CHAPTER 82  ■  VASCULAR SURGERY IN THE INTENSIVE CARE UNIT

ROBERT J. FEEZOR  •  TIMOTHY C. FLYNN

Patients requiring vascular intervention—whether open surgery or endovascular procedures—are elderly and have comorbidities that make their overall care complicated. To achieve a successful outcome, the perioperative care of the vascular surgery patient requires meticulous attention to detail and knowledge about the possible pitfalls these patients can encounter. Even the most seemingly innocuous clinical symptom must be thoroughly investigated and potentially treated in order to achieve acceptable perioperative outcomes. Despite meticulous attention to detail, vascular patients often fall victim to their comorbidities.

A key element in the care of the vascular patient is the recognition of vascular pathology as a systemic disease and not just a focal anatomic problem regardless of the procedure that brings the patient to the intensive care unit (ICU). The main exception to this is the young patient who sustains vascular injury. The nature of atherosclerosis is that it affects the blood vessels of all circulatory beds: cardiac, peripheral, and cerebral. Thus, patients who present with leg ischemia are at significantly higher risk than the general population for having both myocardial infarctions and aneurysms and patients should be monitored closely for such events. Lastly, cardiac dysrhythmias are often caused by ischemia, electrolyte disturbances, or fluid shifts in the postoperative period, and should all be treated with a statin regardless of cholesterol levels (8). Statins have numerous effects other than reduction of cholesterol including anti-inflammatory, immunomodulatory, and anticoagulant effects. Moreover, abrupt discontinuation may lead to a rebound effect and possibly increase cardiovascular complications (9). It is our practice to routinely start patients on a statin preoperatively and continue it throughout the postoperative period. The antiplatelet drug clopidogrel is frequently used in vascular patients. Preoperatively we review the indications for this drug, and if the indications are compelling, we may lead to a rebound effect and possibly increase cardiovascular complications (9). It is our practice to routinely start patients on a statin preoperatively and continue it throughout the postoperative period. The antiplatelet drug clopidogrel is frequently used in vascular patients. Preoperatively we review the indications for this drug, and if the indications are compelling, we will continue the drug through the perioperative period, recognizing that there may be a slightly increased incidence of wound complications. We do not hesitate to start the drug in patients who exhibit cardiac ischemia in the postoperative period.

The electrocardiogram (ECG) should be monitored continuously for any changes suggestive of ischemia. For the diabetic population, angina may present as nausea and must be interpreted as signs of myocardial ischemia until proven otherwise. Lastly, cardiac dysrhythmias are often caused by ischemia, electrolyte disturbances, or fluid shifts in the postoperative period, and patients should be monitored closely for such events.

In order to decrease the risk of perioperative cardiac events in the vascular surgery patient population, much attention has been given to preoperative risk stratification. In the surgical and anesthesia literature, most vascular surgery procedures for occlusive or aneurysmal disease are placed in the “high-risk” category. The question is how to minimize the risk of
perioperative cardiac complications. Data from several randomized, multicenter trials have shown that coronary revascularization (percutaneous or open) before elective major vascular surgery does not decrease the overall mortality (10). Nevertheless, many clinicians request preoperative cardiology consultation to help determine existing cardiac function, usually with an ECG, echocardiogram, or a chemical cardiac stress test.

Even without prior known elevated serum creatinine, many vascular patients have renal insufficiency as determined by creatinine clearance. Nephrotoxic effects of the IV contrast commonly used in revascularization procedures make postoperative renal dysfunction a constant threat. Moreover, perioperative mortality after most vascular procedures is significantly increased in patients with renal failure (11). Strict monitoring of fluid balance, maintenance of serum electrolytes, appropriate dosing of nephrototoxic medications, adequate hydration, and resumption of chronic diuretics will all help to minimize the chance of postoperative renal dysfunction.

A majority of vascular patients have diabetes mellitus and this group is at higher risk for postoperative complications, both vascular and nonvascular. From a vascular perspective, patients with diabetes have a higher rate of postoperative amputations after peripheral bypass surgery for tissue loss (11). Diabetics are also at risk for other postoperative morbidities including postoperative wound infections. They should be maintained euglycemic, even if that requires a constant intravenous infusion of insulin, with a target blood glucose of 80 to 110 mg/dL.

There is a subset of patients with vascular disease who under hypercoagulable state should be raised with patients with seemingly advanced atherosclerotic disease at a younger age. A careful history can assist with determining these patients, but when identified, they should be started on appropriate anticoagulation. Hematology consultation should be obtained, but may be of somewhat limited value in the setting of the acute thrombotic event.

**VASCULAR CARE IN THE INTENSIVE CARE UNIT**

All patients in an ICU have a propensity for developing venous thromboembolic events. Virchow’s triad dictates that patients at risk include those with trauma, endocardial injury, and a hypercoagulable state. In the postsurgical population, venous stasis is inevitable due to the patients’ relative immobility. Endothelial injury occurs during the course of the surgical procedure. It is our practice that all patients receive chemical and/or mechanical prophylaxis; we routinely use low-molecular-weight or unfractionated heparin and/or sequential compression devices when there is no existing contraindication. We avoid lower extremity sequential compression devices in patients with severe peripheral arterial occlusive disease, although the data for this practice are anecdotal. The incidence of heparin-induced thrombocytopenia is relatively rare, and when suggested by a decline in platelet count, we promptly cease all systemic or local heparin and transition to purely mechanical prophylaxis.

Stress gastritis is a constant threat in the vascular ICU patient. Patients are routinely placed on either histamine-receptor blockers or proton pump inhibitors, irrespective of any clinically detected gastrointestinal hemorrhage. Opponents of this practice suggest that in doing so, one of the body’s natural defense mechanisms (gastric acidity) is altered, but we find that the risk of stress gastritis exceeds the diminution in host defenses.

Ventilator-associated pneumonia has been well documented to increase in-hospital mortality, length of stay, and overall cost of hospitalization. We employ routine suctioning, aggressive bronchoscopy to control secretions, and head elevation for all our intubated patients. Once extubated, activity is encouraged and adequate pain control is important for patients with an abdominal or a thoracic incision.

The routine assessment of the vascular ICU patient includes not only all the usual cardiovascular, pulmonary, and metabolic parameters, but also frequent and detailed physical exams. All incisions should be inspected for signs of early wound complications such as infection, separation, or hematoma. Objective assessment of distal perfusion should be performed regularly, even hourly, in the immediate postoperative period. This assessment includes looking at the extremity for cutaneous signs of malperfusion, assessing motor function, and palpating the major muscle groups for tenuity, which may signify compartment syndrome in patients who are too sedated to relate the classic “pain with passive motion.” The best exam, in our opinion, is to elevate the lower extremity by placing the hand behind the Achilles tendon and palpating the anterior calf compartment with the posterior leg off the bed. A patient should be alert enough to follow commands of simple dorsiflexion, again ensuring that the posterior knee is off the bed. (Dorsiflexing the foot with the heel resting on the bed can be achieved with flexion of the quadriceps muscle concern for a hypercoagulable state should be raised with patients with seemingly advanced atherosclerotic disease at a younger age. A careful history can assist with determining these patients, but when identified, they should be started on appropriate anticoagulation. Hematology consultation should be obtained, but may be of somewhat limited value in the setting of the acute thrombotic event.

Ventilator-associated pneumonia has been well documented to increase in-hospital mortality, length of stay, and overall cost of hospitalization. We employ routine suctioning, aggressive bronchoscopy to control secretions, and head elevation for all our intubated patients. Once extubated, activity is encouraged and adequate pain control is important for patients with an abdominal or a thoracic incision.

The routine assessment of the vascular ICU patient includes not only all the usual cardiovascular, pulmonary, and metabolic parameters, but also frequent and detailed physical exams. All incisions should be inspected for signs of early wound complications such as infection, separation, or hematoma. Objective assessment of distal perfusion should be performed regularly, even hourly, in the immediate postoperative period. This assessment includes looking at the extremity for cutaneous signs of malperfusion, assessing motor function, and palpating the major muscle groups for tenuity, which may signify compartment syndrome in patients who are too sedated to relate the classic “pain with passive motion.” The best exam, in our opinion, is to elevate the lower extremity by placing the hand behind the Achilles tendon and palpating the anterior calf compartment with the posterior leg off the bed. A patient should be alert enough to follow commands of simple dorsiflexion, again ensuring that the posterior knee is off the bed. (Dorsiflexing the foot with the heel resting on the bed can be achieved with flexion of the quadriceps muscle concern for a hypercoagulable state should be raised with patients with seemingly advanced atherosclerotic disease at a younger age. A careful history can assist with determining these patients, but when identified, they should be started on appropriate anticoagulation. Hematology consultation should be obtained, but may be of somewhat limited value in the setting of the acute thrombotic event.

Ventilator-associated pneumonia has been well documented to increase in-hospital mortality, length of stay, and overall cost of hospitalization. We employ routine suctioning, aggressive bronchoscopy to control secretions, and head elevation for all our intubated patients. Once extubated, activity is encouraged and adequate pain control is important for patients with an abdominal or a thoracic incision.
of the aorta. Doppler signals distal to an obstruction may be characterized as biphasic or monophasic signals with the latter signifying significantly diminished blood flow. Sometimes it is difficult to tell if the sound is venous or arterial. If the sound disappears with gentle pressure on the Doppler probe, it is likely a venous sound. Also, if the sound in one of the pedal pulses disappears with gentle compression around the forefoot, it may be a venous and not arterial sound. At the conclusion of any vascular procedure, extremity perfusion is assessed prior to leaving the operating room. The operating surgeon should relay to the ICU team of physicians and nurses the quality and location of each Doppler signal or palpable pulse, as well as the frequency that he or she wants the perfusion assessed. Any change in the exam or inability of the examiner to detect the signal may potentially constitute an emergent trip back to the operating room to restore perfusion. Loss of a palpable pulse even if the pulse remains by Doppler should always be cause for alarm and the operating team should be alerted.

As an objective marker of extremity perfusion, we advocate bedside ankle-brachial index (ABI) measurements. This is done by inflating blood pressure cuffs on each arm and listening to the Doppler signal of the brachial arteries and comparing the values to the Doppler signals auscultated at the dorsalis pedis (DP) and posterior tibial (PT) arteries after inflating the cuff on the calves. The pressure at which arterial perfusion is restored as the cuff deflates is noted in each location. The ABI is the quotient of the pressure in the higher of the DP or PT pressures and the higher of the arm pressures. Each leg has a single ABI. Any change of greater than 0.15 is significant and should be reported, independent of any other clinical event.

All vascular surgery wounds should be examined daily for signs of infection. Of particular difficulty are the incisions made in the groins. The incidence of groin wound complications in the vascular surgery patient has been estimated to be up to 44% in some series (12,13). Although most surgeons try to close the vascular graft or anastomosis with the devastating potential for anastomotic disruption. When the bypass graft is noted to be exposed, patients should be scheduled for the operating room for exploration and attempted reclosure of the wound, preferably with autogenous tissue such as a sartorius or rectus flap. Until the patient can go back to the operating room (OK), it is imperative that all health care personnel treating the patient be aware of exposed vasculature. We have instituted a “blowout precaution” protocol wherein patients are kept at bedrest and blood typed and crossed. Any bleeding from the wound is a potential emergency. Immediate pressure should be held on the wound, the patient intubated and anesthetized, the surgeon scrubbed in, and the operative field prepped (even if this includes the team member’s gloved hand being prepped into the field).

For the vascular patient, meticulous care of the skin is mandatory, and even modest duration of pressure on the heel by the bed mattress can lead to skin breakdown and turn a successful revascularization into an amputation. Since the vast majority of vascular patients have compromised distal perfusion, we try to keep the heels off of the bed by placing the extremity on pillows, which allows the weight of the leg to be borne over a larger surface area. There is no substitute for frequent inspection of all pressure-sensitive areas and this should be part of the physician’s and nurse’s practice.

Pharmacologic prophylaxis against thromboembolic events is the routine. However, many patients require systemic anticoagulation after vascular surgical procedures (14) such as with distal bypasses when there is compromised outflow or less than ideal conduit. The need for systemic anticoagulation must be balanced with the risk of bleeding complications, and usually we hold off full anticoagulation until postoperative day 2 or 3. In most patients we will give subtherapeutic heparin (400–500 U/hour) in the early postoperative period. Patients are monitored for any decline in platelet counts, and if seen, a heparin antibody panel is sent. If the clinical suspicion is high, we stop all heparin and switch to anticoagulation with other agents. Regardless of the agent chosen, it is imperative that the anticoagulation be monitored closely and is best accomplished with protocol-driven therapy.

Acute limb ischemia in the ICU setting can have disastrous consequences. The pathologic differential includes embolic events (usually from cardiac or aortic sources) or as in situ thrombosis of pre-existing atherosclerotic lesions that likely is a consequence of plaque instability and the aggregation of platelets, which then occludes the vessel. If identified acutely, there may be a role for intra-arterial thrombolysis, although in the setting of the postoperative patient, this role is limited due to excessive bleeding risk. When an ischemic extremity is identified, either catheter-based therapy or open surgical thrombectomy, should be entertained. In general, if the acute arterial occlusion is associated with motor or sensory deficits, then an emergent exploration is indicated. More aggressive intervention, either catheter-based therapy or open surgical thrombectomy, should be entertained. In general, if the acute arterial occlusion is associated with motor or sensory deficits, then an emergent exploration is indicated. On rare occasions, patients present with acute lower extremity paralysis secondary to acute infrarenal aortic occlusion. There is often a delay in diagnosis owing to an investigation of neurologic causes of the paraplegia. Absence of femoral pulses is a clue to the vascular nature of the paralysis. These patients typically require emergent procedures, and despite operative success, the perioperative mortality rate exceeds 50% (16).

Common ICU causes of arterial occlusion include sequelae of invasive monitoring, usually intra-arterial lines. In a recent review of brachial artery cannulations for cardiac catheterizations, the overall complication rate was an astonishing 36% (17). Not infrequently we are called to assess lack of distal perfusion in an extremity with an indwelling arterial line. The first step is to remove the catheter and to observe for restoration of perfusion. The collateral blood supply should also be assessed (usually the ulnar pulse in the event of radial artery occlusion).
as well as the distal perfusion, including motor and sensory assessment. Choices of therapy include observation, systemic anticoagulation, local thrombolysis, and operative thrombectomy with the potential for bypass.

The choice of invasive arterial and venous monitoring can represent a continuous challenge in any ICU patient, but in particular the vascular ICU patient. Lower extremity intravenous and arterial lines are contraindicated in patients with peripheral arterial occlusive disease. Furthermore, patients with dialysis access fistulae should have that extremity kept free of IVs, central venous catheters, invasive arterial lines, and noninvasive blood pressure cuffs. If a patient is identified as likely to require permanent vascular access in the future, duplex ultrasonography should be used to identify a potential arm for future access, and the identified extremity should be preserved.

Various bleeding complications can occur in the postoperative vascular wound. These can range from simple “skin edge” bleeding to frank exsanguination. Skin edge bleeding may be a nuisance, and may be treated with manual compression, application of silver nitrate, or a simple suture. Hematomas are monitored closely. Recurrent blood transfusion requirements, overlying skin or wound compromise, delusitious mass effects, and hemodynamic instability are all indications for operative evacuation of the hematoma. Patients who have had percutaneous interventions (usually through the groin at the common femoral artery) should also be monitored for hematomas, and in these instances, simple manual compression may be adequate. Attempted femoral artery punctures that are aimed more cephalad may in fact be external iliac artery punctures. Compression for hemostasis may be ineffective due to the retroperitoneal location of the arteriotomy. A progressive hematoma in such a location more often requires surgical repair (open or endovascular).

Specific Conditions

Aneurysmal Disease

Infrarenal Abdominal Aortic Aneurysm

Approximately 90% of the extracranial aneurysms found in the human body involve the infrarenal aorta. The natural history of aneurysms of the aorta is to expand and rupture. The tension felt by the thinning aortic wall can be estimated by the Law of Laplace, which describes the relationship between aortic diameter and wall tension. The results of randomized trials and observational studies have led vascular surgeons to recommend operative repair when the diameter of the aorta reaches 5.5 cm in asymptomatic patients (18), but the numeric value varies, especially with female patients. Most aneurysms are asymptomatic and are discovered during radiographic workup of other problems. Patients who have symptomatic aneurysms generally complain of back or abdominal pain. These symptoms should be interpreted as a sign of impending rupture necessitating urgent repair. We no longer place pulmonary artery catheters routinely, but all patients have arterial lines and Foley catheters and most have central lines. All patients get a single dose of preoperative antibiotics, which are not continued postoperatively.

Endovascular Repair. Depending on patient anatomy and institutional expertise, abdominal aortic aneurysm repair can be performed either via an open or endovascular approach. The endovascular approach holds great appeal in terms of reduced physiologic insult to the patient. Typically, both common femoral arteries are accessed either percutaneously or via an open groin exposure, and the device is placed from within the arterial lumen using fluoroscopic guidance. The weakened arterial wall is bolstered from within with stents made of a malleable metal alloy and a woven fabric. These patients rarely require admission to the ICU but are monitored for hematomas and lower extremity pulses. The devices used to deploy endovascular stents can be as large as 26 French, and these are introduced through femoral or external iliac arteries. There is a possibility of local arterial damage or dislodging of plaque that may embolize distally.

Open Repair. Open aneurysms, on the other hand, require surgical ICU monitoring postoperatively. The overall perioperative mortality is approximately 5% (19). Because a prosthetic graft has been sewn to the abdominal aorta, the main concern is bleeding. Furthermore, because the blood supply to the lower extremities is occluded intraoperatively during the aortic repair, it is vital to objectively assess and document lower extremity perfusion. Lower extremity ischemic events after open abdominal aortic aneurysm repairs occur in 2% to 5% of patients (20). Any inability to detect a Doppler signal or palpate a pulse when there previously was one is a potential surgical emergency.

A major complication of open abdominal aortic aneurysm surgery is gastrointestinal problems. A large retrospective study estimated the incidence of postoperative prolonged ileus to be 11.1% and nonspecific diarrhea to be 7.1% (20). All patients will have a brief period of postoperative ileus that may be shortened by use of a retroperitoneal approach to aneurysmorrhaphy (21). However, the dreaded complication is abdominal aortic aneurysm with an estimated prevalence of 0.6% (20). Most instances present as bloody stools 3 to 5 days postoperatively but may occur as early as the first 24 hours after surgery and are cause for considerable concern. Warning signs include fever, abdominal pain, thrombocytopenia, unexplained leukocytosis, or lactic acidosis. Any suspicion of colonic ischemia should prompt endoscopic evaluation, with the obvious caveat that endoscopy will only view the mucosal changes, and cannot evaluate for transmural ischemia. However, in the appropriate clinical setting, mucosal ischemia may justify operative exploration with possibly colon resection and end colostomy. These patients require invasive ivaseive ICU monitoring, as they often progress to multisystem organ failure as a result of their colonic ischemia. Routine broad-spectrum antibiotics to include Gram-negative and anaerobic coverage are used.

Other potential gastrointestinal complications known to occur include cholecystitis and pancreatitis. The latter is probably related to direct surgical trauma during aortic exposure and is usually self-limited. Cholecystitis may be ischemia related or may be a variant of acalculous cholecystitis seen in ICU patients. Treatment options range from percutaneous cholecystotomy to surgical cholecystectomy. Much like the problem of colon ischemia in the setting of an aortic graft, an infected gallbladder should not be overlooked or minimized.

