of the effect of hyperventilation on ischaemic cerebral injury have not identified any benefit attributable to an "inverse steal" [33-35]. Available data does not support reduction of $PaCO_2$ as a routine intervention to reduce cerebral injury and therefore normocapnia seems to be most appropriate during CEA.

TEMPERATURE MANAGEMENT

Another important aspect of preoperative management of patients with CEA is maintenance of normothermia. A randomized controlled trial comparing "routine" thermal care (mean end of case core temperature 35.4°C) in patients with CAD or at high risk for CAD undergoing noncardiac surgery found that maintenance of normothermia was associated with a 55% reduction in risk of serious morbid cardiac events (unstable angina/ischemia, cardiac arrest or MI).

CHOICE OF ANESTHESIA

Debate over choice of regional anesthesia or general anesthesia continues because of differing conclusions of various studies of risk and benefits,

REGIONAL ANESTHESIA

Advantages of regional anesthesia include:

1. Continuos monitoring of neurologic function.
2. Delineate the minority of patients who require shunting during internal carotid artery (ICA) clamping.
3. Hemodynamic stability associated with lower incidence of perioperative MI.
4. Built in pain management for initial post operative period but without the sedation, which might interfere with immediate evaluation of neurologic status.
5. Shorter length of stay in the hospital.

DISADVANTAGES OF REGIONAL ANESTHESIA ARE

1. Difficulty in securing control of the airway during an intraoperative emergency.
2. Patient discomfort during 2-4 hours of surgery because of minimal sedation, monitoring for subtle, early signs of cerebral ischemia require that verbal communication is maintained. Squeezing the hand only confirms that patient has not suffered a major neurologic event.
3. Complications and side effects of deep cervical plexus block are: Ispsilateral hemidiaphragm paralysis resulting in 30% decrease in vital capacity, intravascular injection, total spinal anesthesia, cervical epidural anesthesia, hoarseness and high rate of block failure (20%).

Strategy for Regional Anesthesia

1. Premedicate with good preoperative interview.
2. Carefully monitor cardiac status using lead II and V5. All patients will require an arterial line for BP monitoring and a large bore IV canula.
3. Neurologic monitoring, a "rubber duck" (toy which makes noise when squeezed) is placed in the patients contralateral hand and squeezed on demand repeatedly during the procedure. Also continuous verbal contact is maintained to assess cerebral function reversible. Light sedation with propofol and analgesia with fentanyl can be used as these will not interfere in the monitoring.

METHODS FOR REGIONAL ANESTHESIA

1. Local infiltration
2. Combined superficial and deep cervical plexus blocks.
3. Interscalene cervical plexus block (CA)

For a deep cervical plexus block the transverse process of C6 is located by extending a line directly posterior from the Cricoid cartilage. Another line is drawn

between the transverse process of C6 and the mastoid process above. The transverse process of the 2nd to 5th cervical vertebrae are located along this line. A block needle is inserted perpendicular to the skin at the level of C2, C3 and C4, directed somewhat caudally to avoid sliding up the gutter of the transverse process end causing a subarchnoid injection.[39] Paraesthesias are elicited at each transverse process and 3 ml of 1% lidocaine with epinephrine is injected. Alternatively 10 ml of local anesthetic can be infiltrated if no paraesthesias are elicited. Complications of a deep cervical plexus block include intravascular injection, subarachnoid injection and block of the vagus nerve and Horner's syndrome.[39] A superficial cervical plexus block, performed by infiltrating the posterior lateral border of the sternocleidomastoid muscle in a fan like manner, may accompany the deep cervical plexus block to provide cutaneous analgesia. Some recommend the interscalene approach to the cervical plexus as an alternate to provide analgesia to the neck and shoulder. This method circumvents the need to elicit numerous paraesthesias.[40] A single injection of 10-15 ml of 0.5% bupivacaine is injected into the interscalene groove at the level of C4.

GENERAL ANESTHESIA

It is used in most of the centers and its advantages are:

1. Built in control of airway control with endotracheal intubation
2. Patient comfort maximized intraoperatively.
3. $PaCO_2$ is easy to control although benefits of hypocapnia or hypercapnia are in doubt to maintain normocarbia.
4. Certain agents produce reduction in cerebral metabolic requirements although benefits are unproven.
5. Facilitates use of intraoperative transesophageal echocardiography (TEE) to monitor cardiac status.

DISADVANTAGES ARE

1. Difficulty in maintaining smooth hemodynamics. Increased incidence of MI compared to regional anesthesia.
2. No currently available monitoring system for cerebral ischemia during clamping is as reliable as the continuous evaluation of the neurologic status of the awake patient.
3. General anesthesia tends to involve greater use of shunts, with their associated morbidity.
4. Postoperative pain management is more difficult due to conflict between the need for patient comfort required for hemodynamic stability and the need for mental alertness required for neurologic assessment.

