The anesthetic management of patients undergoing carotid endarterectomy can be accomplished by one of three methods: local, regional or general anesthesia. Our goal should be choosing one which provides optimal operating conditions. Numerous factors which must be considered are the patient's welfare, comfort, control of $\text{PaO}_2$ and $\text{PaCO}_2$, and maintaining adequate cerebral blood flow to help prevent any neurological sequelae.

Cerebrovascular accidents (CVAs) or strokes are responsible for 200,000 deaths each year in the United States and rank third as a cause of death. Intracranial vascular disease had been considered to be the major cause of CVAs until the last 20 years. Advances in angiography, however, have since demonstrated that occlusion of extracranial arteries may account for as many as 80% of all strokes and that atherosclerosis is the major source of occlusion.

Blood is supplied to the brain via the two internal carotid arteries, which provide 85% of the arterial circulation, and the vertebral arteries, which provide the remaining 15%. In order to deprive the brain of sufficient blood supply, stenosis or narrowing of a single vessel must be at least 80% or be compounded by several successive narrowings or bilateral stenosis.

Over a period of time collateral circulation develops, primarily at the circle of Willis, to maintain cerebral perfusion despite decreased blood flow through stenotic vessels. Therefore, neurological deficits may vary considerably among individuals or may be absent, depending on the location and extent of stenosis as well as compensation from collateral circulation.

Patients who are most likely to benefit from a carotid endarterectomy procedure are those who have a history of transient ischemic attacks (TIAs), recent mild strokes, old strokes that have developed new symptoms, asymptomatic carotid bruits, and chronic cerebral ischemia. Contraindications include the presence of stroke in evolution, recent acute stroke, remote stroke with poor neurological recovery, concurrent intracranial vascular obstruction, and total occlusion of the internal carotid artery.

Controlling mechanisms of cerebral blood flow
Metabolic control. On a global level, the brain has a stable metabolic rate of oxygen and cerebral blood flow (CBF), but on a regional level the variations in brain activity produce patterns similar to those found in other organs. In the brain, an increase in the work produces a rise in the oxidative metabolism and blood flow. Changes in metabolism are usually parallel with changes in flow. Just how this occurs is still not clearly understood. Current theories hold that increased concentrations of $\text{H}^+$ and $\text{K}^+$ in the extracellular fluid (ECF) bath the arterioles of the brain. Blood flow to the brain is closely linked to metabolic or nerve cell activity.
An increase in cerebral metabolism and cerebral blood flow is present in seizure activity and when a patient experiences a painful stimulus. Light anesthesia is an example of this. A decreased metabolism and decreased cerebral blood flow can be exhibited in a coma patient or one who is in a state of barbiturate intoxication.

_Autoregulation_ is the ability of the vessels in the brain to maintain the blood flow at a constant level between the range of 60-150 mmHg arterial blood pressure, even when there are wide variates in systemic or cerebral arterial pressure. It is thought that this mechanism is probably a myogenic response of the smooth muscle cells of the arterial walls.

We should understand this mechanism because of the importance of the autoregulatory curve and its variations in the upper and lower limits of mean arterial blood pressure according to the patient’s condition or the type of surgery being done. When making reference to the upper limit (150 mmHg) and the lower limit (60 mmHg), we must remember that these apply to a normotensive patient.

In the chronic hypertensive patient, the autoregulatory curve is displaced to the right. It is usually through hypertrophy that the cerebral vessels have adjusted to the constant higher pressures. The upper limit of autoregulation is therefore increased in hypertensive patients. The normotensive patient would probably not tolerate an induced hypertensive episode. This would cause interference with the functions of the blood-brain barrier, its damage and the formation of edema.

The hypertensive patient is not able to tolerate the same lower limit of autoregulation or ischemic level that the normotensive patient can. This is a very important consideration in the hypertensive patient who is having an arterial-venous malformation repaired or an aneurysm clipped. Knowing that the patient’s lower limit of autoregulation is elevated, the anesthetist should discuss with the surgeon the level of blood pressure that is safe for warding against ischemia. Remember, too, that autoregulation can be influenced by disease and various drugs used in anesthesia.