The incidence of postoperative renal dysfunction can be as high as 5.4% after open infrarenal aortic surgery, but dialysis requirement is much less at 0.6% (20). Renal dysfunction is significantly lower in patients who have undergone infrarenal...
Vascular Surgery in the Intensive Care Unit

Aortic cross-clamp, thereby avoiding the obligate renal ischemia-reperfusion. The exact etiology of the renal dysfunction after infrarenal clamping is largely speculative, but may involve migration of atheroemboli leading to acute tubular necrosis. In the early postoperative period, oliguria is most frequently due to intravascular depletion and not intrinsically renal dysfunction. However, patients with baseline renal insufficiency, those more than 2 days postoperative, or those who do not respond appropriately to intravenous fluid challenges should be investigated for acute tubular necrosis or other intrinsic (nonprerenal) cause of oliguria.

In the absence of other causes (e.g., colon ischemia), patients may experience postoperative thrombocytopenia. Although an inciting event or agent is not always identifiable, there are several likely etiologies. Before occluding the aorta in the operating room, all patients are systemically heparinized, and although our practice is to reverse the anticoagulant effects of heparin toward the end of the case, the drug’s side effects may persist. Unless there is evidence of ongoing bleeding, mild thrombocytopenia is usually well tolerated.

The cohort of patients who get abdominal aneurysms may have coronary artery disease and are at risk for postoperative myocardial infarctions, dysrhythmias, and episodes of congestive heart failure. Johnston reported an incidence of myocardial infarctions (5.2%), heart failure (8.9%), and dysrhythmia requiring treatment (10.5%). The overall incidence of any perioperative cardiac event was 15.1% (20). Unless contraindicated, patients undergoing open aneurysm repair should be on a medical regimen consisting of a β-blocker with a target heart rate of 70 to 75, a statin (independent of serum cholesterol levels), and some form of antiplatelet therapy, usually aspirin.

Ruptured Aortic Aneurysms

A meta-analysis found the operative mortality rate of ruptured abdominal aortic aneurysm (AAA) to be 48%, with a small decline in mortality for each decade from the 1930s to the 1990s (22) (much higher than an elective AAA repair of ~5% mortality). With ruptured AAA, there are impressive fluid shifts that transpire during such an emergent operation, independent of overt blood loss. These fluid shifts, associated with the hypotension and the physiologic strain of an emergent procedure, contribute to a tenuous postoperative course. The incidence of colonic ischemia is significantly higher after ruptured aneurysm repair compared to elective open aneurysmorrhaphy, and some authors recommend empiric and routine endoscopic evaluation of the colonic mucosa.

Juxtarenal or Suprarenal Aortic Aneurysms

Most aortic aneurysms are infrarenal, meaning that the proximal extent of the dilated segment of aorta is caudal to the lowest renal artery. Therefore, operative repair usually can be performed with infrarenal aortic occlusion in the operating room. If the aneurysm extends to the level of the renal arteries, or involves the para-vascular aorta, the repair becomes technically more challenging. The postoperative complications escalate dramatically due to renal and possibly mesenteric ischemia-reperfusion. Depending on the length of intraoperative ischemia, there is a resultant release of pro- and anti-inflammatory cytokines that drives a systemic inflammatory reaction resulting in multisystem organ failure (23). There is considerable third spacing of fluid in the first 24 hours as edema collects in the interstitial spaces. Attempts to improve mortality and morbidity by a hybrid approach involving multiple visceral bypasses and endovascular repair of the aneurysm have met with mixed results (24). If the thoracic cavity is violated as a part of the aneurysm repair, the patient will have an even greater risk of pulmonary complications. Routinely a chest tube is placed intraoperatively to drain any pleural fluid that may accumulate. Adequate pain control is key in these patients.

Infected Aortic Graft

One of the more dreaded complications of aortic surgery is infection of the prosthesis. This rarely happens in the early postoperative period, and the majority occurs months to years later with unexplained fevers and a computed tomography (CT) scan that shows fluid around an aortic graft. Other patients present with gastrointestinal bleeding (a manifestation of an aortoenteric fistula) or a draining sinus in the groin. These are serious surgical problems, and patients should be treated aggressively. Broad-spectrum antibiotics (although the causative organism is usually Staphylococcus), IV rehydration, and close hemodynamic monitoring should be undertaken. Patients should be medically optimized and prepared for a staged procedure. The initial step is usually an extra-anatomic bypass in the form of an axillofemoral bypass, with subsequent laparotomy and excision of the aortic graft. In patients who are good operative candidates, a single-stage aortic replacement using autogenous tissue (syndactylized bilateral femoral veins) of cadaveric vessels can be entertained. These patients are routinely sent to the ICU since some may become floridly septic after manipulation of the infected retroperitoneum.

Despite the misnomer of a “dissecting aneurysm,” aneurysms do not dissect. Rather, dissections may become aneurysmal. Dissections start as an intimal flap and blood escapes the true lumen and channels down the aorta, shearing apart the layers of the wall. Dissections that involve the ascending aorta (Stanford type A) are cardiac surgical emergencies for fear of retrograde dissection, causing coronary malperfusion or cardiac tamponade. Aortic dissections that do not involve the ascending aorta (Stanford type B) are usually treated medically with aggressive blood pressure control. The four indications for operative intervention are branch vessel malperfusion (usually celiac, superior mesenteric, renal, or iliac), inability to control hypertension, persistent pain related to the dissection, or aneurysmal degeneration. Dissections can be repaired via open techniques or endovascularly. The postoperative implications and precautions are the same as with any thoracic aortic intervention, with the additional caveat that blood pressure control is paramount.

Arterial Occlusive Disease

Many patients experience narrowing or occlusion of their aortoiliac arterial tree. This can be detected by the absence of a palpable femoral pulse and symptoms of lower extremity cardiovascular compromise. Claudication, tissue loss, or ischemic rest pain. The specific diagnosis and management of these problems are beyond the scope of this chapter. The most durable surgical solution for aortic occlusive disease is an aortobifemoral (ABF) bypass. This is accomplished using a celiotomy incision as well as two groin incisions. The prosthetic graft (usually Dacron) is sewn to the aorta just below the renal
arteries with similar complications as with open aneurysmorthoraphy (i.e., bleeding, postoperative ileus, colonic ischemia, renal insufficiency, lower extremity ischemia, cholecystitis, pancreatitis). The limbs of the bifurcated graft are then tunneled beneath the ureters and sewn into the femoral bifurcation, usually hooded onto the profunda femoris. The groin incisions, similar to those used for infrainguinal bypasses, should be monitored for wound breakdown, infection, and drainage. Peripheral pulses are regularly monitored and any deviation from the immediate postoperative result is a potential emergency as it may represent a graft thrombosis. Another complication is distal embolization with ischemia of the toes (trash foot). Management is expectant and most often this resolves with minimal or no permanent tissue loss.

As endovascular technology evolves, many iliac lesions are treated with angioplasty and possible stent placement. Although better tolerated by patients, the stents may not be as durable as the surgical bypass procedures. The wound is much smaller in stent placement (puncture sites) compared to the larger abdominal and groin incisions seen in ABE. A small subset of patients, namely patients under the age of 55, are believed to have better long-term vascular durability for infrarenal aortic reconstruction with autogenous tissue rather than Dacron (25). Femoral veins can be harvested and syndactylized to be used as aortic replacement. This requires more extensive operations with longer OR times and larger leg incisions, which can be a cause of significant morbidity.

As with any surgical procedure, redo aortic surgery is fraught with intraoperative and postoperative complications. Patients generally require longer recovery periods. If the decision is made to proceed with a redo procedure, the field (abdomen and retroperitoneum), extra-anatomatic bypasses may be performed, usually axillofemoral. These procedures are considered to be less invasive but also still require the same vascular monitoring as any other bypass procedures. Although abdominal complications are not seen, patients still require groin and auxiliary incisions and there is significant subcutaneous tunneling for the graft placement. With rehabilitation, trapzede devices are contraindicated to avoid undue stress on a fresh arterial auxiliary anastomosis.

**Infrainguinal Bypasses**

Infrainguinal bypasses are commonly performed to alleviate symptoms of vascular compromise. The principles for vascular surgery are simple: the patient must have adequate inflow (from the femoral artery), adequate outflow (of the popliteal, tributary, peroneal, or pedal arteries), and conduit ("pipe" to perform the bypass). The incisions that are made are significant, and may not only be located on the extremity being reperfused, but also may be on either leg or arm as a site of vein harvest. We are particularly aggressive about harvesting autogenous tissue for vein conduit as the patency of infrainguinal bypass grafts using autogenous tissue, especially the greater saphenous vein, is clearly superior to that using prosthetic tissue (e.g., polytetrafluoroethylene [PTFE]) (26). The main sources of morbidity from these procedures are arterial occlusion (which can be detected with routine close pulse/Doppler monitoring), bleeding, and wound complications. The mortality from peripheral bypasses is estimated to be between 2% and 8%, and the cause of mortality is primarily cardiac, so aggressive cardiac medical management, judicious use of antithrombotic therapy, and careful fluid status monitoring are essential (27,28).

Any revascularization procedure is associated with a reperfusion syndrome that is usually mild and well tolerated. However, the reperfused extremity should be monitored for compartment syndrome and acted upon early. Details about the technique of detecting compartment syndrome and fasciotomies have been described above. Electrolytes and cardiac rhythm should be monitored, and the urine assessed for myoglobinuria, even if that means a simple visual inspection of the urine color.

A major complication of any revascularization procedure is graft thrombosis with the highest risk in the immediate postoperative period most likely due to platelet aggregation on a surgically damaged endothelium. Patients with "high-risk" grafts (i.e., multiple segments of vein sewn together as a conduit, small distal target arteries, or poor-quality arteries) are routinely systemically anticoagulated postoperatively with heparin (14). At our institution, there has been a slight increase in the incidence of postoperative wound hematomas, but the fraction that need operative evacuation is small. In addition to full anticoagulation, patients with endovascular stents are routinely placed on clopidogrel to decrease the incidence of in-stent restenosis. All patients should be on aspirin unless otherwise contraindicated.

**Carotid Endarterectomy**

Carotid endarterectomy has been shown to decrease the chance of a future cerebrovascular accident in certain patients with carotid stenosis. The procedure involves a neck incision along the anterior border of the sternocleidomastoid muscle, and occlusion of the carotid artery to attain vascular control. Once occluded, the operating surgeon then may place a plastic shunt to resuscitate same surgical and distal perfusion while the endarterectomy is being performed. Although there are many intraoperative variables in technique (mode of anesthesia, whether or not to shunt, and type of shunt), the key outcome variable is perioperative stroke related to disruption of cerebral blood flow or embolic event from clamping an atherosclerotic vessel. Aspirin should be continued in the recovery phase, but full anticoagulation is seldom indicated unless carotid occlusion has occurred. Neurologic deficit may manifest itself upon awakening or occur in the early postoperative period. Any change in neurologic function that occurs after awakening with a normal neurologic examination should be reported to the operating team. Opinions vary whether to investigate with imaging or return directly to the operating room depending on whether the deficit is transient or seems to be dense and progressive.

Due to the baroreceptors in the carotid bulb, patients often experience large fluctuation in blood pressures, which should be targeted to the "normal range" of 120 to 140 mm Hg. Additionally, any wound hematoma, because the neck is a relative closed space, can cause carotid compression and result in bradycardia or potentially airway compression. The operating team should be alerted if hematoma is suspected. Because the field of dissection is intimately associated with the cranial nerves, a detailed head and neck exam is mandatory at regular intervals. Particular attention should be paid to assessing the function of the marginal mandibular nerve and the hypoglossal nerve. Headache and even seizure activity may be a manifestation of cerebral reperfusion syndrome. This is rarely seen in the immediate postoperative period, but may cause readmission for blood pressure control in the weeks after carotid endarterectomy. These patients should also have a
CT scan because of the incidence of intracranial bleeding that accompanies these symptoms.

Mesenteric Revascularization

Mesenteric ischemia, whether acute or chronic, can have lethal consequences. The restoration of intestinal perfusion sets in motion a cascade of inflammatory cytokines that frequently progresses to the systemic inflammatory response syndrome, multisystem organ failure, and even death. After restoration of blood flow, patients typically have a period of hemodynamic stability for 24 to 48 hours after the procedure, but then progress to retaining more fluid and show signs of systemic inflammation. Subtle early changes such as a diminution in platelet count should elicit concern. To date, despite numerous anticytokine therapies, the treatment of the systemic inflammatory response syndrome is largely supportive (29). As this response is not uniform, efforts to predict which patient will progress to clinical deterioration have been unsuccessful. As with any other revascularization bypass, the patency of the mesenteric graft should be assessed objectively. Duplex ultrasound is noninvasive and is highly sensitive. Despite all the usual supportive measures, the average postoperative length of stay is over 3 weeks (30).

VASCULAR TRAUMA

The care of the trauma patient with vascular injuries shares many of the same principles as care of other vascular surgery patients with the exception that this cohort frequently, but not always, lacks the systemic comorbidities of the typical atherosclerotic vascular patient. Most extremity vascular injuries are associated with orthopedic fractures and dislocations; some injuries are nearly synonymous with vascular injuries, such as a posterior knee dislocation and popliteal artery injury. In the secondary survey as part of the Advanced Trauma Life Support (ATLS) evaluation of the trauma patient, extremity pulses should be assessed and clearly documented. For any patient recovering from an orthopedic procedure, the same attention to distal perfusion is merited. Any change in pulse exam or hard sign of vascular injury mandates radiographic evaluation, usually with an arteriogram, although a CT angiogram is sufficient.

Although most surgeons no longer explore extremities when a penetrating injury is in proximity to a vessel, penetrating trauma associated with hard signs of vascular injury (decreased distal perfusion, active arterial hemorrhage, or a rapidly expanding hematoma) should be evaluated immediately after lifesaving measures are undertaken. Often, the area of interest is operatively explored and the vessel visually inspected. If injured, it is either repaired or blood is rerouted around the “blunt field” (e.g., an external iliac artery injury in a contaminated field may be repaired with vessel ligation and a femoral-femoral bypass to perfuse the ipsilateral leg). Venous injuries are ligated unless easily repaired. Revascularization of an extremity that has too extensive musculoskeletal damage to be salvageable can be treated with simple ligation and amputation. Lastly, trauma to an artery and adjacent vein can result in a traumatic arteriovenous fistula. This can occur even months after the inciting trauma, and unexplained extremity swelling, distal ischemic symptoms, heart failure, or an audible bruit over an extremity should alert the clinician to the presence of a fistula.

The perceived incidence of aortic trauma is increasing, possibly due to the increased use of CT scans in trauma management. In the abdomen, any central periaortic hematoma should be operatively evaluated. With the resolution of the current scanners we are seeing a number of initial injuries and short segments of dissection in the infrarenal aorta and iliac vessels that previously were not detected. In the absence of hemodynamic compromise most of these can be observed. Thoracic aortic injuries are increasingly treated with endovascular devices. Initial care is directed toward treating the urgent life-threatening injuries and controlling blood pressure with β-blockers. Open surgical repair is still a viable option, though in most series the morbidity is clearly greater.

Penetrating neck trauma can involve the carotid artery, which can be exposed readily in certain locations (zone 2) or require more extensive operations to expose adequately (zones 3 and 1). Our current management of neck trauma in the stable patient with a zone 2 injury is to cover the wound in the trauma bay, perform the global assessment of the patient including abdominal sonography and intravenous resuscitation, and then take the patient to the OR for exploration. Only then is the injury exposed since unroofing a clot and losing hemostasis is best done with good exposure in the OR. For more proximal or distal injuries, angiography (standard contrast angiography or CT angiography) plays a vital role in both diagnosing and planning either open or endovascular treatment.

Blunt trauma to the head can result in injury to the carotid or vertebral arteries. Because these injuries are relatively rare (<1% of blunt trauma patients), controversy remains about the best way to diagnose and treat these patients. The Eastern Association for the Surgery of Trauma (EAST) has recently published practice management guidelines on the management of vascular injury (31). They recommended screening, preferably with angiography, for blunt trauma patients who present with or develop an unexplained neurologic deficit or who have cervical spine fractures, LeFort II or III fractures, petrous bone fracture, or fracture through the foramen transversum, and for those with a Glasgow coma scale score <8 or diffuse axonal injury. While CT angiography has been reportedly used as screening, some have questioned its sensitivity, although it is likely that the newest generation of devices may eventually be accurate enough for diagnosis in this situation (32). The most common lesion discovered is a dissection or intramural hematoma. The general consensus is that these lesions should be treated with either full anticoagulation or an antiplaquelet agent that should continue for 3 to 6 months. Occasionally pseudoaneurysms are seen and endovascular repair seems to be the evolving treatment modality for this lesion. Morbidity from this lesion remains high since many patients present with a deficit. In one large series there was a 26% mortality and only 31% of patients were discharged to home. However, in the asymptomatic group that was treated with either anticoagulation or antiplaquelet therapy, the failure rate was only 9% (33).

Traumatic amputations, although grossly impressive, typically are not life threatening. Traumatic amputations of a major extremity (digits not included) should be wrapped with warm gauze, and manual pressure applied while the protocol-driven...
trauma evaluation proceeds. Once other life-threatening injuries have been evaluated, the amputated stump may be examined. The treatment priority should be hemostasis and local debridement to remove large debris. Patients should be given tetanus toxoid if there are no other contraindications. Dressing changes should be initiated, and when stabilized, a formal, closed amputation can be undertaken, with an emphasis on leaving a functional stump for the patient to use.

HEMODIALYSIS

Although the annual mortality of patients on hemodialysis approaches 25%, many patients in the ICU are on chronic hemodialysis with functional fistulae. The extremity with the fistula should be preserved from invasive and noninvasive monitoring devices, and all IVs and central lines should be placed away from that extremity unless there are no other options. Because of the presence of the fistula, the extremity distal to it is at risk of ischemic events, and should be monitored closely. Additionally, tunneled catheters already in place should not be routinely used as a convenient intravenous line except in dire circumstances. These lines can often be a source of infection, and limiting their use to their intended purpose will decrease the chance of infection. When they are accessed for dialysis purposes, it is routine practice to “lock” the catheter with concentrated heparin to minimize the chance of a mechanical catheter complication. Flushing this heparin “lock” will systemically anticoagulate the patient, even if transiently.

SUMMARY

The care of the vascular patient in the ICU setting can be complex and challenging; it requires not only meticulous attention to detail, but also comprehensive knowledge of cardiovascular anatomy and physiology. As vascular disease is a systemic process, the patients typically have comorbidities that are symptomatic before surgery or unmasked with the stress of a surgical intervention. Regular and careful assessment of the patient can minimize, but not eliminate, the risks of perioperative complications.

PEARLS

- Peripheral vascular disease is one manifestation of a systemic process that is proinflammatory in nature and affects the coronary, cerebral, and peripheral vasculature.
- Ninety-three percent of patients undergoing the most common vascular procedures (AAA repair, carotid endarterectomy, peripheral bypass) have documented coronary artery disease; all patients should be managed accordingly.
- Patients with diabetes can manifest angina as nausea, diaphoresis, or “indigestion.”
- Any objective change in the assessment of distal perfusion—either by palpation of pulses or auscultation of Doppler signals—is a potential surgical emergency.
- All patients with obstructive or aneurysmal vascular disease should be placed on a β-blocker, an aspirin, and a statin, unless otherwise contraindicated.

- Compartment syndrome may be subtle, especially in the sedated ICU patient. The disappearance of pulses is a late finding. Clinicians should have a low threshold to perform fasciotomies.
- Colon ischemia after aortic surgery may present as hematochezia or melena, or may be more insidious: leukocytosis, thrombocytopenia, or fever.

