Neurologic Complications of Cardiac Surgery

Rafael Llinas, Denise Barbut, and Louis R. Caplan

The average age of patients undergoing cardiac surgery and the number of comorbidities they possess will continue to increase as surgical technology advances. Toxic/metabolic encephalopathy, hemispheric strokes, hypoxic injury, and peripheral nerve lesions all can occur as a result of cardiac surgery. Therefore, an understanding of the neurologic risk, recognizable syndromes, and preventative measures will continue to be important. Careful preoperative assessment, operative risk factor reduction, and careful postoperative assessments and management may reduce the neurologic risk for cardiac surgery.

In 1953, Gibbon successfully performed the first open heart procedure on a patient. Since that time, the number of patients undergoing this procedure has increased and almost 400,000 coronary artery bypass graft (CABG) surgeries are performed annually. Impressive improvements in the techniques for CABG have increased its safety and reduced the rate of postprocedural complications. Improved medical treatments include better control of hypertension, the use of statins, and drugs that decrease platelet function. Now many patients survive who would have died in past decades. Angiographic-guided angioplasty with stenting has also improved survival rates. As a result of better medical treatments and improved surgical techniques, older patients now undergo surgery. The average age of patients and the number of comorbidities that patients have presenting to cardiac surgery is increasing. Nervous system complications are very common among elderly patients with high comorbidities.

The rate of complications from cardiac surgery varies widely and depends heavily on the thoroughness of postoperative neurologic evaluation. Rates of complication can range from 7% to 61% for transient neurologic deficits and 1.6% to 23% for permanent deficits. A prospective series showed the complication rates to be between 16.8% and 61%. Thus, a systematic approach to the evaluation of the neurologic complications caused by cardiac surgery should be used. We divide the complications by problems based on the nature of the consults we have received. Four common categories of neurologic complications are encephalopathy, stroke (embolization), cognitive dysfunction, and lesions of the peripheral nervous system.
Not Waking
A common call for neurologic consultation after any cardiac surgery is a patient who does not awaken soon after surgery. The differential diagnosis is broad.

Toxic/Metabolic Encephalopathy
Patients in this category have decreased alertness, even stupor, but careful neurologic examination does not show major focal motor, sensory, or visual abnormalities. This is a common scenario. This category is usually called an encephalopathy, a broad term referring to several different cognitive states including delirium, prolonged lethargy, confusion, disorientation, drowsiness, and change in behavior all without significant focal findings on neurologic examination or findings on neuro-imaging. In one series, 11.6% of postoperative patients were considered encephalopathic even 4 days after surgery. Infections involving the urine, lungs, or wounds are important causes of toxic-metabolic encephalopathies. Risk factors for prolonged encephalopathy after surgery are renal or hepatic disease, old age, previous strokes, and mild baseline dementia. Medications are the most important factor. The most common medications given that prolong poor recovery from anesthesia are anticholnergic drugs, sedative hypnotics, narcotic analgesics, and histamine-2 receptor blockers. Haloperidol or Haldol (McNeil Inc, Springhouse, PA) is probably the worst offender in the elderly, especially in patients with baseline cognitive problems. Not only does Haldol produce depressed alertness, stiffness, and lethargy, it can remain in the body for weeks. One study showed that Haldol could retard recovery from brain lesions such as stroke for weeks, even after a single dose. The use of sedatives and analgesic medications should be kept at an absolute minimum. Antipsychotic medications should not be used.

Large Hemispheric Strokes
Retrospective studies have shown that the rate of ischemic stroke in consecutive patients undergoing CABG is between 2% to 6%.5,6,10,11 Large hemispheric infarcts can result in a prolonged time to arouse from anesthesia. Depression of consciousness can occur from either right- or left-sided lesions. Typically, acute or subacute ischemic strokes present with focal neurologic deficits, but when lesions are in the prefrontal lobe, nondominant parietal lobe, or either occipital lobe, patients may have few localizing neurologic findings except for alteration in mental status that may translate into prolonged depressed alertness after surgery. Evaluation, including a careful neurologic examination, is necessary. The examining physician should be looking for evidence of (1) gaze deviation; (2) visual field abnormalities; (3) weakness; (4) reduced response to pain; and (5) asymmetric reflexes. Finally, brain imaging will be necessary in these patients because examination of an unarousible patient who is uncooperative with the examination can be difficult.

