Anesthesia for Interventional Neuroradiology

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Therapeutic interventions unique to the neuroradiology suite include the endovascular treatment of cerebral aneurysms, arteriovenous malformations (AVM), acute thromboembolic disease, cerebral vasospasm, and extracranial and intracranial cerebrovascular atherosclerotic disease. A recent review of the National Inpatient Sample reported a doubling of the number of endovascular cases from 1993 to 2003, particularly in elderly patients.[1] The increase in endovascular neurosurgical volume for aneurysmal cerebrovascular disease mirrors the evolution of coil technology since its initial FDA approval in the mid-1990s, and reflects the increasing treatment of ruptured and unruptured aneurysms, especially when surgical clipping is thought to involve unacceptable patient risk.[2] Endovascular treatment volumes for cerebrovascular disease will likely continue to increase as coil and stent technologies improve, the number of aged individuals with these pathologies increases, and the population comorbid disease burden contributes to net patient movement from the operating room to the endovascular suite. However, recent studies of the comparative efficacy of interventional versus medical therapy are tempering the push toward

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intervention in some patients with intracranial atherosclerotic disease,[3] unruptured AVMs,[4] and acute ischemic stroke.[5]

A number of considerations specific to this unique and growing anesthetic practice must be understood and managed if the anesthesiologist is to perform effectively to optimize patient outcome. Maintenance of patient immobility, ensuring rapid recovery to facilitate postprocedure neurological evaluation, and close hemodynamic management to avoid cerebral hypo/hyperperfusion are common to neuroanesthetic practice. Management of sudden interventional neuroradiology (IR) procedure-specific complications and critical care patients during transport to and from the endovascular suite, meeting appropriate "off-site" equipment needs if endovascular procedures are not performed in the main OR complex, and providing for appropriate personal radiation safety are unique to interventional neuroradiology practice.[6] This review addresses general considerations for anesthetic practice in the interventional neuroradiology setting, anesthetic management of specific procedures and disease states, and a concise discussion of femoral vessel closure devices for the anesthesiologist.

■ General Considerations and Choice of Anesthetic Technique

All patients undergoing interventional neuroradiologic procedures require careful preoperative evaluation, with specific attention given to underlying neurovascular pathology. Neurological evaluation should include assessment and documentation of any focal deficits, including level of consciousness.[7] Anesthesia may unmask prior neurological injury, in which patients who have had full recovery of a prior fixed deficit through neural "rewiring" may emerge from anesthesia with temporary reversal of compensation (ie, reemergence of prior deficits).[6,8,9] Evaluation of comorbidities requires particular attention related to tolerance of intravenous (IV) contrast (renal disease, allergy to IV contrast, shellfish, iodine), protamine (allergy), and the choice of general versus monitored anesthesia care (specifically cardiovascular reserve, presence of arthritis of neck, back, or major joints, and potential for difficult airway).[7] Notation of preoperative blood pressure is also important, as hypertension and underlying neurovascular disease may narrow and/or shift the autoregulatory window to higher values.[10,11] Hematological status is relevant due to the frequent use of anticoagulation during IR procedures. In particular, patients undergoing endovascular stent placement typically need therapeutic dual antiplatelet therapy before the procedure.[12] Interventionalists may want laboratory evidence of platelet inhibition (such as a platelet function assay) before the endovascular procedure given the pharmacogenomics

of P2Y12 inhibition with clopidogrel and potential interindividual variability in aspirin effect.[13]

■ Choice of Anesthetic Technique

Both general anesthesia (GA) and sedation are used routinely in IR procedures; preference varies by center, case type, and surgeon.[7]

General Anesthesia (GA)