References

HEAD INJURY

The Problem

Each year in the United States, nearly 1.4 million people sustain a traumatic brain injury (TBI) (1). Of those, an estimated 235,000 are hospitalized, and approximately 50,000 die. In children younger than 14 years, TBI results in 2,685 deaths per year with 37,000 hospitalizations and 435,000 emergency department visits. Worldwide, an estimated 57 million people are hospitalized with TBI with 10 million deaths annually (2). Compared to females, males have at least double the risk of TBI (1). In 1994, the National Center for Health Statistics (NCHS) reported that the TBI death rate for males was 3.3 times higher than for females (30.7 per 100,000 males vs. 9.3 per 100,000 females). Death rates were highest in persons aged 75 years or older (46.3 per 100,000) (3).

The leading causes of TBI are falls (28%), motor vehicle collisions (MVC) (20%), “struck by/against” events, which include colliding with a moving or stationary object (19%) and assaults (11%) (4). The rate of falls is highest with children younger than 4 years and adults age 75 years and older. MVC-related TBI is highest in adolescents age 15 to 19 years and results in the greatest number of TBI-related hospitalizations (1). Firearm-inflicted TBI has the highest mortality of 90.4% compared with 10.2% associated with falls (3,5). In the military population, TBI is often due to blast injuries with concussion, contusion, subdural hematoma, and axonal shear injury as the primary injuries (6). The Defense and Veterans Brain Injury Coalition estimates that during the recent wars, 40% of injured soldiers suffer from mild TBI (7).

Over 5.3 million Americans require assistance with activities of daily living as a consequence of the long-term effects on cognition and behavior with emotional and physical impairment following TBI (3,8). In the United States, lifetime costs of TBI, which include medical costs and lost productivity, are estimated at $60 billion annually (9).

Although outcome is chiefly determined by the severity of the initial injury, with appropriate neurologic support, secondary brain injury from hypotension, hypoxia, hyperthermia, and hyperglycemia may be prevented and decrease morbidity and mortality.

Prevention

Primary prevention of TBI includes strategies to increase public awareness to wear seat belts, use child safety seats, wear helmets, avoid driving while intoxicated, and install window guards and safety gates. For the elderly, available exercise programs, appropriate lighting, and handrails may prevent injury (10).

PRIMARY BRAIN INJURY

Primary focal neurologic injury following TBI includes hemorrhage into the subdural or epidural spaces, intraparenchymal hematomas (IPH) and cerebral contusions and lacerations. Primary diffuse injury includes subarachnoid (SAH) and intraventricular hemorrhage (IVH) and diffuse axonal injury (DAI).

Subdural Hematoma

Subdural hematomas (SDH) are more common than epidural hematomas (EDH) and were seen in 23% of patients with...
Section VIII: The Surgical Patient

Severe head injury entered into the Traumatic Coma Data Bank (TCDB) supported by the National Institute of Neurological Disorders and Stroke (NINDS) between 1980 and 1988 (11,12). SDH typically results from tearing of the bridging veins between the brain and the draining venous sinuses. The mechanism usually involves high-velocity acceleration and deceleration forces.

Imaging shows a crescent-shaped hyperdensity that follows the contours of the skull, and hemorrhages of SDH in anemic patients are isointense on initial CT and may be overlooked. Acute SDH carries a poor prognosis and is one of the most lethal of all head injuries. Fifty to 60% of patients with SDH die, and only 19% to 38% will achieve functional recovery despite surgical treatment (13,14). Early evacuation within the first 4 hours of injury decreased mortality from 90% to 30% in a single study of 82 consecutive comatose patients. This suggested that preventable secondary injury was the cause of the high mortality, despite multiple studies, these findings have not been replicated. More likely the high mortality is a result of the severity of initial forces from the primary mechanism of injury (15,16).

Epidural Hematomas

Epidural hematoma was found in 9% of 1,030 patients in the TCDB (17). An EDH requires a great impact force and is often associated with a skull fracture that disrupts the middle meningeal artery in the supratentorial space or causes injury to the venous sinuses in the posterior fossa (18). Classically, patients present awake and alert, known as the lucid interval, and quickly lapse into unconsciousness. Imaging shows a lenticular-shaped hyperdensity. With rapid evacuation, EDH has a relatively good prognosis, and the mortality rate is 5% to 10% (19). Factors determining mortality and functional outcome include age, best motor response on the Glasgow coma score (GCS), hematoma volume, and degree of midline shift (19-21). In patients with EDH who are comatose with either a very short or no period of wakefulness following injury, mortality can be as high as 40%. The motor score immediately before surgical evaluation is predictive; two thirds of patients with scores of 3 or less become vegetative or die (22).

Cerebral Contusions

Cerebral contusions result from direct impact of the brain on the skull. These are known as coup injuries. Alternatively, acceleration/deceleration injury of the brain against the contralateral side of the direct impact causes contrecoup injuries. The most common areas of contusion are the frontal, temporal lobes and occipital regions. These lesions are hyperintense areas within the parenchyma on CT scan and are more diffuse than IPH. As discussed below, secondary injury can result when contusions enlarge, which causes cerebral edema and intracranial hypertension (23). Clinical deterioration or elevation in ICP requires urgent repeat cerebral imaging.

Intraparenchymal Hemorrhage

Intraparenchymal hemorrhage (IPH), similar to a contusion, is hyperintense on CT scan. It is a focal process and less diffuse than a contusion as it is caused by direct vascular injury or by stretching of the vessels with brain shift and distortion. Hemorrhage in the upper brainstem (midbrain and pons), known as Duret hemorrhages, can also occur with rapidly evolving transtentorial herniation and may be due to stretching of the perforating arterioles or from venous thrombosis and infarction. Spontaneous causes of IPH are hypertensive hemorrhagic stroke, or hemorrhaging due to arteriovenous malformation, aneurysm, amyloid angiopathy, or tumor. In the setting of hemorrhage in the basal ganglia, cerebellum, or thalamus, the clinician should consider the differential of spontaneous IPH as a possible cause of the traumatic event, rather than the result.

Subarachnoid Hemorrhage

Trauma is the most common cause of subarachnoid hemorrhage. It occurs in 21% to 53% of patients with severe TBI and worsens outcome (24,25). In contrast to aneurysmal SAH, traumatic SAH (tSAH) is less likely concentrated in the basal cisterns and is usually found over the hemispheric convexities. The presence of tSAH in the basal cisterns carries a positive predictive value of unfavorable outcome of up to 70% (26).

Intraventricular Hemorrhage

Intraventricular hemorrhage (IVH) in isolation is not commonly seen in closed head injury (CHI). However, like traumatic SAH, it has been associated with worsened outcome (25). Obstructive hydrocephalus may result and may require cerebrospinal fluid (CSF) diversion by external ventricular drainage.

Diffuse Axonal Injury

Diffuse axonal injury (DAI) occurs in approximately half of patients with severe CHI (12). Sudden acceleration-deceleration impact causes rotational forces and shearing injury to axons. The axon may not be entirely transected, but axoplasmic transport is disrupted causing swelling and disconnection (27). A retrograde ball forms, and the axon undergoes Wallerian degeneration. Since axonal degeneration may be a secondary injury process, eventually pharmacologic strategies to intervene may be developed. Outcome is worsened with severe DAI (28,29). Although microscopic neuronal injury cannot be seen on imaging studies, the diagnosis is best made with MR imaging with gradient echo and susceptibility-weighted sequences that detect blood products from the capillary injury and leak that accompanies DAI (30).

Physiologic Principles

The Monro-Kellie hypothesis describes the skull as a semi-closed compartment containing brain and interstitial fluid (80%), CSF (10%), and blood (10%). Compensatory mechanisms to decrease cerebral blood or CSF volume become active in pathologic conditions where intracranial volume and pressure increase. For example, with hemorrhage or edema after TBI, reductions in CSF production and cerebral blood flow (CBF) are seen. Once these compensatory mechanisms are
overwhelmed, depending on the compliance (volume/pressure relationship) of the intracranial contents, pressure will increase. Patients with atrophy are able to tolerate larger volumes before the intracranial pressure (ICP) increases. A young patient without much atrophy has low cerebral compliance, which increases the risk for early intracranial hypertension and potential cerebral herniation.

Cerebral perfusion pressure (CPP) is determined by the difference of mean arterial pressure (MAP) and ICP. When ICP monitoring is used, the CPP supplants MAP goals in the intensive care unit (ICU). Normal ICP is less than 10 mm Hg. In a study in which ICP and CPP were closely evaluated with respect to outcome, the most powerful predictor of neurologic worsening was the presence of intracranial hypertension defined as an ICP of 20 mm Hg or greater. As long as CPP was maintained greater than 60 mm Hg, CPP did not correlate with neurologic worsening (31). Current Brain Trauma Foundation (BTF) guidelines recommend CPP greater than 60 mm Hg and ICP between 20 and 26 mm Hg. Prior to placement of an intracranial monitor, recommendations are to maintain a MAP of 90 mm Hg or greater (32,33).

EVALUATION

On arrival at the ICU, the initial focus is on respiratory and hemodynamic stability. This will be discussed below. In addition to the usual general examination, in the neurologically injured patient, the evaluation includes an examination of the head for scalp lacerations, which can be a major source of bleeding and orbital, facial, and depressed skull fractures. Evidence for basilar skull fractures include periorbital ecchymoses known as raccoon eyes indicative of frontal skull base injury or postauricular ecchymoses known as the Battle sign seen with middle fossa or temporal bone fractures. Cervical spine precautions are maintained in these patients and will be discussed later.

During the evaluation and observation of the TBI patient, repeated neurologic monitoring includes the vital signs with special attention to extremes in blood pressure. Hypotension may result in secondary injury, whereas hypertension, not always associated with bradycardia, can be a sign of impending cerebral herniation. If ICP rises, cerebral autoregulation elevates MAP to maintain an adequate CPP.

A rapid neurologic assessment includes level of consciousness and ability to speak or understand language by assessing the ability to follow simple commands or to at least mimic clear hand signals. Vision is assessed by asking the patient to count fingers placed in the right or left visual field with one eye covered, or to mimic finger movements. In patients with a lower level of consciousness, vision is assessed by blinking to a visual threat. Pupillary response to light (cranial nerves [CN] II, III), corneal reflex (CN V, VII), and gag and cough (CN IX, X) responses assess cranial nerve and brainstem function. Oculofacical maneuvers (CN III, VI, VIII) should not be performed in patients who have a risk of cervicospinal fracture. Ice water caloric response (CN III, VI, VIII) can be performed if the tympanic membranes are intact. The head of the bed should be up at 30 degrees, and 60 to 90 mL of ice water instilled into the oric canal. A normal response is a slow lateral deviation to the side stimulated with ice water and nystagmus with the fast phase to the opposite ear. Absence may be caused by medica
tions or brainstem injury. Motor response is assessed by verbal commands to move the limbs. In patients with lower levels of consciousness, motor responses are elicited by painful stimuli delivered to the sternum or fingernail bed. During painful stimula
tion, the examiner should also reassess facial movement for asymmetry. If flexion is noted, pain is applied to the supraaur
crinal ridge or by trapezius squeeze to test for localization. In the lower extremities, it is important to recognize a triple flexion response, which is described by the flexion of the ankle, knee, and hip. Triple flexion is a spinal reflex to painful stimulation of the legs or feet. It is stereotyped in appearance independent of the location of pain delivery on the lower extremity. It does not reflect brainstem or upper spinal cord function. Patients who are brain dead or with higher cord complete lesions can triple-flex lower extremities.

The clinical evaluation in TBI includes a GCS, which was first developed and introduced in 1974 to assess the depth and duration of impaired consciousness (34). The GCS has been divided into three categories: (i) A GCS of 8 or less is defined as severe head injury, (ii) a GCS of 9 to 12 represents moderate head injury, and (iii) a GCS of 13 to 15 is mild or minor (Table 83.1). The best GCS following adequate fluid resuscitation and stabilization has previously been shown to be predictive of outcome (35). Interrater variability is often minimal, but can exist (36). In unanesthetized patients, field and arrival GCS correlates highly. A change between the field and arrival GCS can be predictive of outcome (37).

Laboratory evaluation of patients with head injury include complete blood count with platelet counts, partial thromboplastin time (PTT), prothrombin time (PT), electrolytes with

<table>
<thead>
<tr>
<th>Score</th>
<th>Eye opening</th>
<th>Best verbal</th>
<th>Best motor</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No response</td>
<td>No response</td>
<td>No response</td>
</tr>
<tr>
<td>2</td>
<td>To pain</td>
<td>Incomprehensible</td>
<td>Extension</td>
</tr>
<tr>
<td>3</td>
<td>To speech</td>
<td>Inappropriate</td>
<td>Flexor</td>
</tr>
<tr>
<td>4</td>
<td>Spontaneous</td>
<td>Disoriented</td>
<td>Withdraws to pain</td>
</tr>
<tr>
<td>5</td>
<td>Oriented</td>
<td>Oriented</td>
<td>Localizes pain</td>
</tr>
<tr>
<td>6</td>
<td>—</td>
<td>—</td>
<td>Obey command</td>
</tr>
</tbody>
</table>

blood urea nitrogen, creatinine, glucose, and liver function tests to assess for renal insufficiency or liver dysfunction, which may impair clotting ability. A toxoscopy screen including a blood alcohol level is essential to assist in evaluating for other causes of altered mental status and to determine whether delirium tremens may be a factor in the following days of ICU care. Arterial blood gas (ABG) and lactate levels help assess volume status and whether ventilation is adequate.

### Imaging

The initial imaging, often coupled with the neurologic exam, determines the need for acute neurosurgical intervention. Neurosurgical guidelines have been established for focal intracranial lesions (38–40). Noncontrast head CT is the fastest, most widely available noninvasive imaging technique to determine this. All patients with altered mental status and/or focal neurologic findings should have an initial CT scan performed. In minor head injury, a CT scan may not be necessary if the exam is normal and the GCS is 15 unless the patient is older than 60 years, has a headache, emesis, drug or alcohol intoxication, deficits in short-term memory, physical evidence of trauma above the clavicles, or seizures (41).

#### Evolving Injury and Repeat Head CT

Progressive intracranial hemorrhage consistent with an evolving contusion is seen in 14% to 38% (42–44). Although worsening CT findings do not necessarily require treatment, 34% of patients may require neurosurgical intervention including ICP monitoring or craniotomy subsequent to the findings on a repeat scan (23,45–50). A significant risk factor is early initial imaging within 2 hours of injury (24,42,50), and common community standards are to repeat a CT scan within 12 to 24 hours of the initial imaging. In stable patients without clinical neurologic deterioration, the utility of repeat imaging is debated since it is unlikely that neurosurgical intervention will be necessary (24,47,50). Other independent risk factors for progression include associated SAH, SDH, older age, and prolonged partial thromboplastin (23,42,44,50,51). A large initial contusional or intraparenchymal hemorrhage size and effacement of cisterns is strongly predictive of failure of nonoperative management (44).

### Secondary Brain Injury: Prevention, Treatment, and Management

Following immediate impact and anatomic damage, secondary damage at the cellular level from inflammation, edema, free radicals, and excitatory neurotransmitters can worsen outcome. Contributing factors include hypoxia, hyperthermia, seizures, fever, and intracranial hypertension. Immediate postinjury care focuses on the prevention of these problems.

#### Hypoxia and Respiratory Management

Hypoxia, defined as a PaO\textsubscript{2} less than 60 mm Hg or O\textsubscript{2} saturation less than 90% can independently increase mortality from 27% to 50% and increase poor outcome from 28% to 71% (32,33). Early intubation can prevent aspiration and minimize hypoxic and hypercapnic events (54) and is recommended by the Advanced Trauma Life Support (ATLS) guidelines from the American College of Surgeons (55) and the Brain Trauma Foundation Traumatic Brain Injury prehospital guidelines (56). A GCS of 8 or less is the usual threshold for endotracheal intubation.

In the prehospital setting, rapid sequence intubation has been associated with increased mortality (57,58). This may result from decreased cerebral perfusion due to hyperventilation-induced hypocapnia. Positive pressure ventilation may cause hypotension in a hypovolemic patient if central venous return is impaired by high intrathoracic pressures (59). Intubation presents a high-risk procedure for secondary neurologic injury. Sedative/hypnotic medications and bag/mask ventilation with positive pressure ventilation contribute to hypotension and hypercapnia during induction. In addition, direct laryngoscopy causes a marked, transient increase in ICP. Intravenous lidocaine can blunt this ICP response (60).

Hyperventilation with resultant hypocapnia causes cerebral vasodilatation and a reduction in CBF (61–63). Prolonged hypocapnia appears to slow neurologic recovery (64). Propylactic hyperventilation of PaCO\textsubscript{2} less than 35 mm Hg should be avoided, although PaCO\textsubscript{2} as low as 30 mm Hg may be necessary for brief periods for immediate treatment of intracranial hypertension. Options to identify cerebral ischemia in the setting of hyperventilation include the use of jugular venous oxygen saturation, arterial jugular venous oxygen content differences, brain tissue oxygen monitoring (see below), or cerebral blood flow monitoring (32). To achieve adequate ventilation, positive end-expiratory pressure (PEEP) may be necessary. Positive end-expiratory pressure affects CPP and ICP when the lung is compliant and the chest wall is not. The high lung compliance allows for an increased intrathoracic volume, which in the setting of a low compliance chest wall increases intrathoracic pressures. The high intrathoracic pressure decreases cerebral venous outflow, which will increase ICP (65,66). When intrathoracic pressure is elevated, cardiac venous return is diminished and results in lowered mean arterial pressure and CPP. Pulmonary infections were seen in 41% of patients registred in the Traumatic Coma Data Bank and were an independent predictor of unfavorable outcome (67). Bedside management includes adequate pulmonary toilet and strategies such as elevation of the head of the bed (HOB) to decrease the risk for ventilator-associated pneumonia (VAP). In patients with intracranial hypertension, during endotracheal suctioning, adequate sedation is necessary to prevent an increase in ICP (68).

#### Neurogenic Pulmonary Edema

In addition to hyperventilation and aspiration from poor airway protection, a less frequently recognized cause of hypoxemia following TBI is neurogenic pulmonary edema (NPE). Neurogenic pulmonary edema results from central sympathetic stimulation. Pretreatment with adrenergic-blocking agents prevents experimental NPE (69). Experimental lesions in the hypothalamus (70), bilateral nucleus tractus solitarius (71), and the ventrolateral medulla (72) can produce NPE. Traumatic brain injury causes a sympathetic discharge, which increases systemic and pulmonary vascular pressures. The resultant increase in pulmonary capillary pressure increases the hydrostatic pressure
and causes pulmonary capillary injury; this in turn causes leak-
age of fluid and protein, and pulmonary hemorrhages (73–75).

Clinical signs include dyspnea, tachypnea, tachycardia, and
chest pain if the patient is awake. Rates are present on chest auscultation. Laboratory results show hypoxemia and a mild
leukocytosis. Chest radiography shows a bilateral alveolar fill-
ing process (76). Pulmonary capillary wedge pressures and pul-
monary artery pressures can be elevated or normal. There are
two distinct forms of NPE. The classic form appears early,
within minutes to a few hours after acute brain injury. A de-
layed form of NPE slowly progresses over 12 to 72 hours
following injury. Treatment is supportive and often requires
supplemental oxygen and positive pressure ventilation. Dobu-
tamine may be effective by decreasing cardiac afterload and
increasing cardiac contractility (77).