Recall that infarcts may not show on a computed tomography (CT) scan for 6 to 24 hours. Magnetic resonance imaging (MRI) can also be normal unless diffusion imaging (DWI) is included. DWI shows infarct signs soon after the onset of ischemia. Identification of large-hemisphere ischemic or hemorrhagic lesions is very important because often brain swelling develops approximately 24 to 72 hours after the event and can result in uncal hernation (more common in younger patients) and death if not diagnosed and treated properly. Causes for postoperative stroke are discussed later in this article. Recall that old strokes or neurologic problems will seem worse in patients recovering from sedation. Delayed recovery from anesthesia and new focal neurologic deficits not noted on examination before surgery are indications for brain imaging with CT or MRI.

Multifocal Infarctions
Prolonged, poor arousal can be caused by multiple small instead of single large hemispheric lesions. Studies have shown that up to 25% to 65% of strokes after CABG are bilateral or multiple.11-13 The diagnosis of multifocal infarctions can be difficult to make at the bedside. Multiple infarcts or hemorrhages involving bilateral frontal, temporal, or parietal lobes of the brain can cause diffuse brain dysfunction.

The diagnosis of multiple acute lesions from multiple emboli can be a difficult diagnosis based on examination alone because the neurologic
examination is best at evaluating asymmetries. In the case of multiple emboli resulting in multicentric infarction there often is little asymmetry. The patient can have a nonfocal examination. "Patient responds to pain poorly throughout," "moving none of the limbs spontaneously," or "patient is unresponsive to all stimuli," are descriptions written in the chart sometimes seen in patients with multifocal infarction.

Often on careful neurologic examination subtle asymmetries are found, and the patient will be imaged. The resulting image can show multiple infarctions in all the vascular territories of the brain in both the anterior and posterior circulation. If there are multiple infarcts in multiple distributions with or without hemorrhagic conversion this is unlikely to be related to carotid artery stenosis or an atretic vertebral artery on one side. Multiple emboli involving multiple arterial distributions found after surgery is from either cardiac source emboli or more likely from particulate embolic matter from the arch of the aorta released during cross clamping.11,14 Another cause of multiple lesions is either unilateral or bilateral hypoperfusion or watershed infarcts where the borderzone of the brain between the middle cerebral artery (MCA) and anterior cerebral artery (ACA) becomes infarcted secondary to decreased systemic blood pressure. In these patients shoulder and hip flexor weakness is often present with relative sparing of the distal musculature. This creates a man in barrel–type deficit, with weakness sparing the hands and feet and with involvement of the proximal upper and lower extremities.15

Posterior Circulation Strokes

Although it may not be intuitive as to why a posterior circulation stroke might cause slow awakening, it has a good neurologic explanation. About one quarter of cardioembolic strokes are to the posterior circulation. This is probably no different from postprocedural strokes compared with strokes related to atrial fibrillation or hypercoaguable states. Nonneurologists often think of posterior circulation strokes as resulting in the 4 Ds: dizziness, dysarthria, diplopia, and dysphagia.

The posterior circulation feeds the entire brainstem, cerebellum, bilateral thalamus, and much of the occipital lobes. Emboli entering one or both vertebral arteries can occlude the posterior inferior cerebellar artery and the medullary perforating arteries leading to medullary and cerebellar infarcts. Within the medulla there are sympathetic outflow fibers to the heart and vascular system that when damaged can result in dysautonomias. Next is the anterior inferior cerebellar arteries, which can result in pontine and cerebellar infarction. Pontine pathology can result in central neurogenic hypoventilation or apneustic breathing patterns. Superior cerebellar artery occlusions result in midbrain and cerebellar infarction. Within the midbrain is the reticular activating system that mediates arousal, and damage can result in stupor. Finally and most often, emboli reach the top of the basilar artery. At this location a single embolus may restrict blood flow to both posterior cerebral arteries feeding the bilateral occipital lobes, bilateral midbrain, and both thalami. Damage to the thalamus at the top of the reticular activating system can result in a coma. In all of these instances cerebellar stroke can lead to swelling or hemorrhage within the cerebellum.