_Chemical control._ The PaCO₂ and the PaO₂ are the primary factors which produce changes in the CBF. This is brought about by changes of the pH surrounding the smooth muscles of the cerebral arterioles.

Tissue acidosis, which can be produced from an ischemic episode, will cause a vasomotor paralysis from the dilatation of the cerebral vessels. This dilatation abolishes autoregulation, and therefore, cerebral perfusion becomes directly related to and dependent on cerebral perfusion pressure.

_Neurogenic control._ Cerebral arterioles are innervated by sympathetic and parasympathetic nerve fibers. The functional role of these autonomic fibers is poorly understood. Probably only 5-10% of the total control of CBF can be attributed to a neurogenic control.

Cerebral damage from carotid endarterectomy occurs as a result of dislodgement of an embolus from the stenotic site and by reduction of perfusion, either regional or hemispherical. This reduction may be caused by anesthetic management and/or surgical clamping or occlusion. Dislodgement of the emboli can occur from repeated palpation of neck vessels preoperatively and during surgical dissection and palpation of the carotid. Thus, it is through refinements in the surgical techniques, including the specific types of shunt equipment used, that a reduction in the incidence of intraoperative embolization can be achieved.

Reduction of the cerebral perfusion may be caused by hypotension. Depending on the severity of the occlusion in the stenotic area, hypotension may cause reduction of the cerebral perfusion by reducing the gradient across the stenotic area and/or by altering the efficiency of the circle of Willis, and other collaterals. Premedicant drugs, anesthesia and positioning are a few of the variants which could bring about a hypotensive episode.

In several articles, Boysen reported that temporary clamping of the carotid artery reduced regional CBF in most patients. The magnitude of this reduction was a mean of 41% in 17 patients. The flow reduction was greater during hypercapnia in comparison to hypo- or normo-capnia.

Reduction of carotid flow can be compensated for by use of a surgical bypass or shunting of the vessel during the actual surgery. Evaluation and manipulation of cerebral hemodynamics before, during, and after endarterectomy should be a combined effort of the surgeon and anesthesia personnel. It is in the best interest of the patient that this team shares a good line of communication, especially when decisions must be made concerning the upper and lower limits of the blood pressure which will provide the cerebral flow necessary to prevent a crisis.

Two methods of evaluating carotid flow are electroencephalogram (EEG) recording and measurement of the internal carotid artery pressure. Boysen and associates have suggested that stump pressures of 55-60 mmHg or above would provide reasonable assurance against cerebral ischemia. It was also shown that the stump pressure could be
raised proportionately with systemic mean arterial blood pressure. Even though a moderate level of induced hypertension is important during carotid clamping, it does not always prevent neurological damage in patients with stump pressure below 55 mmHg, suggesting poor collaterals.

Response of stump pressures to PaCO₂. An increase in PaCO₂ generally decreases internal carotid artery (ICA) stump pressure even though rCBF measurements show an increase in some patients and no change in others.

Hypercapnia may be very dangerous for those patients who have impaired collateral function in which a steal may occur. This would cause an increase in flow only to the unaffected hemisphere, causing what is called an interhemispheric steal. An intracerebral steal is possible with hypercapnia, thereby causing a decrease in flow to an already compromised area. This phenomenon is seen in patients with localized infarct or ischemia of recent origin. (These patients are not usually selected for a carotid endarterectomy.)

Reduction in stump pressure following carotid occlusion under hypercapnia has two etiological factors: (1) reduction in pressure in the circle of Willis because of an increase in flow to the opposite hemisphere and (2) increased flow resistance across the collateral channels with increasing flow rates through these vessels.

When comparing hypocapnia and normocapnia, the ICA stump pressures are about the same in patients with poor collaterals. The rCBF is higher, though, in normocapnia patients and cerebrovascular resistance is lower. It only stands to reason, then, that patients having a carotid endarterectomy procedure would be best at a normocapnia level. At our institution we are able to keep a close watch on the brain's activity by cerebral function monitoring, PaCO₂ levels and EEG. The EEG is a useful adjunct to carotid endarterectomies because it can indicate whether or not a temporary bypass shunt is necessary during the period of carotid clamping.