Contraction Band Necrosis

In CBN, cardiac enzymes are often elevated and may be dif-
cult to differentiate from an acute coronary syndrome. How-
ever, the treatment for CBN is vastly different and typically
includes observation for arrhythmias and blood pressure sup-
port in contrast to reperfusion therapy with an acute ischemic
myocardial infarction. Clinical differentiation typically relies
on the recognition of patients with higher risk for coronary
artery disease such as older age, hypertension, diabetes, and
hyperlipidemia rather than a young patient with massive head
injury.

Posttraumatic Vasospasm

Following TBI, focal cerebral ischemia as a result of posttrau-
matic vasospasm can occur in 24% to 36% of patients (90,91)
and may manifest as lateralizing neurologic deficits such as
hemiparesis and aphasia between 2 to 37 days following in-
jury (92,93). In patients with severe TBI, small studies have
reported incidence as high as 82% (94). Transcranial Doppler,
while reasonably specific, is not a sensitive test for vasospasm.
If vasospasm is suspected, cerebral angiography can confirm
the diagnosis. The effectiveness of treatment of posttraumatic
vasospasm with modalities used following aneurysmal SAH
(e.g., hypervolemic, hypertensive therapy or nimodipine) has
not been assessed.

Fever

Hyperthermia accelerates neuronal injury by increasing basal
energy requirements (neuronal discharges), excitatory neuro-
transmitters, free radial production, calcium-dependent pro-
tein phosphorylation, ICAM-1 and inflammatory responses,
DNA fragmentation, and apoptosis, causing blood–brain bar-
rier changes as seen by extravasation of protein tracers (95,96).
Despite this, previous TBI studies of prophylactic moderate hy-
othermia (32–33 °C) and their meta-analyses were not able
to show improved outcome (97–99). This may be due to
significant intercenter variability in the management of MAP,
CPP, fluids, and vasopressors (97,100).

In the individual patient, therapeutic hypothermia lowers
ICP by reducing the cerebral metabolic rate 7% for each degree
Celsius decrease. This treatment can be life-saving and result
in reasonable neurologic recovery (101). Pentobarbital coma
and/or neuromuscular blockade may be necessary to achieve
cooling without shivering. New techniques for intravascular
and topical cooling are available. Although complications of
hyperthermia can include increased risk of cardiac arrhyth-
mas, hypotension, bradycardia, thrombocytopenia, and pneu-
monia, in studies evaluating hypothermia in cardiac arrest pa-
tients, there was no statistical increase in these adverse events
(102,103).

Hyperglycemia

Hyperglycemia causes brain tissue acidosis (104), and early
hyperglycemia has been associated with worsened neurologic
outcome following TBI (105,106). It is not fully understood

Hypotension

Hypotension, defined as systemic blood pressure (SBP) less than
90 mm Hg, independently worsens mortality (78,79). Trau-
matic Coma Data Bank reports hypotension was present in
29% of patients and doubled mortality from 27% to 55% (67,78).
If the patient presented in shock, mortality was 65%
independent of age, admission GCS motor score, hypoxia, or
associated severe extracranial trauma. Adequate fluid resusci-
tation with euvolemia is essential. Independent of ICP, mean
arterial pressure or CPP or negative fluid balance of nearly 600
mL was associated with poorer outcome (80). Guidelines rec-
commend adequate fluid resuscitation and blood pressure sup-
port to maintain MAPs greater than 90 mm Hg (33). Once an
ICP monitor is placed, the optimal blood pressure is determined
by a CPP of 60 mm Hg or greater.

Chapter 83: Neurologic Injury: Prevention and Initial Care

Contraction Band Necrosis

Chapter 83: Neurologic Injury: Prevention and Initial Care
whether the hyperglycemia is causative or is a marker for severity of injury and subsequent poor outcome. Although tighter glucose control with TBI is theoretically reasonable, based on current evidence, it is not clear that aggressive treatment of hyperglycemia in the neurologic and neurosurgical population improves outcome.

In the ICU setting, where glycemic control often uses insulin infusion or injection, patients with acutely altered mental status should be urgently evaluated for hypoglycemia.

Coagulopathy

Brain is rich in tissue thromboplastin and following head injury, increased tissue thromboplastin activity in the frontal, parietal, and temporal lobes activates the coagulation cascade and causes a disseminated intravascular coagulopathy (107). The Traumatic Coma Data Bank reported that 19% of patients were coagulopathic (67). Although initial evaluation may show thrombocytopenia in 14% and coagulopathy in 21% of TBI patients, in ensuing days, disseminated intravascular coagulation can be seen in 41% to 60% of patients with blunt brain injury (108,109). It is more common in patients with penetrating head trauma (110).

Abnormalities in PT, PTT, or platelet count have been associated with 55% of patients with progressing hemorrhage after TBI (111,112). Associated coagulopathy and thrombocytopenia increases mortality in TBI (108,109,112). Although there are no guidelines for correction of coagulopathy or thrombocytopenia, usual practice is to transfuse platelets for values <100,000 per mL and fresh frozen plasma for an elevated PTT or a PT international ratio (INR) of 1.5 or more. Other alternatives such as activated factor VII or prothrombin complex concentrate may be effective emergently (110).

Intracranial Pressure Monitoring and Management

Normal ICP is less than 10 mm Hg. The Traumatic Coma Data Bank reports that 72% of patients with severe TBI had ICPs above 20 mm Hg (113). Since multiple studies show worsened outcome with ICP above 20 to 25 mm Hg, published guidelines use this as the threshold to treat (32,33)

Maneuvers for management of ICP begin with those with fewer potential side effects and progress to more invasive treatments with higher complication risk (Fig. 83.1). Elevation of the head of bed to greater than 30 to 45 degrees not only decreases the risk of ventilator-associated pneumonia but can facilitate cerebral venous drainage and lower ICP. In orthostatic, hypovolemic patients, however, head of bed elevation can lower MAP. Adequate fluid resuscitation is necessary.

![Intracranial hypertension](attachment:fig83.1.png)

**FIGURE 83.1.** Algorithm for management of intracranial hypertension. CCP, cerebral perfusion pressure; CSF, cerebrospinal fluid; ICP, intracranial pressure.
Adequate pain therapy with opioids and adequate sedation with sedative-hypnotics decrease ICP. Constant infusion may be hemodynamically better tolerated than bolus administration.

Prophylactic or sustained hyperventilation of PaCO₂ less than 35 mm Hg may be harmful and should be avoided. In the situation of impending herniation or refractory intracranial hypertension, decreasing PaCO₂ to 30 mm Hg for transient “rescue” therapy will give the practitioner time to initiate other maneuvers to lower ICP.

Osmotic therapy is a mainstay in ICP management. Mannitol or hypertonic saline are both effective. However, repetitive dosing by the above agents may worsen the volume of injured brain (114–116). To avoid this, osmotic therapy similarly to hyperventilation, may also be best used as rescue therapy until more definitive therapy is implemented. Doses of 0.25 g/kg to 1 g/kg of mannitol are effective. The lower dose drops ICP and may decrease the risk of vasogenic edema seen with multiple dosing (114). Due to potential renal damage, maximal mannitol dosing traditionally has been when serum osmolality reaches 320 mosm/kg, but this convention is under debate. An osmolar gap of greater than 10 from baseline may be a better indicator for maximizing mannitol administration (117,118). Hyperosmolar saline may be more effective and have a longer duration of action on lowering ICP than mannitol (116,119). Doses include 250 mL of 3% or 7.5% saline or 30 mL of 23.42% saline (120).

Osmotic therapy is best administered through a central venous access as it may sclerose veins. When deciding which osmotic agent to use, elevated ICP with low fluid status would be best treated with hypertonic saline. Studies evaluating the use of hypertonic saline compared to conventional fluids for refractory intracranial hypertension, decreasing PaCO₂ to 30 mm Hg for transient “rescue” therapy will give the practitioner time to initiate other maneuvers to lower ICP.

Neuromuscular blockade also assists in cooling the patient. As noted above, hypothermia is useful in refractory intracranial hypertension. Temperatures of 32°C to 33°C can be well tolerated. The combination of neuromuscular blockade and cooling appears to have a high risk for pneumonia. The patient is unable to effectively clear secretions, and empiric pulmonary toilet with frequent suctioning is often necessary. Barbiturate-induced coma lowers the cerebral metabolic rate. This results in lowered cerebral blood volume and ICP. Thiopental or pentobarbital infusions can be used. Thiopental in long-term infusion, because of its lipophilicity, may take over a week to clear after the infusion is stopped. For pentobarbital, 20 mg/kg is given as a slow loading dose followed by a maintenance infusion of 1 mg/kg/hour. The loading dose may significantly lower mean arterial pressure. Often fluids and vasopressor administration may be necessary. Electroencephalography (EEG) is critical to titrating the dose during barbiturate coma. Although the infusion can be titrated to ICP effect, if the EEG is isoelectric, there is little to be gained in the way of ICP control by increasing the infusion. At this point, worsening side effects result from increasing the infusion. These include hypotension from peripheral vasodilation, decreased cardiac inotropy, and ileus. Also cough reflex is diminished and decreased bronchociliary activity and slowed leukocyte chemotaxis increase the risk for pneumonia. A benefit of barbiturate coma is a quiescent hypothalamus that no longer modulates body temperature. Hypothermia can often be achieved without the need for neuromuscular blockade since shivering is diminished.

Loop diuretics have been used to help manage ICP by decreasing CSF production in the choroid plexus. Loop diuretics will decrease volume status. Unless the patient is hypervolemic, CSF diversion is a more effective method of lowering ICP.

Other therapy for refractory intracranial hypertension requires neurosurgical intervention. Placement of an external ventricular drainage allows for CSF drainage. Hemisrectomy may be life-saving and a viable option depending on the patient. Case series of 19, 23, and 51 children at three different centers had mortalities of 30% to 31.4%. Favorable outcome with return to school and functional independence was reported in 68% to 81%. Eighteen percent to 21% were severely disabled and dependent on caregivers (123–125). To date, there are no randomized controlled trials to evaluate its effectiveness and outcome in the setting of TBI.

**Brain Tissue Oxygenation**

Hypoxic brain injury causes secondary damage. To monitor and help prevent this injury, new modalities to evaluate cerebral oxygenation have been developed. Jugular bulb oximetry (SjvO₂) is a global measure of the balance between oxygen delivery to the brain and oxygen consumption. Local brain tissue partial pressure oxygen (PBrO₂) is measured by a polarographic Clark-type microcatheter. An increase in cerebral oxygen delivery is reflected by increases in SjvO₂ and PBrO₂. Oxygen delivery to the brain is manipulated by increases in blood pressure, cardiac output, and red blood cell transfusion (126). Normobaric hypoxia has not shown to improve cerebral oxygen metabolism on PET imaging, and the use of 100% oxygen is not supported by the available literature (127). Optimal SjvO₂ is generally accepted as 50% oxygen saturation (128). The optimal PBrO₂ has not been established. Various studies show worsened outcome in patients with mean PBrO₂ less than 15 mm Hg. Other thresholds include 25 mm Hg (129–132). Mortality was significantly decreased and functional outcomes improved in one study comparing 25 patients treated by traditional ICP/ICP-guided therapy to 28 patients with therapy targeted to a PBrO₂ greater than 25 mm Hg (113). No randomized controlled trials of SjvO₂ or PBrO₂-targeted therapy have been performed to establish effectiveness. Current guidelines for management of TBI do not recommend the use of these modalities. Cerebral microdialysis evaluating the biochemical byproducts of ischemia such as increased lactate and glutamate and lactate/pyruvate ratio as another potential technique to assist bedside care, but has not yet reached clinical use (134).

**Antibiotic Prophylaxis**

Fractures of the skull base and severe facial trauma can result in a CSF leak. Various studies report incidences of 2.6% to 4.6%...
of all patients with basilar or facial fractures (135,136). In one study, otorrhea was three times more common than rhinorhea (135). Approximately 50 percent of CSF leaks stop within 5 days (137). The risk of bacterial meningitis is approximately 12% to 21%. Studies conflict as to whether prophylactic antibotics decrease the risk of infection, and there are no guidelines or recommendations (137,138). Constant surveillance for meningitis is essential.

In the setting of CSF leak, if the spine is stable and blood pressure is adequate, the head of the bed should be elevated to facilitate leak closure. Stool softeners help avoid vigorous Valsalva maneuvers that may worsen the leak. Neurosurgical intervention with CSF diversion (i.e., lumbar drain or external ventricular drain) or surgical closure may be necessary if the leak persists.

Following penetrating head trauma, a CSF leak is the primary predictor of intracranial infection. Infection is seen in 38% to 63% of CSF leaks after military-related penetrating cerebral trauma (139-141). Current recommendations are to treat with empiric broad-spectrum antibiotics following penetrating brain injury (141,142). Optimum duration and regimen are unknown.

For clean neurological procedures, such as external ventricular drain placement or craniotomy, guidelines have been established by the Surgical Infection Prevention and Surgical Care Improvement Projects that recommend cefazolin within 1 hour prior to surgical incision (143).

**Posttraumatic Seizures**

Early posttraumatic seizures occur within 7 days of injury. These manifest as weakness, incoordination, emotional lability, and long-term cognitive, motor, sensory, and emotional deficits. Survivors of traumatic brain injury variably suffer from late seizures, and it is not recommended for closed or penetrating head injury (33,147-149). Of note, valproate showed no benefit for seizures following brain injury and had a trend to higher mortality (150). There is no evidence that continuing prophylactic antiseizure medications beyond a week prevents late seizures, and it is not recommended for closed or penetrating head injury (33,149).

**Thromboprophylaxis**

In the general postoperative neurosurgical population, the risk for deep venous thrombosis (DVT) is 3% to 14% (131-134). Following major head injury, the risk for DVT is as high as 54% (155). The Brain Trauma Foundation has no recommendations for thromboprophylaxis (33). However, current recommendations from the seventh conference of the American College of Chest Physicians (ACCPC) are that patients undergoing major neurosurgical procedures receive thromboprophylaxis in the form of intermittent pneumatic compression (IPC) devices with or without graduated compression stockings (GCP) (grade 1A) (154). Acceptable alternatives include subcutaneous unfractionated heparin (grade 2B) or postoperative low-molecular-weight heparin (LMWH) (grade 2A). In high-risk neurosurgery patients, the combination of mechanical and pharmacologic prophylaxis is recommended (grade 2B). In patients who are not neurosurgical candidates, a grade 1A recommendation is that trauma patients receive LMWH as soon as it is considered safe to do so. In the meantime, mechanical prophylaxis, intermittent pneumatic compression and/or graded compression stockings should be used (grade 1B). Inferior vena cava filters are not considered acceptable primary prophylaxis in trauma patients (grade 1C).

**Nutrition**

Following severe traumatic brain injury, patients have a hypermetabolic, catabolic state with rapid weight loss associated with a negative nitrogen balance and protein wasting. In experimental models of TBI, 3 hours after injury morphologic changes are seen in the gut mucosa that include shedding of epithelial cells, fracture of villi, focal ulcers, fusion of adjacent villi, mucosal atrophy, and edema in the villous interstitium and lamina propria. On electron microscopy, there is a loss of tight junctions between enterocytes, damage of mitochondria and endoplasm, and apoptosis of epithelial cells (156). These changes in gut permeability increase bacteria translocation and endotoxin, which increases the risk of the systemic inflammatory response. Arginine and glutamine modulate gut permeability. There is some debate whether glutamine should be used in brain injury patients due to the potential increase in cerebral glutamate with neuroexcitatory properties and cell damage. Early parenteral or enteral nutrition can speed neurologic recovery and decrease disability and mortality (157-160). Early enteral feeding may have benefit over parenteral feeding by protecting against intestinal apoptosis and atrophy (161) and decreasing infection clinically (162). Early enteral nutrition with glutamine and probiotics may decrease the infection rate and length of ICU stay (163). Current guidelines in severe TBI are to replace 140% of resting metabolism in nonparalyzed patients and 100% of resting metabolism in paralyzed patients within 7 days. Either enteral or parenteral nutrition should contain at least 15% of calories from protein (32).

**Stress Gastritis**

Stress gastritis was seen in 91% of 44 comatose mechanically ventilated patients within 24 hours of head injury. Lesions were most commonly seen in the fundus and body of the stomach (164). Mucosal ulceration is typically prevented by maintaining intraluminal pH above 5 or by H2 receptor blockade (165). In TBI patients, stress ulcer bleeding prophylaxis is typically given. Sucralfate, H2-antagonists, proton pump inhibitors, or antacids in patients with mechanical ventilation may be effective; none are recommended over another with regard to risk of ventilator-associated pneumonia.

**Prognosis**

Survivors of traumatic brain injury variably suffer from long-term cognitive, motor, sensory, and emotional deficits. These manifest as weakness, incoordination, emotional lability,
impulsivity, and difficulty with vision, concentration, memory, judgment, and mood. Nearly 5.3 million in the United States live with disabilities as a result of TBI (4). When the postresuscitation GCS is not complicated by medications or intubation, approximately 20% of patients with GCS 3 will survive and 8% to 10% will have moderate to good recovery such that they are able to live independently (166). Despite this, 34% to 47% of "minor" head injury patients cannot return to work or their previous lifestyle (167,168). Independent predictors of outcome include older age at time of injury, the postresuscitation Glasgow coma score, injury severity score (ISS), pupillary response on admission, and CT scan findings of diffuse edema, subarachnoid hemorrhage, subdural hematoma, partial obliteration of the basal cisterns, or midline shift (169-174). The Traumatic Coma Data Bank reports a mortality rate of 76% for severe TBI patients with postresuscitation GCS of 3 to 18% for patients with GCS of 6 to 8, respectively. Overall mortality was 36% in 746 patients (12). In another study of 1,311 head-injured patients, the highest mortality was associated with spinal cord injury, obstructed airway, difficulty breathing, and shock, although none of these was independently predictive of survival when adjusted for GCS (175).

**SPINAL CORD INJURY**

The annual incidence of acute spinal cord injury (ASCI) in North America is between 27 and 47 cases per million (176). The United States has approximately 11,000 cases per year (177). Those at highest risk are young males. ASCI is most commonly caused by MVC (46.9%) followed by falls, acts of violence, particularly gunshot wounds, and recreational sporting activities (177).

**Management**

Primary spinal cord injury (SCI) results from cord compression from discs, bone, ligament, or hematoma or from distractive forces such as flexion, extension, dislocation, or rotation, which cause shearing of the neuronal axons or vasculature. Similar to head injury, the spinal cord undergoes both primary and secondary injury. Secondary injury results from systemic and local vascular insults, which may be a result of hypotension, electrolyte changes, edema, and excitotoxicity (178).

**Immobilization**

Three percent to 25% of spinal cord injuries may occur after the initial traumatic event, and nearly 20% of SCI involves multiple noncontiguous vertebral levels (179,180). For this reason, early management of patients with SCI includes immediate immobilization of the entire spine. Spine immobilization can be uncomfortable and carries potential morbidity of pressure sores and risk of aspiration, and may limit respiratory function; however, it is the usual treatment for all patients with a mechanism of injury that may cause spinal injury (179). Various methods for complete spine immobilization can be used, but a rigid cervical collar with supportive blocks on a rigid backboard is effective.

**Radiologic Evaluation**

No cervical radiologic evaluation is recommended in awake, alert, nonintoxicated trauma patients who have no neck pain or tenderness unless there are significant associated injuries that would interfere with their history and physical examination (181). In patients with neck pain or tenderness, a combination of radiologic techniques may be necessary to clear the cervical spine of significant injury. The CT scan is better than MRI for evaluating bones; however, flexion/extension radiographs in an awake patient or MR within 48 hours of injury can best detect ligamentous injury. Newer-generation CT scanners are sensitive, and some centers no longer perform plain radiographs. However, current American Association of Neurological Surgeons (AANS) standards are for an anteroposterior, lateral, and odontoid cervical spine series supplemented with CT scan in the initial evaluation. In an awake patient with neck pain, options to clear the cervical spine include normal flexion/extension films or a normal MRI within 48 hours of injury. In obtunded patients with normal cervical spine films, options to clear the spine include (a) dynamic flexion/extension studies under fluoroscopy, (b) normal MRI obtained within 48 hours of injury, or (c) the discretion of the treating physician (182).