Swelling of the cerebellum from a large stroke or hemorrhage can lead to downward herniation or occlusion of the aqueduct of Sylvius and the fourth ventricle, and acute hydrocephalus can present with coma before hydrocephalus occurs. Clues to a posterior circulation event are delayed awakening after surgery, abnormalities of cranial nerves, dysautonomias, visual dysfunction, change in respiratory pattern, and decerebrate posturing. Imaging, preferably MRI, of the patient with suspected posterior circulation events usually shows the location and extent of the infarction. In general, MRI is preferable because of better visualization of the posterior fossa and because it allows evaluation of the cerebral vascular system by MR angiography. Unless diffusion imaging is performed, both MRI and CT scans may show no pathology in the acute setting for 6 to 12 hours after the event.

Hypoxic Injury

Hypoxic brain injury is usually associated with very prolonged periods on cardiac-pulmonary bypass, cardiac arrests during or right after surgery, and prolonged or difficult ventilation of the patient. In these cases, as sedatives and narcotics
are weaned off, patients do not awaken and they may in fact continue to have no normal cranial nerve function. Although initially flaccid, they may later develop decorticate or decerebrate rigidity and posturing. Multifocal seizures can develop though there was no seizure disorder previously. Often patients develop multifocal myoclonus, which may also be mistaken for seizure activity. Myoclonus is a rapid twitching or jerking that occurs multifocally over the face and limbs and usually does not occur rhythmically. This should be separated from convulsive and nonconvulsive status epilepticus caused by acute anticonvulsant withdrawal, the use of proepileptogenic medications like aminophyline, electrolyte abnormalities, numerous beta-lactam antibiotics, or intoxication with lidocaine. Depending on the severity of the anoxic injury some patients recover during 24 to 48 hours. Outcomes from anoxic coma can be predicted by the length of time abnormal cranial nerve function, spontaneous motor function, and decorticate/decerbrate posturing persists. Those individuals who have absent pupillary responses, absent corneal reflexes, and absence of conjugate eye movements initially and at 24 hours have the worst prognosis.16

Electroencephalogram (EEG) evaluation and neuroimaging are the diagnostic tests of choice. In hypoxic injury, on EEG, a burst suppression pattern is an extremely poor prognostic indicator; in one series, 95% of patients with hypoxic coma and this pattern died.17,18 Alpha coma on EEG in which there is an alpha rhythm with no change in rhythm in response to eye opening or closing that persists for more than 24 hours from the onset of coma carries a poor prognosis.17,18 Persistent absence of detectable EEG activity in an individual with an anoxic injury who has not received any sedative hypnotic carries a poor prognosis. In one series only 2 of 187 patients with no detectable EEG activity after anoxic injury survived.18,19

Confusion

Patients who recover uneventfully from cardiac surgery but are confused can have multiple and frequently reversible causes of their problems. Confusion is very common in postoperative patients, and in one series 3.4% were confused after CABG.8 Certain focal brain lesions such as infarcts or hemorrhages of the nondominant frontal and temporal lobes, unilateral or bilateral occipital temporal strokes, and occasionally strokes in the unilateral or bilateral anterior cerebral artery distribution may cause confusion.20,21 Of primary importance is differentiating a metabolic-related confusional state from an aphasia, the latter being more suggestive of a new focal cerebral lesion.

Comorbidities and Iatrogenic Causes

Encephalopathy is more common in the elderly. They are more sensitive to the affects of sensory deprivation, sedative hypnotic drugs, electrolyte abnormalities, mild cerebral hypoxia, and the delayed effects of anesthesia. Older people with dementing disorders are more likely to respond poorly to bypass surgery and be hypersensitive to sedative/hypnotic and narcotic medications. Other risk factors for prolonged or severe encephalopathy include renal disease, liver disease, and a history of heavy alcohol consumption, which can lead to withdrawal states. In general, the alcohol or sedative hypnotic (barbiturates and benzodiazepines) withdrawal states are an agitated confusion with active hallucinations, hyperautonomic states (rapid heart rate, jumps in blood pressure, sweating, and tremor), and may lead to dangerous withdrawal symptoms like frank delirium tremens and sedative withdrawal seizures.