The human brain's tolerance of ischemia or flow reduction has not been exactly determined. One way of determining the adequacy of a measured rCBF value is to correlate it with the electrical activity of the brain, hence, the EEG. Studies have shown the relationship between CBF and cerebral cortical electrical activity (EEG) in animals established a critical value of 16-25 ml/100 gm/min, below which EEG activity becomes abnormal. Below 15 ml/100 gm/min, EEG activity is absent. The normal value in man is 50 ml/100 gm/min. More than 85% of patients can tolerate carotid occlusion by utilizing their own collateral channels.

**Anesthesia management**

A well-documented preoperative neurological assessment cannot be overemphasized. By being systematic in the assessment, the anesthetist can then also assess the postoperative neurological status quickly and thoroughly. The motor function can be determined by assessing muscle strength and tone bilaterally. Comparison of flexor-extension movement and hand grasps may demonstrate a weakness of hemiplegia.

The level of consciousness is the most important measure for mental status. Pupils should be checked for their size, equality and reactivity. Cranial nerve II, which is responsible for vision, can be assessed by having the patient count fingers. Cranial nerves VII, X, XI and XII should also be assessed preoperatively because intraoperative traction can disturb their functioning.

Successful management of these patients during anesthesia and operation demands that the whole team understand the scope and requirements of the operation and the possibility of complications. The main hazard of the procedure still is cerebral damage; thus, prevention is better than cure.

**Local techniques** may be used but they lack the cerebral protection that can be attained with a regional or general anesthetic. Local anesthesia is also not as well accepted by the patient. The baroreflex function, stimulated by manipulation or carotid occlusion, is usually more pronounced during a local anesthetic.

The intimate anatomic relationship between the carotid sinus and the surgical site poses two intraoperative implications. First, surgical manipulation is interpreted as stretch by the carotid sinus and may result in bradycardia and hypotension. Secondly, during carotid occlusion, the decreased pressure within the carotid bifurcation may cause tachycardia and hypertension.

The effects of these intraoperative baroreceptors may be modified through the following: (1) afferent block of 9th nerve by local anesthetic; (2) efferent block of vagus with atropine; or (3) blockade of the reflex arc with anesthetic agents.

**Regional anesthesia** is preferred by many to general anesthesia for carotid endarterectomies. The ideal regional technique is a combined superficial and deep cervical plexus block. Analgesia is usually adequate and muscle relaxation is satisfactory except when exposure of the upper portion...
of the ICA necessitates retraction of the lower jaw and associated structures.4

Advantages claimed for regional block anesthesia include the following:4

1. It provides a conscious and cooperative patient (whose neurological status is the best available monitor of cerebral blood flow and collateral function when carotid blood flow is occluded at the site of operation).
2. It is easy to recognize transient neurological deficits resulting from undetectable emboli dislodged during dissection which otherwise would likely become permanent subsequent to carotid clamping.
3. There is no race against time as there is under general anesthesia.
4. It is possible to avoid routine shunts and their complications in all patients who tolerate a test occlusion.
5. The possible cardiovascular depression of general anesthesia can be avoided.
6. A smooth transition from the operative to postoperative phase is possible without emergence phenomena.

Disadvantages of regional block anesthesia include the following:4

1. Complications of deep cervical plexus block such as intraarterial injection and subarachnoid injection are serious.
2. Minor complications such as block of adjacent nerves (hypoglossal, phrenic, recurrent laryngeal and facial) are inconvenient and occasionally dangerous, although transient.
3. Neck massage, if used to aid the spread of anesthetic agents (very much an unnecessary manipulation) may produce cerebral embolization.
4. Delayed intolerance to carotid occlusion is not unheard of; management is even more difficult than under general anesthesia.

Moreover, cervical plexus block anesthesia at its best is uncomfortable for some patients in the specific surgical position employed. Tranquilizers and sedatives, when employed to alleviate such discomfort, may defeat the whole idea of regional block.4

General anesthesia. We have seen that most patients undergoing a carotid endarterectomy are usually fair to poor anesthetic risks, usually ASA status 3 or 4. These elderly patients usually have a history that includes symptoms and signs of hypertension and/or atherosclerotic cardiovascular disease. In addition to their arterial disease, many have degrees of chronic obstructive pulmonary disease.