**Hemodynamic Support**

Systemic hypotension, which contributes to secondary spinal cord injury, can result from trauma-related hypovolemia and from neurogenic shock (183-186). Neurogenic shock is defined as the loss of sympathetic innervation that causes loss of peripheral vasocstriction and cardiac compensatory mechanisms of tachycardia and increased stroke volume and cardiac output. In experimental models, microvascular spasm, thrombosis, and rupture disrupt spinal cord vascular autoregulation and make the spinal cord more susceptible to systemic hypotension. This worsens spinal cord ischemia several hours after injury (186).

Augmentation of mean arterial pressures to 85 to 90 mm Hg for 3 to 7 days postinjury has been shown to reduce morbidity and mortality and shorten length of stay (187-190).

Treatment typically includes volume resuscitation with crystalloids or red blood cell transfusion if the patient is anemic. Vasooactive medications such as norepinephrine, dopamine, and phenylephrine are used as needed. Volume-resistant hypotension is fivefold more common among patients with complete spinal cord injury above the thoracic sympathetic innervation (188). In the subset of patients requiring vasopressors and intropes, central venous catheters and invasive monitoring with arterial catheters should be used. Some investigators use pulmonar y artery catheters to establish volume status. No studies have been performed to compare these modalities.

**Surgical Intervention**

The timing of surgical decompression, reduction of bony structures, and fusion in the treatment of acute spinal cord injury remains in debate. A multicenter retrospective study of 385 patients at 36 North American centers applied decompression traction in 47% of patients with cervical injury and successfully decompressed the cord in 42% of patients. Neurologic deterioration occurred in 8.1% of cases with traction. Surgical intervention was undertaken in 65.4% of patients. Surgery was performed within 24 hours of trauma in 23.5%, between 25 to 48 hours in 15.8%, between 48 and 96 hours in 19%, and more than 5 days in 41.7% (191). This wide variation confirmed that there is little agreement on optimal timing. Early surgery may be associated with shorter hospitalizations and reduced pulmonary complications (192-194).
A meta-analysis of studies performed between 1986 to 2000 of 1,687 eligible patients comparing early decompression to conservative management or later decompression concluded that there was weak evidence that surgery within 24 hours improved neurologic outcome (195). Multicenter studies attempting to evaluate outcome of early decompression within 8 or 12 hours are much needed but have been difficult to perform due to the barriers to operative treatment within this time frame (196). At this time, surgical intervention of patients with incomplete injury with persisting compression from dislocation with bilateral locked facets, burst fracture, or disc rupture, especially in patients with neurologic deterioration, is considered a practice option (193).

Pharmacologic Intervention
Following acute spinal cord injury, a cascade of biochemical processes is activated that produces excitatory amino acids, calcium fluxes, free radicals, acidosis, protein phosphorylation, phospholipases, and apoptosis, which can further injure surrounding tissue (186). Pharmacologic agents targeted to interrupt this cascade may provide neuroprotection by preventing secondary injury. Naloxone, GM-1 ganglioside, and methylprednisolone have undergone randomized clinical trials to examine their effects following spinal cord injury. Despite multiple trials evaluating the use of methylprednisolone, meta-analyses do not agree with respect to recommendations for its use (197,198). A multicenter National Acute Spinal Cord Injury Study (NASCIS) II trial in 1985 of 30 mg/kg bolus followed by 5.4 mg/kg per hour infusion for 23 hours given within 8 hours of injury reported significant improvement in motor function and pin and light touch at 6 months compared to those treated after 8 hours and those treated with naloxone or placebo (199). Criticism of this study include the use of only the right side for motor scores, the lack of anatomic level injury limit or required motor deficit (i.e., injury below T12 and normal motor examination were included), and lack of functional outcome measures. NASCIS III, which compared methylprednisolone to a free radical scavenger, trilazidamide, for 2–4 or 48-hour infusions, showed that a 48-hour infusion initiated within 3 to 8 hours improved motor scores at 6 weeks and 6 months (200). Multiple trials have failed to show the benefit seen in the NASCIS trials (201), and increased complications such as a higher rate of respiratory complications including pneumonia, gastrointestinal hemorrhage, sepsis, and longer hospital stays have been reported (202–204). The NASCIS III trial showed a higher rate of sepsis and pneumonia in the patients treated for 48 hours. For this reason, the American Association of Neurosurgical Surgeons CNS guidelines offer it as an option, but suggest that harmful side effects may be more consistent with the data than actual clinical benefit. Medical evidence also does not support the use of GM-1 ganglioside in acute spinal cord injury (201).

Pulmonary Support
The most common cause of death in patients with spinal cord injury is due to pneumonia, pulmonary emboli, and sepsis. In patients with tetraplegia, pneumonia and other respiratory complications occur in 40% to 70% of patients (206,207). Aggressive pulmonary toilet is essential. Although diaphragmatic innervation arises from the cervical levels of 3 through 5, an effective cough and deep inspiration requires intercostal musculature and thoracic innervation to split the chest wall while the diaphragm descends. Patients with high-level cervical injuries (C1–5) may fatigue over the first few hours to days. Also, patients such as smokers with lower cervical injuries with increased pulmonary secretions or those who have aspirated fluid such as blood, water, or stomach contents may have difficulty clearing their airway and should be monitored closely for failing pulmonary reserve. Early measurements indicating the need for elective endotracheal intubation include the use of vital capacity (<20 mL/kg) and negative inspiratory forces (less negative than –20 cm H2O). Hypoxia and hypercapnia are late signs of respiratory failure, and intubation should not await these findings.

Associated Vascular Injury
Blunt cervical spinal trauma can result in vertebral artery injury and cause posterior circulation ischemia (208). Incidence varies from 0.05% to 1% and may depend on screening methods (209,210). Mortality ranges from 23% to 28%, whereas 48% to 58% of survivors have significant neurologic deficits (211). The most common mechanism of injury is MVC, followed by falls and pedestrian and motorcycle crashes. Patients at risk include those with cervical fractures with subluxation or with a fracture through the transverse foramen, especially those with displaced or complex midface or mandibular fracture, a basilar skull fracture involving the carotid canal or sphenoid sinus, near-hanging resulting in cerebral hypoxia, and cervical vertebral body fracture or distraction injury (209). Suspicion should be high if the patient develops a lateralizing neurologic deficit with normal initial CAT scan, or evidence of a recent ischemic stroke on cerebral imaging. Although CT angiography with a 16-channel detector shows high sensitivity for screening, the gold standard is four-vessel cerebral angiography (212,213).

A grading system of injury has been described by Biffl (214) (Table 83.2). Fifty-seven percent of grade I injuries heal spontaneously in 10 days independent of therapy (215); therefore, they can be treated with aspirin. Retrospective studies of grade II through IV injuries show no difference between antithrombotic agent or heparin therapy although heparinization increases hemorrhage risk (216,217).

TABLE 83.2

<table>
<thead>
<tr>
<th>Grading Scale for Blunt Cervical Vascular Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Grade I:</strong> Luminal irregularity or dissection &lt;25% luminal narrowing</td>
</tr>
<tr>
<td><strong>Grade II:</strong> Dissection or intramural hematoma with ≥25% luminal narrowing, intraluminal thrombus, or raised intimal flap</td>
</tr>
<tr>
<td><strong>Grade III:</strong> Pseudoaneurysm</td>
</tr>
<tr>
<td><strong>Grade IV:</strong> Occlusion</td>
</tr>
<tr>
<td><strong>Grade V:</strong> Transection with free extravasation</td>
</tr>
</tbody>
</table>

Nutritional Support and Metabolic Changes

As described above, traumatic injury is associated with a hypermetabolic, catabolic state with nitrogen loss. In spinal cord injury victims, indirect calorimetry will be more accurate than the Harris-Benedict equation to determine metabolic needs (157,226). Although metabolic needs may be increased, the resting energy expenditure may be lower than expected.

Autonomic Dysreflexia

Autonomic dysreflexia (AD) is a life-threatening hypertensive emergency that typically occurs in patients with motor-complete SCI above the T6 neurologic level (227,228). Autonomic dysreflexia is typically seen in the rehabilitative phase of SCI; it has been recognized as early as 4 days after injury (229). Noxious stimuli including fecal impaction, bladder distention, or pain to the lower extremities increases sympathetic outflow below the injury level. Resultant vasodilatation of the splanchic bed forces blood into the systemic circulation and increases blood pressure. Reflex parasympathetic outflow rostral to the injury allows flushing of the skin above the level of the lesion and bradycardia. Recognition of this entity and detection and removal of the inciting noxious stimulus is primary. Blood pressure treatment traditionally included ganglionic blockers, although intravenous antihypertensives such as nicardipine or nitroprusside can be effective.

Prognosis

To better standardize the language used to describe SCI, the American Spinal Injury Association developed the ASIA Spinal Cord Injury Classification (Table 83.3) (230), which has shown good interrater reliability, making the classification useful for studies and comparison of outcome (231). Incomplete injury has a better prognosis than those that are complete. The ASIA Impairment Scale severity on presentation of injury is one of the strongest predictors for outcome (232,233). Those in group A are unlikely to have significant recovery (233) whereas those in groups C and D recover better than those in B. Magnetic resonance imaging shows that complete spinal cord injury was associated with more substantial maximum canal compromise, spinal cord compression, length of lesion, hemorrhage, and cord edema. Substantial canal compromise, intramedullary hemorrhage, and cord edema at time of presentation were predictive of a poorer prognosis (234).

Two clinical entities described in SCI are pertinent to prognosis: the first, spinal shock, is a transient loss of spinal cord function and parasympathetic outflow below the injury level. Resultant vasoconstriction of the splanchnic bed forces blood into the systemic circulation and increases blood pressure. Reflex parasympathetic outflow rostral to the injury allows flushing of the skin above the level of the lesion and bradycardia. Recognition of this entity and detection and removal of the inciting noxious stimulus is primary. Blood pressure treatment traditionally included ganglionic blockers, although intravenous antihypertensives such as nicardipine or nitroprusside can be effective.

Table 83.3

<table>
<thead>
<tr>
<th>ASIA Impairment Scale</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A: Complete: No motor or sensory function is preserved below the sacral segments S4-S5.</td>
<td></td>
</tr>
<tr>
<td>B: Incomplete: Sensory but not motor function is preserved below the neurologic level and includes the sacral segments S4-S5.</td>
<td></td>
</tr>
<tr>
<td>C: Incomplete: Motor function is preserved below the neurologic level and includes the sacral segments S4-S5.</td>
<td></td>
</tr>
<tr>
<td>D: Incomplete: Motor function is preserved below the neurologic level and includes the sacral segments S4-S5.</td>
<td></td>
</tr>
<tr>
<td>E: Normal: Motor and sensory function are normal.</td>
<td></td>
</tr>
</tbody>
</table>

The second is the central cord syndrome (CCS) in which the motor deficit in the upper extremities is disproportionately worse than that in the lower extremities, with bowel and bladder dysfunction and variable sensory loss below the level of injury (235,236). Typically, central cord syndrome results from hyperextension injury without a fracture in older patients as a result of a stenotic spondylotic cervical canal (238). Motor recovery is improved when there is a higher motor score at the time of injury (236). The natural history of central cord syndrome is good neurologic recovery, although some patients have persistent neurologic deficits. Conservative nonsurgical treatment is the usual course. Another group predisposed to central cord syndrome includes a younger population with acute central cervical disc herniation or with spinal instability that may require surgical decompression or stabilization.

### SUMMARY

Primary prevention and avoidance of neurologic injury would be ideal. Once neurologic injury ensues, a key principle is that the primary injury helps determine prognosis; however, secondary injury will also play a significant role in the overall functional outcome. Preventable significant secondary neurologic injury can occur in the ICU. The role of the intensivist is to identify these mechanisms of injury and to optimize management. Priority concerns are adequate cerebral perfusion pressure, maintenance of adequate blood pressure and eu- volemia, good oxygenation, fever control, adequate nutrition, and avoiding complications of critical care.

### References

66. Huseby JS, Pavlin EG, Butler J. Effect of positive end-expiratory pressure
hyperventilation in patients with severe head injury: a prospective multicenter
64. Diringer MN, Videen TO, Yundt K, et al. Regional cerebrovascular and
metabolic effects of hyperventilation after severe traumatic brain injury. J
63. Coles JP, Minhas PS, Fryer TD, et al. Effect of hyperventilation on cere-
bral blood flow in traumatic head injury: clinical relevance and monitoring
62. Diringer MN, Videen TO, Yundt K, et al. Regional cerebrovascular and
metabolic effects of hyperventilation after severe traumatic brain injury. J
61. Coles JP, Minhas PS, Fryer TD, et al. Effect of hyperventilation on cere-
bral blood flow in traumatic head injury: clinical relevance and monitoring
terized tomography after head trauma: a systematic review. J Trauma.
58. Davis DP, Fakhry SM, Wang HE, et al. Paramedic rapid sequence intuba-
2006;34:1134–1141.
57. SinghG. Significance of preclinical emergency treatment for the prog-
osis of patients with severe craniocerebral trauma [in German]. Acta Neurochir.
56. Gabriel EJ, Ghajar J, Jacoda A, et al. Guidelines for Prehospital Manage-
55. Bernard SA. Parametric imputation of patients with severe head injury: a
54. Singbartl G. Significance of preclinical emergency treatment for the prog-
osis of patients with severe craniocerebral trauma [in German]. Acta Neurochir.
with neurological outcome and lesion progression in traumatic subarachnoid
terized tomography after blunt head trauma: a systematic review. J Trauma.
48. Schuster R, Waxman K. Is repeated head computed tomography necessary
47. Stupar J, Schraufnagel DE, Patel KR. Sphincters in pulmonary veins.
46. Givner A, Guerney J, O’Connor D, et al. Reimaging in pediatric neuro-
45. Theodore J, Robin ED. Speculation on neurogenic pulmonary edema. Clin
44. Hunt JM, Davis K, Johnson DJ, et al. Cost and complications during in-
Trauma. 1992;33:582–585.
43. Geerts A, Guzman J, O’Connell D, et al. Reimaging in pediatric neuro-
42. Piek J, Chestnut RM, Marshall LF, et al. Extracranial complications of
41. Huseby JS, Pavlin EG, Butler J. Effect of positive end-expiratory pressure
40. Schraufnagel DE, Patel KR. Sphincters in pulmonary veins. Int J Legal
39. Bein R, Gray CJ, Shaib A. Effects of hypothermia on infant vol-
480.
37. Knudson FJ, Jensen PJ, Poteroff PL. Neurogenic pulmonary edema: treat-
35. Marie FW, Patton HD. Neural structures involved in the genesis of preoptic
34. Blessing WW, West MJ, Chalmers J. Hypertension, bradycardia and
hypothermia to improve the neurologic outcome after cardiac arrest. J
33. Huseby JS, Pavlin EG, Butler J. Effect of positive end-expiratory pressure
32. Lao MC, Huseby JS, Klotz W, et al. Mechanisms by which positive end-
31. Huseby JS, Pavlin EG, Butler J. Effect of positive end-expiratory pressure
30. Singh G. Significance of preclinical emergency treatment for the prog-
osis of patients with severe craniocerebral trauma [in German]. Acta Neurochir.
29. Schraufnagel DE, Patel KR. Sphincters in pulmonary veins. Int J Legal
28. Bein R, Gray CJ, Shaib A. Effects of hypothermia on infant vol-
25. Bernard SA. Parametric imputation of patients with severe head injury: a
2006;34:1134–1141.
23. Singh G. Significance of preclinical emergency treatment for the prog-
osis of patients with severe craniocerebral trauma [in German]. Acta Neurochir.
22. Bein R, Gray CJ, Shaib A. Effects of hypothermia on infant vol-
20. Bein R, Gray CJ, Shaib A. Effects of hypothermia on infant vol-


CHAPTER 84  CNS VASCULAR DISEASE
DAVID A. DECKER • BRIAN L. HOH • MICHAEL F. WATERS

IMMEDIATE CONCERNS

Major Problems
The first concern is to establish the diagnosis of stroke and determine if the patient is a candidate for thrombolytic therapy.

Stress Points
1. Strokes may be ischemic, resulting from the occlusion of small or large arteries, or hemorrhagic, resulting from the rupture of a conducting artery or an intraparenchymal artery.

2. An abrupt focal lateralizing neurologic deficit attributable to a cerebrovascular distribution is the hallmark of ischemic stroke.

3. A depressed level of consciousness is rarely the presenting symptom of ischemic stroke and much more commonly occurs in the setting of a hemorrhagic event.

4. Patients with acute ischemic stroke may be candidates for thrombolytic therapy, but the therapeutic window is extremely narrow, so timely diagnosis and evaluation is of the utmost importance.

Essential Diagnostic Tests and Procedures
1. A computed tomography (CT) scan of the brain is critical for the initial evaluation and management of the stroke patient. Additionally, when available, CT angiography and perfusion studies may aid in diagnosis and management.

2. Magnetic resonance imaging (MRI) is more sensitive than CT, but it is usually less available urgently, and patients must remain still for a much longer period of time.

3. Vascular ultrasound allows rapid bedside assessment of abnormal flow within the major intracranial and extracranial arteries and can provide valuable immediate information about the vascular physiology to supplement the anatomic information provided by the CT scan.

4. A transthoracic or transesophageal echocardiogram may identify potential cardiac sources of cerebral emboli.

5. An electrocardiogram (ECG) followed by continuous cardiac telemetry monitoring is often necessary to identify arrhythmias associated with stroke.

6. A lumbar puncture may be necessary to rule out subarachnoid hemorrhage (SAH) in patients in whom the diagnosis is strongly suspected but CT is unrevealing.

Initial Therapy
1. Thrombolytics are the mainstay of treatment of acute ischemic stroke in eligible patients.

2. Careful attention to blood pressure may reduce complications such as hemorrhage.

3. Supportive care, with special attention paid to prevention of aspiration pneumonia and deep vein thromboses, is essential to reduce mortality associated with stroke.

4. Rapid initiations of secondary preventive therapies are effective in reducing risk for recurrent stroke.

5. Certain patients, such as those with large ischemic or hemorrhagic strokes, will require monitoring of intracranial pressure and, potentially, decompressive surgery.

DIFFERENTIAL DIAGNOSIS

Most patients who suddenly develop a lateralized focal neurologic problem have, in fact, had a stroke. It is by far the most common acute, focal, nontraumatic brain disease. When the presentation differs from this definition, further investigation and supportive evidence should be sought before establishing the diagnosis. As shown in Table 84.1, there are many stroke symptoms that may occur alone, unaccompanied by other
often occur. These symptoms may occur with or without the
ena, such as bright lines, and blurriness or loss of vision are
toms that may be misinterpreted as stroke. Visual phenom-
deficits.
ritary electrolyte
etion, alcohol, or metabolic abnormalities. Extreme electrolyte
serum glucose derangements can produce temporary focal

demonstration of a hemorrhage on CT are usually all that is
primary complaint in these cases. Findings consistent with infection or
demanda of a hemorrhage on CT are usually all that is
required to differentiate these disorders from ischemic stroke.
The next largest group of mistaken diagnoses occurs in patients
suffering confusion and neurologic deficits from drug intoxica-
tion, alcohol, or metabolic abnormalities. Extreme electrolyte
or serum glucose derangements can produce temporary focal
deficits.