One medication in common use that the authors feel is a significant offender in causing encephalopathy is haloperidol. Haldol produces depressed alertness, increased tone and stiffness, and drowsiness. Haloperidol has been shown to stay in the body for an extended period of time and has been shown to retard recovery from cerebral insults in animals.9,22

Bypass-Related Causes of Encephalopathy

One of the published accounts that recognized that the neurobehavioral changes after heart surgery were not psychiatric in origin was made in a prospective series of patients undergoing open-heart surgery in 1965.23 Postsurgical encephalopathy was believed to be caused by embolization of particulate matter through the bypass pump, which allowed emboli to be introduced into the systemic arterial system. Advances such as using membrane instead of bubble oxygenators and in-line filtration were found to reduce the number of embolic particles that were greater than 25 µm
in diameter from reentering the systemic arterial system. In 1990, Moody et al. reported a necropsy study of 5 patients and 6 dogs that underwent cardiopulmonary bypass. In these necropsies, 10 of the 11 brains evaluated had 10-µm to 40-µm sausage-shaped small capillary and arteriolar dilations (SCADs). One of the patients was estimated to have 15.1 million SCADs. These were not found in patients or dogs that did not have cardiopulmonary bypass. Fifty percent of the dilatations were birefringent material suggestive of atheromatous material; the rest were believed to be caused by fatty or gaseous emboli.

A follow-up study was performed to assess the role of arterial line filters and the rates of postoperative neuropsychological abnormalities. Among 100 patients who presented for cardiopulmonary bypass, 50 had surgery with arterial filters, and 50 were performed without filters. Transcranial Doppler (TCD) was used to monitor the number of microemboli. Patients who had arterial filters had fewer microemboli seen on TCD during surgery. These patients had neuropsychological testing 8 days and 8 weeks after surgery. Neuropsychological deficits were more common in patients who did not have arterial filters and correlated with the number of microemboli seen on TCD. Thus, the number of microemboli correlated with the presence and severity of acute and long-term encephalopathy. Because the use of arterial filters (20 µm to 40 µm) is more common now, this is becoming less of an issue.

Weakness

When patients awaken postoperatively with weakness or numbness of a single limb or just the proximal arm and leg, they may not receive any evaluation as to the cause. More severe weakness with field cuts and aphasias usually are evaluated. Strokes and peripheral nerve lesions explain most single limb deficits found postoperatively.

Stroke

Stroke is a feared complication of cardiac surgery. Most focal neurologic deficits from cerebrovascular disease are noted after surgery. The risk of ischemic stroke during CABG is between 2% and 6%. In one study only 2% of patients had severe deficits. In evaluating a patient with weakness, a few basic observations should be made: Is there aphasia, altered cognitive function, or altered behavior with the weakness that was not previously present? Is there forced gaze deviation suggestive of left or right prefrontal damage? Are there new cranial nerve deficits such as new facial weakness or visual field loss, or new inability to swallow? Is there a pattern to the weakness, for instance, proximal weakness in the upper and lower extremities unilaterally or bilaterally? Is there distal versus proximal weakness in the arm or leg? Is there sensory loss and loss of coordination and ataxia? Are the reflexes symmetric? Are the plantar responses normal?

Cardioembolic strokes are more common during intracardiac surgery than in CABG alone (13% v 4.2%). In one study it was shown that in 2,264 patients, those who had intracardiac surgery with CABG had double the frequency of neurologic deficits as those who had CABG alone. This is possibly related to freeing of atheroembolic material from sclerotic valves and the secondary atrial dilatation and frequency of arrhythmias seen with these cardiac lesions. Strokes purely from a cardiac source can be the result of long-standing preoperative or intraoperative arrhythmias such as atrial fibrillation, known septal aneurysm, large areas of focal myocardial hypokinesia and/or dyskinesia, atrial or ventricular clot, decreased global left ventricular ejection fraction (LVEF), dilated left atrium, and discontinuation of coumadin for surgery. In clinical practice, cardiac source emboli from these cardiac sources tend to be solitary large emboli that produce a single large arterial occlusive event. This is why a transesophageal echocardiography (TEE) study is recommended before surgery, to identify possible cardiac anatomic lesions predisposing to clot formation. Postoperatively, neurologic events are most often explained by cardiac and aortic abnormalities. The authors recommend postoperative cardiac evaluation in episodes of cerebrovascular events during surgery to assess for the presence of cardiac thrombus. This is an important subtype of stroke because it is the only cause of stroke that the authors feel require heparinization during the postoperative period.