Another factor which plays a big part in the anesthetic management of these patients is their prior drug therapy which may include digitalis, diuretics, anticonvulsants, hypotensive agents, bronchodilators and possibly tranquilizers. The anesthetist can help regulate or at least control some of the factors which affect the CBF and the cerebral metabolic rate of O2 consumption (CMRO2). This includes blood pressure, depth of anesthesia, ventilation and PaCO2. It is through these variants that we can help maintain the cerebral oxygenation at a satisfactory level and prevent neurological sequelae.

Hypotension in these elderly patients is not tolerable and during the clamping of the carotid, it could be disastrous. Hypotension does not affect CBF when autoregulation is present but during clamping, autoregulation is likely to be lost. Hypertension during this period of surgery may help to improve the blood flow to the hemisphere in question. The usual agent of choice for blood pressure support in this particular type of surgery at our institution is a phenylephrine drip. The blood pressure should be raised approximately 20-30% to provide adequate cerebral circulation during the period of clamping.

In providing general anesthesia, our goal is to avoid hypoxia and hypotension. Deep anesthesia would hardly be tolerated by these patients, so it is the anesthetist's job to administer an anesthetic that will provide analgesia, amnesia and immobility with the best physiological supportive care possible. Muscle relaxants will provide the immobility and help with controlling the ventilation to the desired PaCO2 level, which as stated before should be normocapnic.

The anesthesia combination of nitrous oxide, Pentothal® drip, fentanyl and muscle relaxants produces only minimal physiological changes in cerebral hemodynamics. Barbiturates decrease the intracranial pressure and therefore result in an increase in the cerebral perfusion pressure which is beneficial in this type of surgery. Even more advantageous is the fact that with barbiturate anesthesia, the cerebral oxygen requirements are decreased and areas of the brain which are partially ischemic may be protected.

Positioning the patient should be accomplished with care. A head-up position with flexion of the table at the middle is probably best because venous congestion of the neck is eliminated. The patient's legs should be either wrapped with ace bandages, or Ted® stockings should be applied to prevent venous pooling. The patient's head will be facing towards the anesthetist, but excessive rotation of the neck may cause obstruction of the
vertebral artery flow. The anesthetist should pay careful attention to the patient's blood pressure and ECG tracing at this time.

Close monitoring of the patient during positioning and induction is essential. The blood pressure should not be allowed to fall below preoperative levels. If this occurs, it should be treated with colloids or by starting the phenylephrine drip. Anytime during the procedure that a change is noted in the ST segment of the ECG may be the first sign that the blood pressure is too low and not adequate for the perfusion needed to avoid neurological sequelae. If the blood pressure is too high, cardiac arrhythmias of ventricular origin may also be present. Maintaining a steady blood pressure is usually a tedious task and is by no means an easy one.

To aid and maintain cerebral oxygenation during carotid clamping, an increase in FIO$_2$ to 50% is beneficial for the patient.

Once the surgery is completed and the muscle relaxant is reversed, the patient is extubated only after spontaneous respirations are established and anesthesia personnel are certain that neurological deterioration is not present. The termination and emergence of anesthesia is just as important as the induction. Bradycardia, hypotension, hypertension, and extreme coughing or straining by the patient on the endotracheal tube should be avoided.

The patient should be transferred to the post-anesthesia recovery area (PAR) in a slight head-up position; his blood pressure should be constantly monitored by the arterial line. Complications of carotid endarterectomies already alluded to include hypotension, reflex bradycardia and cardiac arrhythmias. All of these factors reduce cerebral blood flow, thus, it is very important that prophylactic care be given.

Neurological damage is by far the most serious complication, and if present would probably be related to a reduced cerebral blood flow during anesthesia and surgery. Proper postoperative evaluation of the patient requires a neurological status which is at least equal to the preoperative status.

REFERENCES

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