Table 84.1 lists the diseases most commonly mistaken for
stroke. Epilepsy mimics stroke more often than any other con-
dition. In one study of 821 consecutive patients admitted to
a stroke unit, only 13% had a disease other than stroke, but
almost 40% of these misdiagnosed patients had seizures (1).
Focal onset seizures may leave a portion of the ictal brain dys-
functional for a prolonged period (hours or more), and the
deficits may be indistinguishable from those of ischemic stroke.

The only clues will be a history of seizures or absence of
diagnostic evidence of ischemia.

Intracranial hemorrhage, encephalitis, or other structural
brain lesions, such as tumors, may produce focal deficits iden-
tical to ischemic stroke. However, headache and altered or de-
pressed level of consciousness are more likely to be the primary
complaint in these cases. Findings consistent with infection or
demonstration of a hemorrhage on CT are usually all that is
required to differentiate these disorders from ischemic stroke.

The next largest group of mistaken diagnoses occurs in patients
suffering confusion and neurologic deficits from drug intoxica-
tion, alcohol, or metabolic abnormalities. Extreme electrolyte
or serum glucose derangements can produce temporary focal
deficits.

Migraine may produce several transient neurologic symp-
toms that may be misinterpreted as stroke. Visual phenom-
ena, such as bright lines, and blurriness or loss of vision are
commonly described. Sensory disturbances, and particularly
well-demarcated regions on the upper extremity and personally
often occur. These symptoms may occur with or without the
associated head pain but do not respect laterality or vascular
territories as ischemic strokes do. People who suffer migraine,
however, are at increased risk for ischemic stroke, so a thor-
ough evaluation is warranted before dismissing the symptoms,
particularly if it is the first occurrence. Motor deficits only very
rarely result from migraine, so they should be attributed to
stroke until proven otherwise.

Occasionally, peripheral nerve lesions, such as Bell palsy,
may appear suddenly and mimic an ischemic stroke. Care-
ful differentiation between upper and lower motor signs will
most often clarify the diagnosis, but incomplete presentations
may be confusing. In general, dense paralysis in the absence
of other neurologic signs or complaints is more likely the re-
sult of a peripheral lesion. Although uncommon, stroke may
present with bizarre or otherwise unbelievable symptoms, so
the diagnosis of a psychogenic disorder should remain one of
exclusion.

Establishing a correct diagnosis of these stroke mimics usu-
ally depends heavily on the patient’s history, and the physician
must specifically probe for characteristics of these diseases. A
thorough history and physical examination, combined with ap-
propriate laboratory testing and brain imaging such as MRI or
CT scan, can usually exclude most conditions that mimic a
stroke.

### ISCHEMIC STROKE

#### Pathogenesis

Ischemic stroke occurs when the supply of blood to brain tis-
sue is acutely interrupted. Normal cerebral blood flow in gray
matter is about 80 mL/100 g tissue per minute, whereas white
matter is about 20 mL/100 g tissue per minute. A global av-
erage in cortical mantle (assuming a 50:50 mix of gray and
white matter) is about 80 mL/100 g tissue per minute, whereas
white matter is acutely interrupted. Normal cerebral blood flow in gray
matter is about 80 mL/100 g tissue per minute, whereas white
matter is about 20 mL/100 g tissue per minute. A global av-
erage in cortical mantle (assuming a 50:50 mix of gray and
white matter) is about 50 mL/100 g tissue per minute. Mod-
est perturbations of the amount of cerebral blood flow can be
accommodated by the autoregulatory capacity of the cerebral
vasculature. When systemic blood pressure drops, resistance
vessels in the brain dilate to increase flow. Once these vessels
are maximally dilated, further drops in systemic pressure will
reduce cerebral blood flow. If the average cerebral blood flow
drops below 35 mL/100 g tissue per minute, protein synthesis
stops; below 20 mL/100 g tissue per minute, synaptic failure oc-
curs and neurons cease to function. As this threshold is crossed,
patients become suddenly symptomatic. When cerebral blood
flow drops below 10 mL/100 g tissue per minute, metabolic
failure and irreversible cell death occur.

Disruption of blood flow to the brain does not affect all tis-
sues within the vascular distribution equally. Most often, there
is a region with markedly reduced flow that quickly undergoes
infarction; surrounding that area are regions with diminished
flow that will survive longer, but not indefinitely. This poten-
tially salvageable area is referred to as the penumbra. The pur-
pose of acute stroke therapies is to salvage the penumbra.

In the general adult population, the causes of disruptions
of arterial flow can be separated into three major categories
based on cause: (i) large vessel atherothrombotic disease, (ii)
small vessel (lacunar) infarction, and (iii) cardioembolic em-
bolism. This categorization will focus the diagnostic evalua-
tion and therapy and has prognostic implications. Determin-
ing the cause will also guide the clinician in administering the

<p>| TABLE 84.1 |</p>
<table>
<thead>
<tr>
<th>SYMPTOMS Seldom Resulting FROM CEREBROVASCULAR DISEASE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vertigo alone</td>
</tr>
<tr>
<td>Dysarthria alone</td>
</tr>
<tr>
<td>Dysphagia alone</td>
</tr>
<tr>
<td>Diplopia alone</td>
</tr>
<tr>
<td>Headache</td>
</tr>
<tr>
<td>Tremor</td>
</tr>
<tr>
<td>Tonic-clonic motor activity</td>
</tr>
</tbody>
</table>

<p>| TABLE 84.2 |</p>
<table>
<thead>
<tr>
<th>CONDITIONS MOST FREQUENTLY MISTAKEN FOR STROKE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seizures</td>
</tr>
<tr>
<td>Metabolic encephalopathy</td>
</tr>
<tr>
<td>Cerebral tumor</td>
</tr>
<tr>
<td>Subdural hematoma</td>
</tr>
<tr>
<td>Cerebral abscess</td>
</tr>
<tr>
<td>Vertigo, Meniere disease</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
appropriate level of care based on the possibility of progress-
and anticipated complications. For example, subtle speech
changes and right hand weakness could be the presenting symp-
toms of any of the three stroke subtypes outlined above. If a
lacunar infarction is the cause, symptoms will most likely be
maximal at onset and rarely will the patient experience compli-
cations compromising respiration or circulation. The volume
of brain tissue involved is by definition small, and the risk for
progression or recurrence in the acute period is quite small. A
cardiogenic embolus would also be expected to cause symp-
toms maximal at onset, but the infarct volume could be large
enough to cause mass effect and, depending on the underlying
cardiac pathology, the risk for complications could be substan-
tial.

Large Vessel Atherothrombosis

Large vessel atherothrombosis encompasses approximately
15% of all strokes, of these, two thirds are of extracranial
internal carotid artery (ICA) origin, and one third are due to
intracranial atherosomatic disease (2). Atherosomatic disease
of large vessels is a slowly degenerative process, but as the disease
progresses, the chance for lesion instability increases. Vascu-
lar plaques may fragment, exposing an ulcerated surface that
is highly thrombogenic and leading to local occlusion or cre-
ation of emboli material. Thus, in large vessel disease, throm-
bus or artery-to-artery embolism are often parts of the same
underlying pathologic process. Atherosomatic lesions tend to
occur at bifurcations or sharp turns in the vessel, both of which
are associated with increased blood flow turbulence. The pro-
totypic example of this is carotid stenosis at the origin of the
ICA. Other common extracranial sites include the origin of
the vertebral artery and the other great vessels. Intracranially,
common sites include the distal vertebral artery, the midbasilar
artery, the phthion of the ICA, and the proximal middle cerebral
artery (MCA).

Lacunar Strokes

Lacunar strokes represent approximately one quarter of all
such events. They are caused by occlusion of a single perfo-
rating artery, such as those that supply deep brain structures
like the thalamus, pons, or basal ganglia (Fig. 84-1). The result
is a small infarction—by most definitions, less than 1 cm3—
that undergoes liquefaction necrosis with time, leaving a tiny
fluid-filled space for which they are named. The primary risk
factor for lacunar infarction is hypertension, which results in
lipohyalinosis of the vessel wall with progressive concentric
stenosis and eventual thrombosis (3).

Cardiogenic Embolism

Approximately 60% of all ischemic strokes are caused by cere-
bral embolism, of which only one third have a definitively
known clinical source (4). Cerebral emboli may be composed of
atherosomatic plaque material, clotted blood, or, in rare cases,
air or fat. Once free in the arterial circulation, the emboli will
tend to follow the straightest path formed by the most blood.
Therefore, most emboli will affect distal branches of the MCA,
although other locations are possible; the larger the embolus,
the more proximal it will lodge.

Cerebral emboli may originate from atherosomatic disease
of more proximal large vessels, such as the aortic arch, as out-
lined above, or the heart (5). Atrial fibrillation is the most com-
mon cardiac cause, but others include valvular heart disease,
intracardiac thrombus, atrial myxoma, dilated cardiomyopa-
thy, patent foramen ovale (PFO), especially when accompanied
by an atrial septal defect, and endocarditis. Air emboli are usu-
ally iatrogenic and result when a large amount of air enters
the venous circulation (e.g., through a central venous catheter)
and bypasses the lungs through a PFO, thereby entering the
arterial circulation. Fat emboli are generally the result of long-
bone fractures in severe trauma. It is important to seek out
the definitive source whenever possible, as it may have a pro-
found impact on the management of secondary stroke preven-
tion. For example, although most sources of cardiogenic emboli
are treated with oral anticoagulation, several conditions may
contraindicate it, such as bacterial endocarditis (6) or atrial
myxoma (7).

Arterial Dissections

Although arterial dissections may occur at any age, they are
probably the most common cause of stroke in young patients
(younger than 50 years old) who are unlikely to have typical
risk factors. Arterial dissections may arise spontaneously or
following a traumatic head or neck injury (8). These lesions
typically rupture the petrous portion of the ICA or at the cervi-
ocal 1–2 level in the vertebral artery. Thrombus may form at
the site of intimal tear, extending into the media, with subsequent
artery-to-artery embolism. If a large intimal flap or intramural
hematoma forms, occlusion of the affected vessel may occur.
Dissection may also lead to subarachnoid hemorrhage when a
pseudoaneurysm forms after the artery passes intradurally
(9).

Clinical Evaluation

History

The history, when available, is the key instrument in diagnosing
neurologic disease. If the patient is unable to provide a reliable
history, which is often the case in acute brain dysfunction, then
historic details should be sought from witnesses, family, EMS
records, or whatever sources are available; no other diagnostic
tool will so quickly narrow the differential diagnosis.

The key historic element in ischemic cerebrovascular disease
is sudden onset of symptoms, which are typically maximal at
onset. Under certain circumstances, the symptoms may follow
a stuttering or stepwise progression, but in each instance there
is a sudden change. Contrast this with the waxing and waning
character of delirium or the symptoms that may develop over
days to weeks from a brain tumor. There is an unfortunate
tendency to dismiss symptoms of short duration, but this is a
serious mistake, as the duration of the symptoms speaks little
to the underlying pathogenesis. For example, a patient with
occult atrial fibrillation may suffer transient neurologic deficits
from an embolus that happens to spontaneously lyse before
infarction has completed. Without addressing the underlying
cause, the patient is unlikely to continue to be so fortunate,
and an opportunity to prevent a devastating neurologic injury
will have been lost.

If the ictal event is consistent with a stroke, the remainder
of the initial history should be focused on determining if the
patient is a candidate for thrombolytic therapy and identify-
ning possible risk factors. Whereas other details not elucidated
in the initial history can be revisited at a later time, it is of

1262 Section VIII: The Surgical Patient
FIGURE 84.1. Intracerebral hemorrhage of the left basal ganglia. Hyperdense appearance of blood on the CT scan easily defines the extent of hemorrhage.

The utmost importance to obtain the exact time of symptom onset, as current acute stroke treatment protocols depend on this for inclusion. If the onset was not witnessed, then the time when last known to be normal is used. For example, if the patient awakes with symptoms, then the time when the patient retired is used (assuming he or she was asymptomatic then).
of time to identify patients with a salvageable penumbra, but these techniques have yet to mature. Aside from time of onset, factors that may place the patient at increased risk for bleeding must be sought. Tables 84.3 through 84.5 list common inclusion and exclusion criteria for intravenous (IV)-tPA (tissue plasminogen activator), which are based on those used in the pivotal trials. Most institutions have their own protocols, which may vary somewhat from those used in the trials.

### TABLE 84.3

**INCLUSION CRITERIA FOR tPA USE**

1. Symptoms consistent with acute ischemic stroke, with a clearly defined onset of less than three hours before rt-PA will be given (if the onset was not witnessed, the ictus is measured from the time the patient was last seen to be at baseline);
2. A significant neurological deficit is expected to result in long-term disability
3. A non-contrast CT with no evidence of hemorrhage or well-established infarction.

### TABLE 84.4

**ABSOLUTE CONTRAINDICATIONS TO tPA USE**

1. Mild or rapidly improving deficits
2. Hemorrhage on CT, well-established acute infarct on CT, or any other CT diagnosis that contraindicates treatment, including abscess or tumor (excluding small meningiomas)
3. A known CNS vascular malformation or tumor
4. Bacterial endocarditis
5. Hemorrhage on CT; CNS, central nervous system.
6. Seizure at onset of stroke
7. Known bleeding diathesis (includes renal and hepatic insufficiency)
8. Peritoneal dialysis or hemodialysis
9. Arterial puncture at a noncompressible site within past 14 days
10. Major surgery within the past 14 days
11. History of intracranial hemorrhage, or symptoms suggestive of subarachnoid hemorrhage
12. Ischemic stroke within 3 months
13. Significant trauma within the past 3 months (including CPR with chest compressions within the past 10 days)
14. History of intracranial hemorrhage, or symptoms suggestive of subarachnoid hemorrhage
15. Major surgery within the past 14 days
16. Minor surgery within past 19 days, including liver and kidney biopsy, thoracentesis, and lumbar puncture
17. Gastrointestinal, urologic, or respiratory hemorrhage within past 21 days
18. Known bleeding diathesis (includes renal and hepatic insufficiency)
19. Peritoneal dialysis or hemodialysis
20. Pregnancy (and 10 days postpartum)

### TABLE 84.5

**RELATIVE CONTRAINDICATIONS TO tPA USE**

1. Symptoms consistent with acute ischemic stroke, with a clearly defined onset of less than three hours before rt-PA will be given (if the onset was not witnessed, the ictus is measured from the time the patient was last seen to be at baseline);
2. A significant neurological deficit is expected to result in long-term disability
3. A non-contrast CT with no evidence of hemorrhage or well-established infarction.

Once a decision regarding acute treatment has been made, attention can be directed to identifying conditions that may have caused the patient's stroke. Cerebrovascular and cardiovascular diseases share many of the same risk factors, including hypertension, hyperlipidemia, diabetes mellitus, cigarette smoking, and obesity. Other risk factors that may be important include obstructive sleep apnea, migraine headaches with an aura, and drug abuse. Family history may provide insight into heritable causes of stroke such as cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) or Fabry disease.
Neurologic Examination

The neurologic examination will allow the clinician to quickly determine which brain areas are dysfunctional and further narrow the differential diagnosis. The neurologic deficits caused by ischemic cerebrovascular disease are expected to be lateralizing and confined to a vascular distribution. For example, the triad of language disturbance and right face and arm motor deficits is typical for occlusion of the left MCA. However, sudden-onset motor and sensory deficit of both arms is not lateralizing, nor readily explained by a cerebrovascular occlusion, and thus is more likely due to spinal cord pathology.

The first step in localization of vascular lesions is determining, based on the signs and symptoms, whether they arise from the anterior circulation (carotid artery and its main branches) or the posterior circulation (vertebral, basilar, and posterior cerebral arteries). This finding will guide the remainder of the diagnostic evaluation, therapy, and prognosis. Ideally, these two separate circulations would be robustly connected such that a failure in one could be compensated by the other, but this is rarely the case.

The two symptoms that most accurately reflect carotid circulatory disease are aphasia and monocular blindness. Aphasia is a deficit in either the expression or comprehension of language and may involve both in the acute period. Aphasia must be distinguished from dysarthria, which is the inability to correctly produce words due to motor impairment of facial, lingual, or pharyngeal muscles; dysarthria may result from either anterior or posterior circulation infarcts. The areas responsible for language reside in the dominant (nearly always left) hemispheric cortex, within the territory of the MCA; a stroke causing aphasia, therefore, involves this circulation. Similarly, the blood supply of the eye arises largely from the ophthalmic artery, a direct branch from the carotid artery, and monocular ischemia therefore implicates the carotid circulation. The prototypic example of this process is amaurosis fugax, or transient monocular blindness in which vision is lost in one eye for minutes. This must be contrasted with a visual field defect, which affects one field of both eyes, as this is more likely the result of posterior circulation ischemia. Pain is rarely a significant complaint, but when present, especially if following the course of a major blood vessel, arterial dissection should be considered. Involvement of the carotid artery may cause a Horner syndrome.

Because of the density of discrete populations of neurons supplied by the posterior circulation, the clinical syndromes that result from strokes in this area are usually more complex than those in the cerebral hemispheres. The medulla, pons, midbrain, cerebellum, parts of the thalamus, and the visual cortices are the major structures involved. Strokes involving the brainstem often manifest with cranial nerve dysfunction (dysarthria, dysphagia, diplopia). Crossed signs, with motor or sensory deficits affecting one side of the face and the opposite side of the body, may occur as major decussations in these pathways and occur in the pons and medulla. The unique vascular anatomy of the basilar artery, with a single midline vessel supplying both sides of the pons and the posterior cerebral arteries, may lead to bilateral neurologic deficits.

Lacunar infarctions also have a set of clinical features that may be used to differentiate them from other stroke subtypes. Classic lacunar syndromes include pure motor hemiparesis (caused by infarction in the internal capsule or basis pontis), pure hemisensory symptoms (caused by infarction in the ventral posterolateral [VPL] thalamic nucleus), dysarthria-clumsy hand syndrome (with pontine or internal capsule infarcts), and ataxia-hemiparesis (caused by infarction in the pons). Lacunar syndromes may be differentiated from other stroke syndromes based on their characteristic features, which include a sudden onset, rapid resolution, and a high rate of recurrence.

Vascular System Examination

Physical examination of the vascular system itself is usually surprisingly unrewarding. Atherosclerosis may present few outward signs. Although carotid bruits were classically emphasized, modern ultrasound techniques have proven them to be of low sensitivity and specificity for predicting vascular disease. No characteristic feature, including the volume, pitch, or duration of the bruit, reliably indicates the degree or the nature of constriction of the vascular lumen. Many bruits reflect benign conditions. The clinical significance of carotid bruits is minimized because they are audible in many asymptomatic persons without atherosclerosis who never suffer from cerebrovascular disease, but may be absent in severely diseased vessels. Therefore, even if a carotid bruit is detected, it may be difficult to decide whether it is relevant to the patient’s symptoms, and it should not be given undue emphasis in the overall evaluation. Clinical decisions should be based on definitive assessment of blood vessels using ultrasound or angiography.

Examination of the heart should focus on detecting thrombogenic diseases, including myocardial infarction, congestive heart failure, arrhythmias, prosthetic valves, and bacterial endocarditis. Heart disease is a key risk factor for stroke and may complicate the acute period. Patients with stroke may also present concurrently with a myocardial infarction (MI) and, without careful examination, the less dramatic of the two may go undetected. Elevation of serum troponin levels is a very sensitive and specific indicator of an MI. Isolated creatine kinase-MB elevations should be interpreted with caution, however, as the MB fraction is expressed in brain tissue, and small elevations are not uncommon in stroke (10).