Postsurgical atrial fibrillation occurs in up to 32% of postoperative patients undergoing CABG and 64% of patients undergoing CABG and aortic valve replacement. It is usually self-limited and antiarrhythmic medication is often not insti-
tuted unless there is hemodynamic instability, and anticoagulation is not necessary if the atrial fibrillation lasts less than 72 hours. If there is a postoperative stroke believed to be secondary to atrial fibrillation, anticoagulation can be used and TEE is required to assess for clots within the atrial appendage.

Aortoembolic sources of emboli from ulcerative atherosclerotic lesions in the ascending aorta is one of the most important causes of stroke in patients undergoing CABG and cardiac surgery. During cardiac bypass the aorta is cross clamped to allow anastomosis of vein grafts. There is often an audible crunch heard as the aorta is cross clamped. TCD of the middle cerebral artery shows large numbers of emboli during the placement of the cardioplegia needle, aortic cannulation, and the start and termination of the cardiopulmonary bypass as well as cardiac ejection right after aortic cross clamp (Figs 1 and 2). Clamp- ing and unclamping of the aorta accounts for greater than 60% of the total emboli detected. Correlation between severity of atheromatous disease of the ascending aorta and the amount of atheroembolic disease was found when 46 of 123 (37%) patients with severe aortoatheromatous disease were found on necropsy to have atheroemboli in the brain whereas only 2 of 98 (2%) patients who did not have severe disease were found to have similar findings. In a retrospective study of 3,279 consecutive patients with CABG, severe atheromatous disease of the ascending aortic arch was found to be one of the clearest risk factors for postoperative neurologic deficits.

It is the authors' opinion that the incidence of stroke related to atheroembolic disease can be significantly reduced not by palpation of the cross-clamp site but by ultrasound of the chosen site, which has been shown to identify atheromatous material better than palpation alone. In another patient series with severe aortic atheromatous disease, Doppler probes were used to assess the ascending aorta. Modifications to the typical bypass grafting were used to avoid cross clamping of atheromatous material including alteration of the site of cannulation and cross clamping with some patients receiving hypothermic cardiac arrest to avoid cross clamping altogether. None of the 68 patients undergoing surgery in this manner had strokes. Aortic emboli are typically small and numerous and tend to flow to the anterior circulation—MCA branches and MCA/posterior cerebral artery (PCA) borderzones and the posterior circulation affecting the occipital lobe, cerebellum, and brainstem. Monitoring of the aorta by TEE during bypass also shows flurries of emboli in the aorta after unclamping (Fig 3). Figure 4 shows an ulcerated aorta in a patient who had multiple aortic-origin emboli detected during surgery and never awakened after surgery.

Embolization occurs mainly at the time of clamping release, and a TCD recorded white out made by a massive shower of emboli can be seen

---

Fig 1. TCD recording from MCA during steady-state cardiac bypass surgery at the time when the aorta was being manipulated. The white streaks represent microemboli.
A large percent of the emboli are gaseous particles but a significant percent are atheroembolic material in the 0.85-mm range. The flurry of small-diameter emboli often go to the border-zones of the MCA/PCA and MCA/ACA. This gives rise to strokes that resemble hypoperfusion strokes. The reduced mean arterial pressure in these patients inhibits normal washout of these microemboli.

Carotid embolization is a much-feared complication of CABG and has led to the practice of screening patients with carotid ultrasound and the use of staged or combined carotid endarterectomy in asymptomatic patients. There are a number of studies examining this problem and, unfortunately, the results are discordant. The incidence of hypoperfusion strokes related to carotid artery disease is probably lower than previously believed. The most important guide to risk is the presence of symptoms of brain ischemia (trans-ischemic attacks [TIAs] or strokes) during the previous 3 to 6 months. When patients are asymptomatic the risk of stroke after surgery because of carotid disease is relatively low. As
C. Miller Fisher noted, “If the patient can stand up, they can probably lie down,” without major risk. There is a very low rate of ipsilateral stroke in a pre-CABG patient with 50% to 75% asymptomatic carotid stenosis. One study reviewed the results of 144 asymptomatic patients with 155 arteries with greater than 50% obstruction; there was only 1.1% of ipsilateral strokes with 50% to 90% stenosis, 2% ipsilateral stroke with greater than 90% stenosis, and 2% with complete carotid occlusion.36 Patients with asymptomatic carotid stenosis with greater than 75% stenosis are probably at only slightly increased risk. Patients with bilateral carotid stenosis or occlusion are also probably at slightly increased risk. The carotid arteries have been given too much attention and the aorta and heart too little attention in the preoperative assessment. The data does support the use of prophylactic carotid endarterectomy in asymptomatic patients.11