MONITORING

Intraoperative monitoring of patients scheduled for CEA should include basic ASA monitoring. An intra-arterial catheter for blood pressure monitoring allows detection of beat-to-beat changes in pressure to facilitate prompt treatment. Lead II and V5 of the EKG for ST-T segment changes. Automated ST-T segment analysis allows easier detection of myocardial ischemia.41 EKG monitoring detect almost 60% of all myocardial ischemic events in vascular patients if systolic wall motion abnormality is considered the gold standard for myocardial ischemia.[42]

It is rarely necessary to use pulmonary artery catheter in CEA surgery, for increase in pulmonary capillary wedge pressure may not be as sensitive indicator as once thought.[43]

Most commonly used anesthetic agents for induction and/or maintenance of general anesthesia 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 the reduction of cerebral metabloism.[43]

While isoflurane has been associated with fewer EEG changes during carotid clamping when compared with older volatile agents such as halothane or enflurane and with a lower critical regional cerebral blood flow then reported for halothane or enflurane[44,45]. Retrospective comparison of these three anesthetics could not identify any difference either in neurologic outcome or in cardiac morbidity after CEA[44,45,46]. However newer anesthetic agents like sevoflurane and desflurane are now used commonly and no single agents has shown specific advantages.

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 which result in serum levels at the end of the procedure which will compromise rapid emergence. Remifentanil, the newest opioid, offers significant pharmacokinetic advantage in this regard, since it can be administered at doses, which will be effective to control hemodynamic aberrancies at the end of CEA yet not inhibit prompt recovery. Judicious administration of beta blockers is also useful to minimize surges in heart rate and blood pressure during stressful intraoperative period and perioperative beta-blockade have beneficial effects on cardiac outcome in vascular surgical patients. [47,48] Alpha-2 agonists are also useful to attenuate adverse hemodynamic responses during CEA.[49] Reduced hemodynamic changes and control of coughing or straining upon awakening from general anesthesia can be achieved with lidocaine instilled intratracheally, without the prolongation of wake-up time seen with intravenous lidocaine.[50] The patient should be awake and extubated while on the table so as to assess the neurological status.

POSTOPERATIVE CONSIDERATIONS

Postoperative Neurological Dysfunction

The efficacy of CEA for prevention of stroke has been demonstrated for both symptomatic as well as asymptomatic patients. Perioperative stroke remains an important complication of CEA. Majority of strokes after CEA have surgical etiology (ischemia during carotid clamping, postoperative thrombosis and embolism), with remainder due to other factors such as reperfusion injury, international hemorrhage or other postoperative events.[51] The incidence of neurologic complications occurring during regional anaesthesia is reported to be between 10-25%, which is similar to the incidence of ischemic EEG changes observed during general anaesthesia. Although previous data suggest a significant relationship of intraoperative ischemic events with post operative neurologic complications.[53] A recent study of 261 patients undergoing CEA with regional anaesthesia identified postoperative neurologic complication in 3.7% of patients experiencing neurologic deficit during surgery, which was not significantly different patients who were symptom free intraoperatively.[54]

Postoperative Hyperperfusion Syndrome: - describe an abrupt increase in blood flow with loss of autoregulation in surgical reperfused brain. Patients with severe hypertension after CEA are at increased risk of developing this syndrome, which may present with headache, signs of transient cerebral ischemia, seizures, brain edema and even intracereberal hemorrhage.[55] Systemic blood pressure should be controlled meticulously in the immediate recovery period after CEA.

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 pressure, triggering subsequent baroreceptor mediated systemic hypotension. Anesthetizing the carotid sinus can never improve the heart rate and blood pressure stability during carotid artery surgery although it appear that this practice, as well as surgically induce carotid sinus nerve paresis, compound the risk of postoperative hypertension, especially in patients with significant preoperative hypertension.

CRANIAL NERVE AND CAROTID BODY DYSFUNCTION

Transient postoperative dysfunction of adjacent cranial nerves and their branches may occur despite gentle dissection and retraction during CEA. Recurrent laryngeal nerve dysfunction has been reported to occur in 2% of cases.[56] It primarily results in early fatigability of the voice and impairment of high pitched phonation.

Although bilateral CEA is known to result in loss of carotid body function and increase in resting $PaCO_2$, carotid body function is abnormal even after unilateral CEA, resulting in impaired ventilatory response to mild hypoxemia.[57]

AIRWAY AND VENTILATION PROBLEMS

Upper airway obstruction after CEA is a rare but potentially fatal complication, which may occur because of haematoma formation, but more commonly because of tissue edema, possibly secondary to

venous and lymphatic congestion. This diffuse type of neck edema may be associated with markedly edematous supraglottic mucosal fold.[58] Although such edema has been postulated to be the effect of tissue trauma with increase capillary permeability induced by release of vasoactive mediators, steroid administration immediately prior to CEA does not reduce the edema formation. It is important to realise that the presence of such edema may make mask ventilation and intubation difficulties.

Phrenic nerve paresis is surprisingly common 60-70% in cervical plexus block.[59] While this normally has little clinical consequence, it represents a potentially more serious problem in patients with COPD or in patients with preexisting contralateral diaphragmatic dysfunction,

CONCLUSION

In experienced surgical hands, CEA effectively prevents stroke in symptomatic patients with serve (>70%) stenosis. The Anesthesiologist can choose from a variety of techniques for administration of anesthesia, to obtain acceptable results. Maintaining adequate cardiac performance and cerebral perfusion is more important than selecting technique. Continued vigilance, particularly in the first several hours after surgery, is crucial because complications, although uncommon, can be devastating.

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