Laboratory Studies

Laboratory studies will also help in the differential diagnosis and may reveal relevant comorbid conditions. Some studies need to be obtained immediately to determine a patient’s candidacy for thrombolytic therapy, including an electrolyte battery, glucose, platelet count, cardiac enzymes, beta-HCG (human chorionic gonadotropin), and coagulation parameters. Severe electrolyte (specifically hypotension or hypernatremia) or glucose disturbances can cause neurologic dysfunction that may mimic stroke. Cardiac enzymes will determine if cardiac ischemia is part of the current presentation, and a beta-HCG will reveal occult pregnancy, both of which may be contraindications to systemic thrombolysis. Coagulation parameters and a platelet count will identify patients who may be at greater risk for bleeding.

Other laboratory studies will help determine the cause and identity risk factors but do not need to be obtained immediately. A fasting lipid profile should be obtained for potential vascular risk modification. In patients older than 50 years of age, an erythrocyte sedimentation rate and C-reactive protein are essential if giant cell arteritis is suspected. In patients for whom an unusual cause of stroke is suspected (young patients or minimal vascular risk factors), laboratory investigation of prothrombotic states could be considered. The interpretation of these tests is very complex and should be performed in consultation with a hematologist (11). Toxicology screening should be performed on hospital admission, with attention...
Direct imaging studies are used in the evaluation of acute strokes. A noncontrast CT scan of the brain is the standard initial examination. CT is very sensitive to the presence of hematomas, which is the primary reason for its use in the acute setting, and is the only radiologic test necessary to determine eligibility for IV-tPA. CT is not sensitive for detection of an acute cerebral infarction (Fig. 84.2), and the lack of abnormalities within the first 24 hours should be expected. The view of the posterior fossa is also quite limited, and any changes (with the exception of hemorrhage) seen within the brainstem or cerebellum should be confirmed with MRI. When available, CT angiography and perfusion studies can be obtained with very little additional time and provide valuable additional information about blood vessels and blood flow to individual large vessel territories. Pathologic changes on these studies are visible immediately, but they require iodinated contrast, which may be a limiting factor for some patients. The angiographic results from CTA are closest to the traditional standard gold exam: digital subtraction catheter angiography (CTA). When examining the carotid arteries, the results from CTA are often sufficient to differentiate a high-grade stenosis (60%–99%) that is treatable from complete occlusion that is not.

MRI. MRI is far more sensitive for acute infarction than CT. Diffusion MR sequences can detect ischemia within, perhaps, minutes of onset. MRI is of special value in brainstem and posterior circulation strokes, since the images it produces are not obscured by bony artifacts as with CT (Fig. 84.3). The combination of multiple MRI sequences allows for much more specific differentiation between ischemic brain and other structural abnormalities. In addition, MRI can display flow-related enhancement of the vasculature, resulting in a magnetic resonance angiogram (MRA). The resulting image can be manipulated in three dimensions, allowing for more accurate interpretation of small abnormalities. However, MRA tends to slightly overestimate the degree of stenosis, and thus is not usually sufficient to differentiate high-grade stenosis from occlusion (12). MRA uses gadolinium as a contrast agent instead of an iodinated material, making the study available to more patients. Perfusion studies, very similar to those performed with CT, may also be obtained if the right equipment is available. Disadvantages of MRI and MRA include reduced availability and substantially longer scanning times, which place some limitations on their use.

Digital Subtraction Catheter Angiography. Digital subtraction catheter angiography is an invasive imaging technique in which the artery of interest is selectively catheterized under fluoroscopy and dye injection enables a high-resolution view of the vessel that can be obtained in multiple planes. As the quality of noninvasive imaging techniques has improved and the associated morbidity has decreased to approximately 1% (11), catheter angiography is no longer used as a routine screening test. Indications now include precisely delineating critical vascular stenoses and examination of arterial dissections, arteriovenous malformations, and aneurysms.

Carotid Ultrasound. Carotid ultrasound provides a rapid noninvasive assessment of carotid artery disease, based on abnormalities of either flow (Doppler) or morphology (B-mode). As with any ultrasound technique, sensitivity is somewhat dependent on the operator. However, with experienced technicians and interpreters, duplex scanning provides a reproducible, accurate screening examination for carotid disease. However, this technique suffers from the same limitation as MRA in differentiating high-grade stenosis and occlusion. Lesions of the more distal internal carotid artery may also be difficult to visualize in some patients.

Transcranial Doppler. Transcranial Doppler (TCD) allows rapid bedside assessment of abnormal flow within the distal ICA and major intracranial arteries. It is primarily a functional study that provides information about blood flow velocity and vascular resistance rather than structural features. The 2-MHz ultrasonic signal can penetrate various bony "windows" in most patients, and its gated character allows identification of arteries by "depth" of the reflected signal. TCD can examine proximal portions of all major branches of the circle of Willis, but is insensitive for pathology beyond the A1, M1, or P1 segments. Newer applications include detection of microemboli and online monitoring of arterial flow during invasive procedures, such as carotid endarterectomy (Fig. 84.4). TCD may also be useful as an adjunctive therapy to ICA, as continuous insonation may enhance thrombolysis (13). Disadvantages include major dependency on operator skill and the prevalence of acoustically inadequate bony windows.

Transesophageal Echocardiography. Transesophageal echocardiography (TEE) is essential to evaluate cardiac function. Physicians must attend not only to a visualized thrombus, but also to other pathologic states associated with systemic embolization.
including left ventricular wall motion abnormalities, chamber dilatation, valvular disease, ejection fraction, and septal defects. TTE can be routinely performed and is a superior study for the detection of ventricular apex pathology, left ventricle thrombus, and views of prosthetic valves. Transesophageal echocardiography (TEE) provides much greater resolution and is more sensitive for pathology of the left atrial appendage, intra-atrial septum, atrial aspect of mitral-tricuspid valves, and the ascending aorta. TEE is an invasive procedure that requires sedation but can be performed safely on most patients [14].

Management

Acute Therapy

As with any acutely ill patient, attention should initially be focused on the evaluation of airway, breathing, and circulation. A secure airway should be established for patients with a depressed consciousness. Supplemental oxygen or mechanical ventilation should be used as needed to treat any degree of hypoxia. Circulation assessment includes evaluation of blood pressure and cardiac electrical activity with an ECG, as coexistent MI is not uncommon. Patients with acute stroke are often markedly hypertensive, and one should be cautious in

FIGURE 84.3. MRI demonstrates enhancing left lateral medullary infarction in a patient with Wallenberg syndrome.

FIGURE 84.4. Transcranial Doppler. A: The normal flow-velocity profile through the middle cerebral artery (velocity plotted over time during three cardiac cycles) is demonstrated. B: Elevated flow velocities as a result of the arterial spasm associated with subarachnoid hemorrhage is demonstrated. C: Two microemboli are detected through the middle cerebral artery as transient high-intensity signals.
aggressively treating elevated blood pressure before a more complete assessment of the patient has been completed (15). Blood pressure goals are determined by type of stroke, cause, and the presence of comorbid conditions, such as coronary artery disease; this will be discussed in the following sections. The immediate goal will be to determine if the patient is a candidate for thrombolytic therapy; thus attention should be focused on obtaining the relevant history and performing a neurologic examination. The care of patients who will ultimately not receive thrombolytic therapy will be discussed below (see Supportive Care). Crucial for therapy is the proper determiniation of the exact time of onset of the stroke. The patient must be witnessed to have had an abrupt change in neurologic status by a reliable observer; otherwise the time of onset, by default, must be the last time the patient was seen at his or her baseline level of neurologic function. All patients should be evaluated with the National Institutes of Health Stroke Scale (NIHSS), which can help to exclude a patient from potentially harmful therapy on the basis of the stroke being too small or too severe.

Thrombolytic Therapy. Currently, thrombolytic treatment with recombinant tissue plasminogen activator (tPA) in eligible stroke patients is the standard of care based on the results of four large trials: the National Institute of Neurological Disorders and Stroke (NINDS) recombinant t-PA study (16–18), the European Cooperative Acute Stroke Study (ECASS)-I (19), ECASS-II (20), and the ATLANTIS tPA (Alteplase) Acute Stroke Trial (parts A and B) (21,22). The collective results indicate that patients treated with tPA within 3 hours of onset were >30% more likely to have minimal or no disability at 3 months compared with placebo-treated patients (23). The average disability across all groups was, additionally, decreased in the treatment group. The benefit seen by the tPA-treated group existed regardless of patient age or stroke subtype.

To maximize the possible benefit and minimize the risk for hemorrhage, the NINDS study helped to establish strict inclusion criteria for the administration of thrombolytic therapy in acute stroke patients, which are outlined in Table 84.3. Absolute exclusion criteria have been established as well (Table 84.4). In addition, there is a long list of relative contraindications to thrombolytic therapy (Table 84.5), which often vary slightly in institutional protocols. These can be summarized as risks for bleeding from a noncompressible site, the presence of potential stroke mimics, or uncontrollable hypertension. The rationale for excluding patients with improving symptoms is to avoid giving a potentially harmful treatment to patients with epileptic postictal presentations or spontaneous recovery.

Tissue Plasminogen Activator (tPA). When the protocol is followed, the administration of tPA is safe relative to other commonly accepted treatments. Several studies have attempted to examine the risk, but perhaps the most clinically relevant is the number needed to harm. For every 100 patients treated with tPA who match the NINDS trials populations across all levels of final global disability, approximately 32 will receive benefit, and approximately 3 will be harmed (24). The risk–benefit ratio, thus, strongly favors treatment. Many of the hemorrhages that occur are asymptomatic, and of those that are symptomatic, not all symptoms persist to discharge. Most patients who experience intracerebral hemorrhage (ICH) after tPA therapy have severe baseline insults and were already destined for a poor outcome; thus the hemorrhage does little to alter the final outcome (25). One should keep in mind that, although excluding a patient from treatment will mitigate the risk for hemorrhage, it also denies the patient a significantly better chance of recovery.

The dose of tPA is 0.9 mg/kg, with a maximum dose of 90 mg. 10% is given as a bolus over 1 minute, and the remaining 90% is infused over 60 minutes. Following treatment, patients should be monitored in an ICU for more than 24 hours. While many post-tPA patients will not need ventilatory or pressure support, as do many other patients in the ICU, blood pressure monitoring and frequent neurologic exams are critical for a favorable outcome, as hypertension dramatically increases the risk for hemorrhage. Blood pressure must initially be monitored noninvasively as arterial puncture is contraindicated for 24 hours after administration of tPA. All other invasive procedures, such as placement of nasogastric tubes, urinary bladder catheters, central venous lines, intramuscular injections, and rectal temperature must be avoided for 24 hours as well. Also, any drug that impairs hemostasis such as heparin, or antiplatelet or nonsteroidal anti-inflammatory agents, are contraindicated during this 24-hour period.

Vital Signs. Vital signs should be checked every 15 minutes for the first 2 hours, then every 30 minutes for 6 hours, and then every hour for 16 hours. Blood pressure should be strictly controlled for 24 hours, keeping the systolic blood pressure less than 180 mm Hg and the diastolic blood pressure less than 105 mm Hg (Table 84.6). Labetalol is recommended for control of hypertension; 10 mg should be given intravenously over 1 to 2 minutes, and the dose repeated or doubled every 10 to 20 minutes, up to a total of 150 mg. If the blood pressure remains refractory despite these measures, consideration can be given to a continuous infusion of nifedipine or sodium nitroprusside. Neurologic evaluation should be performed every hour. Oxygenation should be checked by continuous pulse oximetry and oxygen provided to keep saturation >95%. The benefit of therapeutic hypothermia is yet to be confirmed, but euthermia is clearly associated with better outcome (26). Acetaminophen, 650 mg every 4 hours orally or rectally, should be given for any fever greater than 100°F. After administration of tPA, 90 mg; 10% is given as a bolus over 1 minute, and the remainder (80 mg) is given intravenously over 60 minutes. Following treatment, neurosurgery should be contacted for possible hematoma evacuation, and anticoagulation should be reversed according to the protocol outlined in Table 84.7.

Vital Signs. Vital signs should be checked every 15 minutes for the first 2 hours, then every 30 minutes for 6 hours, and then every hour for 16 hours. Blood pressure should be strictly controlled for 24 hours, keeping the systolic blood pressure less than 180 mm Hg and the diastolic blood pressure less than 105 mm Hg (Table 84.6). Labetalol is recommended for control of hypertension; 10 mg should be given intravenously over 1 to 2 minutes, and the dose repeated or doubled every 10 to 20 minutes, up to a total of 150 mg. If the blood pressure remains refractory despite these measures, consideration can be given to a continuous infusion of nifedipine or sodium nitroprusside. Neurologic evaluation should be performed every hour. Oxygenation should be checked by continuous pulse oximetry and oxygen provided to keep saturation >95%. The benefit of therapeutic hypothermia is yet to be confirmed, but euthermia is clearly associated with better outcome (26). Acetaminophen, 650 mg every 4 hours orally or rectally, should be given for any fever greater than 100°F. After administration of tPA, 90 mg; 10% is given as a bolus over 1 minute, and the remainder (80 mg) is given intravenously over 60 minutes. Following treatment, neurosurgery should be contacted for possible hematoma evacuation, and anticoagulation should be reversed according to the protocol outlined in Table 84.7.

STAT Head CT. A STAT head CT should be performed for a poor outcome; thus the hemorrhage does little to alter the final outcome (25). One should keep in mind that, although excluding a patient from treatment will mitigate the risk for hemorrhage, it also denies the patient a significantly better chance of recovery.
Chapter 84: CNS Vascular Disease

TABLE 84.6
BLOOD PRESSURE CONTROL AFTER tPA

1. Management of blood pressure during and after treatment with tPA or other acute reperfusion intervention:
   a. Monitor blood pressure every 15 minutes during treatment and then for another 2 hours, then every 30 minutes for 6 hours, and then every hour for 16 hours;
   b. For systolic 180 to 230 mm Hg or diastolic 105 to 120 mm Hg:
      i. Labetalol 10 mg IV over 1 to 2 minutes, may repeat every 10 to 20 minutes, maximum dose of 300 mg; or
      ii. Labetalol 10 mg IV followed by an infusion at 2 to 8 mg/min
   c. For systolic greater than 230 mm Hg or diastolic 121 to 140 mm Hg:
      i. Labetalol 10 mg IV over 1 to 2 minutes, may repeat every 10 to 20 minutes, maximum dose of 300 mg; or
      ii. Labetalol 10 mg IV followed by an infusion at 2 to 8 mg/min; or
      iii. Nicardipine infusion, 5 mg/h, titrate up to desired effect by increasing by 2.5 mg/h every 5 minutes to maximum of 15 mg/h
   iv. If blood pressure not controlled, consider sodium nitroprusside.


Theoretically, there are several potential benefits to treatment of stroke via an intra-arterial approach. First, angiographic confirmation of vessel occlusion can be obtained at the time of treatment. Second, high concentrations of thrombolytic agents can be given directly at the site of thrombosis, thereby minimizing systemic exposure. Third, the response to lysis can be monitored by direct visualization. Fourth, mechanical disruption of the clot (e.g., via balloon angioplasty, MERCI device) may accelerate thrombolysis (28).

According to the American Stroke Association guidelines (29), intra-arterial thrombolysis is an option for treatment of selected patients who have major stroke of <6 hours’ duration due to occlusion of the MCA and who are not otherwise candidates for intravenous tPA. The availability of intra-arterial thrombolysis should not preclude the administration of IV-tPA in otherwise eligible patients. Treatment requires immediate access to cerebral angiography and qualified interventionalists.

Supportive Care

Supportive care lacks the excitement and drama of acute therapy, but nonetheless is critical to patient outcomes. Since the 1970s, mortality from stroke has markedly diminished from a rate of 156 to 56/100,000 cases (30), with only 3% to 10% of stroke patients receiving thrombolytic therapy; this trend

TABLE 84.7
MANAGEMENT OF POST-tPA HEMORRHAGE

1. Blood should be sent STAT for CBC, PT, PTT, platelets, fibrinogen and D-dimer (this should be repeated every two hours until bleeding is controlled).
2. Give two units of fresh frozen plasma every six hours for 24 hours after the thrombolytic agent was given.
3. Give cryoprecipitate (20 units); if the fibrinogen level is <200 mg/dL at one hour, repeat the cryoprecipitate dose.
4. Give 4 units of platelets.
5. Give protamine sulfate (1 mg per 100 units of heparin given in the past 3 hours);
   a. A test dose of 10 mg slow IV push over 10 minutes should be given while observing for anaphylaxis;
   b. Then the remaining dose by slow IV push, up to a maximum dose of 50 mg.
6. Institute frequent neuro checks, as well as management of increased ICP, as needed.
7. Aminocaproic acid (Amicar) can be given as a last resort, in a dose of 5 g in 250 mL normal saline IV over 1 hour.

CBC, complete blood count; PT, prothrombin time; PTT, partial thromboplastin time; IV, intravenous; ICP, intracranial pressure.

Some even advocate the induction of therapeutic hypertension, these collateral vessels maximally dilate, and thus flow through alternative or collateral paths. Through autoregulation, the resulting change in pressure gradients will allow circulation to more areas of the brain. When blood flow is impaired, this practice. Therefore, lowering blood pressure potentially reduces blood flow to the potentially salvageable penumbra. Determining the exact pressure required for adequate blood flow is not readily accomplished, so one must err on the side of hypertension. For most stroke patients with acute hypertension, the general practice is to refrain from intervening until the pressure exceeds an arbitrary limit of 220 mm Hg systolic or 120 mm Hg diastolic (29). Patients with coronary artery disease or those with another comorbidity that may preclude tolerance of such pressures may require a careful decrement in blood pressure. In these cases, it is recommended that blood pressure be lowered slowly, using frequent smaller doses of drug rather than larger ones that may cause rapid changes; patients should be carefully observed for acute worsening as pressure is lowered. In patients having received thrombolysis, the tolerable limit is lower, as the risk for intracerebral hemorrhage increases with increasing blood pressure. In this case, pressures greater than 180 mm Hg systolic or greater than 105 mm Hg diastolic require treatment according to the protocol (Table 84.8).

**Hyperglycemia**

Hyperglycemia will be detected on admission in approximately one third of patients with stroke (38). Predictions for patients with persistent hyperglycemia (blood glucose level >200 mg/dL) during the first 24 hours after stroke are expansion of the volume of ischemic stroke and poor neurologic outcomes (39). Our practice is to aggressively control glucose—keeping it between 80 and 140 mg/dL—but care must be taken to avoid hypoglycemia, as the morbidity from that may abolish the benefit obtained from treating hyperglycemia.

Although most patients eventually improve substantially after a stroke, early clinical deterioration is not uncommon. Neu rologic causes of clinical deterioration include progressive or recurrent stroke, hemorrhagic transformation of the infarct, and local cerebral edema. The latter is the most common cause of deterioration, and may well cause fatal herniation in large MCA infarctions, especially in the young, women, and in patients with involvement of additional vascular territories (40). Brain swelling typically appears about 4 days after the stroke onset (41). Dramatic early swelling has been described, the...
term malignant MCA infarction is used to delineate a group of patients with large territorial infarcts that swell within 24 hours (42).