There is, unfortunately, limited data on the correct way to manage patients who have asymptomatic carotid stenosis. There are 2 basic studies on the necessity for carotid endarterectomy (CEA) before CABG. One study showed that 9% (2 patients) with symptomatic or bilateral carotid disease greater than 70% stenosis had a surgical stroke.11,37 Another study showed that 3 of 10 patients with greater than 50% symptomatic carotid stenosis had an ipsilateral intraoperative stroke.11,30 The decision of whether to do staged or combination CEA and CABG or CABG alone in the end needs to be made on an individual basis weighing the risks and benefits and the overall health of the patient undergoing the procedure.

Hemorrhagic strokes in patients who have undergone cardiac surgery tend to be less common than ischemic strokes. Hemorrhage is much more common during repair of congenital heart disease and after cardiac transplantation than in CABG alone. This is particularly true of repair of transposition of the great vessels and ventricular septal defects.38-42 The cause of these hemorrhages is possibly because of reperfusion or relative hyperperfusion of a chronically underperfused brain. Intracerebral hemorrhage is believed to occur with cardiac transplant because the patient’s low baseline cardiac output is dramatically increased through surgery. The cause is probably similar to the cause of hemorrhage after CEA of highly stenotic carotid lesions.38,39,43,44

Peripheral Nerve Lesions

Lesions of the peripheral nervous system are another, typically more benign, problem causing limb weakness or numbness. In one large series of 421 patients, 63 new peripheral nerve lesions occurred among 55 patients, which represents 13% of patients.11,45 These include 23 brachial plexopathies, 13 saphenous neuropathies, 8 common peroneal palsies, 6 phrenic nerve palsies, 5 ulnar nerve palsies, 5 recurrent laryngeal palsies, 1 radial sensory neuropathy, 1 Horner’s syndrome, and 1 facial nerve palsy.45 The brachial plexus lesions typically involve the lower trunk C8-T1 region and can result in shoulder pain, numbness, hand weakness, dropped reflexes, and a difficult-to-treat pain syndrome. The rate of recovery depends on the cause of the lesion. The causes of the brachial plexopathies are thought to be related to ipsilateral jugular vein cannulation.
or a stretch injury when the chest wall is retracted or occasionally damage to the clavicle with retraction, which damages the brachial plexus as it travels underneath it. Harvesting of the internal mammary artery is believed to be a risk factor for brachial plexopathy because there is more chest retraction. Typically, stretch injuries to the brachial plexus are demyelinating lesions only and patients often recover. In general, a monoplegic arm with weakness, dropped reflexes, sensory loss, and pain in the shoulder and hand is more likely to be a brachial plexus lesion than a stroke or hemorrhage.

The saphenous nerve is primarily a sensory nerve. Lesions of the saphenous nerve present with medial calf numbness and pain from the base of the knee to the instep, typically from local trauma as the saphenous vein is harvested. The common peroneal nerve is a mixed motor and sensory nerve. It travels laterally around the fibular head and compression of this nerve leads to a foot drop weakness of the tibial anterior, extensor hallucis longus, and peroneus longus and brevis. It results in sensory loss across the lateral calf and the dorsum of the foot and is usually compressed secondary to poor positioning of the knee and leg. The phrenic nerve is a pure motor nerve that innervates the ipsilateral diaphragm and leads to inability of the hemidiaphragm to contract. Bilateral damage to the nerve is extremely rare and can result in exertional dyspnea, alveolar hypoventilation, and hypocapnea. The phrenic nerve runs through the mediastinum and can be damaged from stretch or secondary to hypothermia. The ulnar nerve is a mixed motor and sensory nerve that supplies sensation to the last 2 fingers on each hand and motor to the hand alone. There can be weakness of the interosseus muscles, deep flexors of the last 2 fingers, and thumb flexor. It is probably injured because of poor positioning of the elbow. The left recurrent laryngeal nerve loops beneath the aortic arch and then bends back upward to innervate and supply all the muscles of the larynx except the criocothyroid muscle. It presents with hoarseness and unilateral paralysis of the vocal cord.

**Postoperative Cognitive Dysfunction**

Cognitive and behavioral abnormalities are often recognized only when the patients return home or to their work place. The stereotypical phrase is “Grandpa hasn’t been the same since...” The impairment takes the form of loss of memory, attention, concentration, perseverance, and visual perception tasks. The incidence depends on when neuropsychological testing is performed and what tests are used but is quoted at between 33% to 83% of patients after CABG. Many patients improve over a period of a few months time, but up to 35% of patients with cognitive loss still have cognitive dysfunction 1 year postoperatively.

Advanced age and length of bypass are believed to be important risk factors for the development of cognitive dysfunction. The next most important factor is probably the rate and amount of microscopic embolization during clamping and unclamping of the aorta during bypass. Studies show a significant correlation between the use and presence of bypass and microembolization and the presence of postoperative cognitive deficits. Specifically, one study showed that 43% of patients with intraoperative embolic counts of more than 1,000 had cognitive abnormalities at 8 weeks whereas only 8% of patients with embolic counts of less than 200 had cognitive deficits. Another study showed that the average number of emboli at the time of release of aortic clamps was 116 in 6 patients with cognitive abnormalities compared with 73 emboli in 11 patients who showed no loss in cognitive function.

It is the authors’ opinion that the most important way to reduce cognitive abnormalities in patients who underwent CABG is the reduction in the number of emboli during surgery. This is best performed by careful and detailed assessment of cardiac lesions and aortic atherosclerotic lesion burden by TEE performed before CABG. Studies of the aorta help select patients in whom aortic clamping is especially risky and may select patients who should have surgery with TEE guidance of clamp site of the aorta, intraoperative use of filtration devices in the aorta at the time of bypass, and hypothermic fibrillary arrest.

**Basic Risk Assessment for Persons Undergoing CABG and Intraoperative Measures**

Preoperative patients who are undergoing elective CABG should have a detailed preoperative history...
and examination that should include a neurologic history and physical aimed at determining if there has been a stroke or transient ischemic attack in the past 3 months. Patients with a history of TIA in the past 3 months should have imaging of the brain if symptoms are referable to the anterior or posterior circulation and a carotid ultrasound or magnetic resonance angiography if referable to the anterior circulation. Assessment of the heart, its valves, and the ascending aorta is important to assess for moderate to severe atheromatous disease of the ascending aorta and for potential cardiac sources of emboli. Up to 80% of persons older than 75 years of age can have moderate to severe aortic atheromatous disease at necropsy.27,35 Those patients with high-grade symptomatic carotid stenosis should have elective CEA of the symptomatic carotid performed or combined CEA and CABG. For patients found to have high-grade (>75%) symptomatic carotid stenosis bilaterally, the recommendation is usually combined CABG and CEA on the most severely affected side.11

Intraoperatively, it is unclear if hypothermic or tepid bypass is better for the neurologic outcomes of patients. Care in placement of arms and legs during surgery and limitations on amount of chest wall stretching should be considered. More important is the reduction of the number of emboli that reach the brain intraoperatively with the use of arterial filtration as well as TEE to plan the site of cross clamping. In those cases in which TEE is unavailable a new potential technique is being used at some centers. Doppler ultrasound at the right supraclavicular fossa with a left supraclavicular probe can be used to image the proximal descending aorta.11,55 Other intraoperative techniques to use when focal atherosclerotic lesions of the aorta are noted are cannulation of the femoral artery instead of the aorta, graft replacement of the ascending aorta, hypnotic fibrillar arrest without aortic clamping, single aortic clamp technique, use of a cardioplegic needle, and consideration of non–heart-lung coronary artery bypass.11,55,56-58

Postoperatively the most important issues are hypotension and arrhythmias. Hypotension needs to be treated quickly and aggressively with an aortic balloon pump if required. Hyperpyrexa should be controlled as increases in core body temperature leads to increases in cell death in the central nervous system.59 Excessive sedation and the use of haloperidol should be avoided and imaging should be performed if patients require excessive time to awaken or if there are focal or global neurologic deficits. Prompt neurologic consultation can lead to the use of heparin and of thrombolysis of postoperative emboli from postoperative atrial fibrillation. Thrombolysis of clot in patients who are postoperative from CABG with the use of intra-arterial lytic agents is of unknown benefit but is promising. Intravenous thrombolysis is contraindicated for 14 days after surgery.

References