Ischemia-related edema is cytotoxic and unresponsive to treatments useful for vasogenic edema. Corticosteroids, in particular, do not appear helpful, and hyperglycemia associated with their use may worsen clinical outcome (29,43,44). Although no evidence exists that ultimate outcome is improved, certain treatments are often used to reduce intracranial pressure: mannitol (1 g/kg bolus, then 0.3 g/kg every 6 hours) dehydrates viable brain tissue (45) to create more space for swelling tissue, and is primarily useful as a temporizing measure for patients destined for decompressive surgery, as the effects of this medication are transient and associated with eventual rebound. Other measures such as mechanical hyperventilation (to a PaCO₂ of 25–30 mm Hg), or use of albumin and furosemide to raise colloid oncotic pressure (to 25–30 mm Hg) are used, but, again, their efficacy in improving outcome remains to be demonstrated.

Decompressive Surgery

Decompressive surgery, including hemicraniectomy and durotomies with temporal lobe resection, for treatment of brain edema after stroke has been a controversial topic. Many studies in the past have shown conflicting results, but these trials enrolled mixed age groups, and surgery was often not performed until symptomatic herniation occurred (46). Three large European trials (HAMLET, DESTINY, and DECIMAL) (47–49) that enrolled patients younger than 60 years of age and treated within 48 hours of onset were prematurely terminated after it became clear that decompressive craniotomy was associated with a dramatic survival and outcome benefit. In elderly patients, the results have generally shown that, while mortality may be decreased, outcomes remained poor (50).

The likelihood of hemorrhagic transformation of a stroke increases as stroke volume increases. Often, this transformation is limited to petechial transudation of blood products into the ischemic tissue bed. Generally, this phenomenon occurs in a delayed fashion with no associated clinical deterioration. Specific therapy is not usually required, although any ongoing anticoagulation is generally held for 1 to 2 weeks. If the hemorrhage is associated with clinical deterioration, management should follow those principles outlined in the intracerebral hemorrhage section below.

Early intirition of physical, occupational, and speech therapy services hastens functional recovery from stroke. Each patient requires individualized assessment for potential benefit from these services. Speech therapists are also commonly involved in formal assessment of aspiration risk. A video fluoroscopy swallowing study is the most sensitive measure and should be a consideration for most patients with a stroke. At the least, bedside swallowing function should be observed by a trained technician, nurse, or physician before oral intake is resumed.

**INTRACEREBRAL HEMORRHAGE**

Pathogenesis

In contrast to ischemic stroke, primary intracerebral hemorrhage (ICH) involves bleeding, usually of arterial origin, into normally perfused brain, and thus must be distinguished from hemorrhagic transformation of an initially ischemic stroke. The expanding hematoma causes direct injury to local brain tissue and dysfunction in surrounding regions. The onset is typically very sudden, although continued bleeding often progresses over minutes or hours. Very often, there is either depression or loss of consciousness due to an abrupt increase in intracranial pressure (ICP) from the sudden outpouring of blood into the brain. In addition to the initial cerebral insult caused by the hemorrhage, secondary injury can occur by various means, including seizures, hydrocephalus, and edema, all of which can lead to a further increase in ICP.

In younger patients, hypertension is by far the more common cause, and, as such, ICH tends to occur in the same brain areas where other hypertensive pathologies occur, specifically brainstem, cerebellum, and deep supratentorial structures (51). In contrast, lobar ICHs occur more commonly in the elderly population and are often associated with cerebral amyloid angiopathy in the absence of hypertension (52). ICH may also occur in the setting of trauma, use of illicit drugs (e.g., cocaine) or over-the-counter medications (e.g., phenylpropanolamine) (53), excessive alcohol consumption (54), an underlying vascular abnormality (e.g., arteriovenous malformation, cerebral aneurysm), brain tumor (primary or secondary), or a bleeding diathesis.

ICH causes approximately 10% of first-time strokes. The 30-day mortality rate is high at 35% to 50%, with half of the deaths occurring within the first 2 days (12). Outcome in ICH is dependent on several factors, including the location and size of the hemorrhage (55), the age of the patient, the Glasgow Coma Scale (GCS) on presentation (56), and the underlying cause of the hemorrhage. When intraventricular blood is present, the mortality substantially increases (57) and worsens further with increasing volume (58). The presence of hydrocephalus also confers a poor prognosis (59).

**Clinical Evaluation**

Rapid diagnosis of ICH is essential, as progression during the first several hours is the norm. The hallmark is sudden onset of focal neurologic deficit, which progresses over minutes to hours. Steady symptomatic progression of a focal deficit is rare in either ischemic stroke or subarachnoid hemorrhage. Headache, increased blood pressure, and impaired level of consciousness are common features that complete the presentation. History gathering should be directed at elucidating the presence of risk factors as outlined above. Other considerations include the use of antithrombotic medications (e.g., aspirin or warfarin) or hematologic disorders that predispose to bleeding, such as severe liver disease. The initial physical examination is similar to that of patients with ischemic stroke, focusing on airway, breathing, and circulation before assessing the level of consciousness and neurologic deficits. The patient's coagulation parameters should be checked immediately and corrected if abnormal.

Once stabilized, the patient should undergo a noncontrast head CT immediately to verify brain hemorrhage. CT angiography may also be helpful in detecting aneurysms, arteriovenous malformations (AVMs), underlying tumors, or abscesses. Contrast extravasation into the hematoma is thought to represent ongoing bleeding (60). MRI will also provide information...
The mainstays of medical treatment of acute ICH are correction of any coagulopathy and avoidance of hypertension. Most studies suggest that hematoma expansion occurs within the first several hours of onset, and therefore treatment must begin as soon as possible (63). If the patient recently received heparin, protamine sulfate (1 mg/100 units of heparin) should be administered; this drug is given carefully to avoid hypotension. Patients anticoagulated with warfarin with an elevated international normalized ratio (INR) should be reversed with vitamin K (10 mg intravenously administered over 15–20 minutes to prevent anaphylactoid reaction), as well as fresh frozen plasma to normalize the prothrombin time (PT).

Beyond the first few hours after onset, aggressive lowering of blood pressure may be potentially harmful. Large hemorhages will lead to increased ICP, and attention must be focused on maintaining the cerebral perfusion pressure (mean arterial pressure-intracranial pressure [MAP-ICP]) > 70 mm Hg to avoid secondary ischemia (64). Despite several clinical trials, a definitive guideline has yet to emerge. The American Stroke Association guidelines regarding blood pressure, presented in Table 84.8, are based on the best available, albeit incomplete, evidence. Table 84.9 lists recommended antihypertensives.

Seizures—occasionally nonconvulsive—occur commonly after ICH. The published incidence rates vary from 4% in unmonitored populations (65) to 28% in patients with continuous electrophysiologic monitoring in a neurocritical care unit (66). Prophylactic anticonvulsant medications may be considered for patients, especially for those with cortical involvement. If seizures are confirmed, they should be treated aggressively. Phenytoin remains the preferred first-line agent, as it is nonseeding, and loading doses can be given intravenously. Loading doses are 15 to 20 mg/kg and should be given as fosphenytoin if needed quickly to avoid hypotension and potential toxic infusion reactions. Maintenance doses are often 4 to 5 mg/kg and may be given slowly. Total serum levels of phenytoin should be followed daily, at least initially. Free levels may be necessary, as phenytoin is protein bound, and critically ill patients are very frequently protein depleted. Additional antiepileptics should be added as necessary. The duration of therapy with anticonvulsants is unclear, but in seizure-free nonepileptic patients, antiepileptic medications are often arbitrarily withdrawn after 4 to 6 weeks of therapy.

Other general supportive care measures are similar to those described above for ischemic stroke, including including DVT prophylaxis, and treatment of hyperthermia and hyperglycemia. Patients with increased intracranial pressure often develop disturbances of free water homeostasis in the form of either hyponatremia or hypernatremia. As with ischemic stroke, corticosteroids for treatment of edema are of no benefit and actually increase morbidity (67). Treatment of increased ICP should initially focus on more conservative noninvasive measures, such as keeping the head of the bed at 30 degrees, hyperextension if intubated, and osmolar therapy with hypertonic saline or mannitol. An implanted ICP monitor should be considered for those patients with large hematomas. This will inform the decision to place an intraventricular drain or perform a surgical evacuation.

In general, patients with a GCS of 4 or more have a uniformly poor outcome, whether or not surgery is performed.
and thus these patients should be treated medically. Patients with cerebellar hemorrhages > 3 cm in diameter should be considered for emergency decompression, especially if there are signs of brainstem compression, hydrocephalus, or neurologic deterioration (64); whether surgery is indicated in most other patients is not clear. Clinicians treating patients who deteriorate despite maximal medical therapy may turn to surgical decompression, but results from clinical trials have been mixed (68,69). Patients with lobar hemorrhages secondary to amyloid angiopathy have exceptionally friable cortical blood vessels and are poor surgical candidates.

**TABLE 84.10**

<table>
<thead>
<tr>
<th>Severity Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 1: Fully conscious, no neurologic deficit, headache only</td>
<td></td>
</tr>
<tr>
<td>Grade 2: Mild drowsiness, no neurologic deficits other than cranial nerve dysfunction</td>
<td></td>
</tr>
<tr>
<td>Grade 3: Drowsy, mild neurologic deficit</td>
<td></td>
</tr>
<tr>
<td>Grade 4: Stuporous, moderate to severe neurologic deficits</td>
<td></td>
</tr>
<tr>
<td>Grade 5: Coma</td>
<td></td>
</tr>
</tbody>
</table>


**SUBARACHNOID HEMORRHAGE**

Subarachnoid hemorrhage (SAH) is a relatively uncommon but often devastating type of stroke. Incidence is estimated at 30,000 patients per year in the United States, with a mortality that exceeds 30%. Whereas head trauma is the most frequent cause of subarachnoid hemorrhage, aneurysmal rupture results in the greatest morbidity and mortality. Clinically, this is an apoplectic disorder. Most commonly, patients perceive a sudden severe headache with rapid impairment of consciousness, both symptoms related to the sudden release of irritating blood products into the meningeal spaces surrounding the brain. Focal neurologic symptoms such as hemiparesis, sensory loss, or diplopia may occur if localization of subarachnoid blood or intraparenchymal extension of the hemorrhage develops. The most important features of the neurologic examination are the assessment of level of consciousness, cranial nerve function, and motor function. Clinical severity of SAH is graded on these findings (70) (grades I to V, Table 84.10) and can be rough prognostic indicators.

**Diagnosis**

Diagnosis of SAH is based on neuroimaging or cerebrospinal fluid (CSF) analysis. Brain CT scan is a very sensitive indicator of the presence of subarachnoid blood, although close examination must be paid to the subarachnoid spaces surrounding the brainstem and over the cerebral convexities (Fig. 84.5). Brain parenchyma itself most commonly displays no acute abnormalities. Erythrocyte concentration in CSF below approximately 30,000 cells/μL may not result in the diagnostic increased density within CSF on CT scans. In approximately 10% of patients, diagnosis therefore requires CSF analysis through lumbar puncture. In addition to elevated erythrocyte count, CSF xanthochromia and elevation of CSF D-dimer can often be detected in true subarachnoid hemorrhage. The latter two findings may help distinguish bloody CSF from a “traumatic tap,” as these serve as markers of the breakdown of thrombosis or blood products. Serial cell counts should always be obtained, however, whenever SAH is suspected. Cell counts in SAH should be roughly equivalent in all tubes, whereas a declining count is usual in traumatic punctures. It should be stressed that lumbar puncture should be avoided in any patient with a depressed level of consciousness until CT scan excludes a focal mass (such as intraparenchymal or subdural hemorrhage). If bacterial meningitis is a concern, blood

**FIGURE 84.5**. Subarachnoid hemorrhage. Blood is imaged as hyperdense fluid within the cisterns surrounding the brainstem and within bilateral sylvian fissures.
TABLE 84.11

<table>
<thead>
<tr>
<th>Complication</th>
<th>Clinical features</th>
<th>Diagnostic tests</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased ICP</td>
<td>Decreased alertness, worsened headache, herniation syndrome</td>
<td>ICP monitor</td>
<td>Mannitol, steroids, hyperventilation</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>Decreased alertness, worsened headache, herniation syndrome</td>
<td>CT scan</td>
<td>Ventriculostomy drainage or shunt</td>
</tr>
<tr>
<td>Vasospasm</td>
<td>Delayed focal neurologic deficit</td>
<td>TCD, angiography</td>
<td>Nimodipine, hypervolemia, hypertension, angioplasty</td>
</tr>
<tr>
<td>Rebleed</td>
<td>Worsened neurologic condition, especially level of consciousness</td>
<td>CT scan, lumbar puncture</td>
<td>Ablation of aneurysm</td>
</tr>
<tr>
<td>Seizure</td>
<td>Sudden behavioral change or uncontrolled motor activity</td>
<td>EEG</td>
<td>Anticonvulsants</td>
</tr>
<tr>
<td>Hyponatremia</td>
<td>Confusion, lethargy</td>
<td>Serum electrolytes</td>
<td>Isotonic fluids to achieve euvoolemia or hyperalimentation</td>
</tr>
<tr>
<td>Infection</td>
<td>Confusion, lethargy</td>
<td>Panculture, chest radiograph, urinalysis</td>
<td>Appropriate antibiotic</td>
</tr>
</tbody>
</table>

ICP, intracranial pressure; CT, computed tomography; TCD, transcranial Doppler; EEG, electroencephalogram.

Management

Patients with acute SAH are at high risk for a multitude of complications (71) (Table 84.11) that usually mandate admission to an intensive care facility. All patients should be placed on strict bed rest, with appropriate precautions for deep venous thrombosis and aspiration. Patients with progressive lethargy may require intubation for airway protection and mechanical ventilation. Until the aneurysm has been ablated, blood pressure should be kept in the normotensive range, and isotonic intravenous fluids should be used to maintain normovolemia. All patients should be started on nimodipine at 60 mg every 4 hours (duration 21 days), either orally or through a nasogastric tube, for prevention of vasospasm (see below).

Historically, patients were placed on prophylactic anticonvulsants; this practice has recently come into question, as patients enrolled in the international tirilazad trials who were on prophylactic anticonvulsants had significantly more in-hospital complications and worse clinical outcomes (72). ECG changes and elevations in cardiac enzymes, troponin, and CK-MB are commonly seen in SAH patients and may represent the phenomenon of stunned myocardium, in which case management should be aimed at optimizing left ventricular function to support cardiovascular and cerebrovascular perfusion (73–76). Serum electrolytes are closely monitored, as hyponatremia may be seen in more than 30% of patients after SAH; however, hyponatremia can occur as well and is significantly associated with clinical outcome (77). The cause of hyponatremia after SAH is most commonly reported to be due to syndrome of inappropriate antidiuretic hormone (SIADH) but can also be due to cerebral salt-wasting (CSW) syndrome and other causes (78). One theory links SAH-induced hyponatremia to levels of serum brain natriuretic peptide, which is thought to be associated with delayed ischemic neurologic deficits (79). It is intuitive that SIADH and CSW are not treated in the same manner.

Serum glucose levels should also be closely monitored, as hyperglycemia has been significantly associated with mortality and poor functional outcome in SAH patients (80). Because fever in SAH patients has been associated with mortality and poor clinical outcome (80), and has even been linked to vasospasm (81), patients should be kept normothermic. Platelet levels should be monitored, as a relatively significant incidence of heparin-induced thrombocytopenia has been reported in SAH patients (82).

Rebleeding

In those patients surviving the initial hemorrhage, the leading factor associated with mortality is rebleeding from the aneurysm. A second bleed from an aneurysm is associated with a 74% mortality rate (83). Untreated aneurysms rebleed at a rate of 4% on day 1, then 1% to 2% a day for the next 4 weeks. Thus, early treatment to secure a ruptured aneurysm is critical. This can be done either via a craniotomy and clipping, or by endovascular coiling. The findings of the International Cooperative Study on the Timing of Aneurysm Surgery concluded that aneurysmal clipping was best performed either early (0 to 3 days) or late (11 to 14 days), but outcome was worse when performed at 7 to 10 days after the onset of SAH (84). Because of the concern for rebleeding, we have adopted a protocol of treating aneurysms in the ultra-early period (less than 24 hours after presentation). The advent of endovascular therapy to treat ruptured aneurysms may bypass the risks associated with open clipping during the 7- to 10-day period; thus we recommend treatment for the ruptured aneurysm as soon as it is found, and not waiting until the late 11- to 14-day period. Early treatment of the ruptured aneurysm also allows aggressive management.
of vasospasm, manipulations that would increase the risk of rebleeding from an unsecured aneurysm.

Vasospasm

After rebleeding, vasospasm is the next leading cause of mortality and morbidity from a SAH (85,86). The exact cause of arterial vasospasm following SAH is unknown, but its incidence does appear to be correlated with the density of blood products seen on CT scan, the basis for the Fisher score to predict vasospasm (87,88). Severe vasospasm may result in cerebral infarction within the vascular distribution of the involved artery. The risk for vasospasm begins about 3 days after the bleed and may persist for 3 weeks. Transcranial Doppler is a sensitive, noninvasive indicator of the presence and degree of vasospasm within proximal arteries, although it may not detect vasospasm restricted to smaller peripheral vessels. This technique may be used daily to guide and monitor management strategies. Modern techniques, such as CT angiography and CT perfusion studies, have been reported to be successful in diagnosing vasospasm (89) (Fig. 84.6).

The calcium channel blockers nimodipine and nicardipine can reduce the incidence of vasospasm as well as associated cerebral infarction. Trials with magnesium sulfate have yielded promising results in reducing vasospasm (93–95) or achieving better clinical outcomes in patients (93). Studies with statin therapy (HMG Co-A inhibitors), such as pravastatin and simvastatin, have demonstrated promising results with reduced rates of vasospasm and better clinical outcomes (94–97).

Once the aneurysm is secured, vasospasm can be managed aggressively. Triple-H therapy—hypertension, hyper-therapies (HMG Co-A inhibitors), such as pravastatin and simvastatin, have demonstrated promising results with reduced rates of vasospasm and better clinical outcomes (94–97).

Once the aneurysm is secured, vasospasm can be managed aggressively. Triple-H therapy—hypertension, hyper-therapies (HMG Co-A inhibitors), such as pravastatin and simvastatin, have demonstrated promising results with reduced rates of vasospasm and better clinical outcomes (94–97).

Acute Hydrocephalus

Acute hydrocephalus occurs in approximately 20% of survi-vors of SAH, either as a result of direct obstruction of CSF channels or by impeding CSF absorption at arachnoid granulations. The likelihood of hydrocephalus increases with worsening grade of hemorrhage. Ventriculostomy drainage is recommended for patients with acute hydrocephalus and decreased level of consciousness; improvement can be expected in >50% of patients.

Chapter 84: CNS Vascular Disease

Other modalities of monitoring, from pulse-waveform variability (FlowTrak, PICCO, LiDCO) to placement of a pulmonary artery catheter, may be necessary to properly care for these patients. If hypertensive therapy is not adequate to control vasospasm, hypertension can be initiated by cessation of antihypertensives or use of vasoressors (e.g., phenylephrine, 0.1–5 μg/kg per minute, or vasopressin, 0.01–0.04 units per minute), targeting mean arterial pressures of 120 to 140 mm Hg (systolic blood pressure [BP] 180–200 mm Hg). Triple-H therapy does not increase the risk of rupture of other, incidentally found, aneurysms in patients with multiple aneurysms (98).

For symptomatic vasospasm refractory to these therapies, endovascular interventions can be performed, such as percu-taneous transluminal balloon angioplasty, and/or intra-arterial administration of calcium channel blockers or other vasodilating agents (99) may be considered in experienced hands. Even using the most aggressive management strategies, vasospasm remains a leading cause of morbidity and mortality after subarachnoid hemorrhage.

References

1276

Section VIII: The Surgical Patient


Chapter 84: CNS Vascular Disease

1277