Imaging quality and safe device deployment (coil, stent, embolic material) is highly dependent upon patient immobility, which remains the primary reason for the selection of GA in interventional neuroradiology cases. Techniques including high-resolution fluoroscopy, high-speed digital subtraction angiography (DSA), and *road mapping*, in which an image of vascular anatomy obtained through intraluminal contrast bolus is superimposed onto live DSA fluoroscopic imaging, enabling the effective use of microcatheters in distal small vessels.[7] Both the technical considerations of accurate small vessel imaging and the higher risk of vascular puncture in delicate end-vasculature make GA an attractive option for many IR cases. In addition, as vessel manipulation is often painful, many interventionalists prefer GA to improve patient comfort and respiratory and hemodynamic control. Disadvantages of GA include inability to assess intraoperative neurological function, possible anesthetic-induced hypotension causing cerebral hypoperfusion, unsatisfactory hemodynamics during tracheal intubation and/or extubation, and coughing and straining on emergence with resultant groin complications or potential intracranial pressure (ICP) surges.[7]

Premedication must be judicious and individualized to patient needs. A laryngeal mask airway may be used in appropriate cases to avoid hemodynamic variability with endotracheal intubation and extubation; however, the need to provide immobility and occasional "breath-holds" usually necessitate endotracheal intubation and neuromuscular blockade.[7] Topical lidocaine (ie, laryngotracheal analgesia), remifentanil boluses (1 to 2 mcg/kg), or esmolol boluses can be used to blunt the hemodynamic response to endotracheal intubation. Sevoflurane seems to provide a more rapid recovery than propofol-based total intravenous anesthesia (TIVA).[14] Although not evidence based, in our experience a combination of low-dose sevoflurane and propofol infusion can provide a rapid emergence while minimizing interference with neuromonitoring techniques (ie, cases requiring electroencephalography and motor-evoked potential monitoring). Nitrous oxide is best *avoided* in IR cases, as intra-arterial air bubbles injected in contrast boluses or during catheter/wire exchanges can cause cerebral infarction and could enlarge/worsen in the presence of nitrous oxide. Remifentanil

is an ideal intraoperative analgesic due to the minimal postoperative pain associated with most IR procedures and its lack of context sensitivity that allows deep opioid analgesia and subsequent rapid emergence.[15] Remifentanil decreases hypnotic anesthetic requirements and facilitates a smooth emergence without coughing or residual effects (the so-called "remi wake-up"). The use of intraoperative monitoring with motor, somatosensory, or brainstem auditory-evoked potentials, or the use of continuous electroencephalographic monitoring may necessitate certain anesthetic regimens.[16]

Sedation

Benefits of sedation include the ability to perform intraoperative neurological testing and avoidance of the hemodynamic consequences of GA. In addition, retrospective data suggest that sedation may be superior to GA for endovascular treatment of acute ischemic stroke with respect to patient outcome.[17] Disadvantages of sedation for IR procedures include lack of airway control and aspiration risk in nonfasted patients, suboptimal access to the airway in the event of intraoperative airway emergencies, potential for hypoxemia and/or hypercapnia, and patient movement or agitation. Propofol and dexmedetomidine are both commonly used for sedation in this population.[18] Dexmedetomidine may confer advantages through decreased depressant effects on respiration and level of consciousness.[7]

■ Equipment

The overall configuration of the neuroradiology suite is ergonomically challenging (Fig. 1). Biplanar fluoroscopy, image display monitors, and ultrasound devices surround a procedural patient table that is typically fixed to the floor. In addition, imaging devices must be able to rotate freely around the patient's head during IR procedures, requiring anesthetic equipment to be out of the way. The interventionalist also needs unimpeded access to the groin for catheter insertion. Extension tubing is often needed to facilitate IV fluid and drug delivery along with extensions on the breathing circuit. Care should still be taken to ensure the delivery of vasoactive, primary anesthetic, or anticoagulant drugs as proximal to the patient as possible to minimize tubing dead space, preferably through dedicated intravenous access.[18]

■ Management of Critically Ill Patients During Transfer

Transport of a critically ill patient, often with multiple comorbidities and rapidly deteriorating neurological function, requires careful planning, anticipation of staffing and equipment needs, and attention

Figure 1. *The endovascular neurosurgery suite may pose ergonomic challenges and may be geographically remote from the main OR suite.* full color online

to clinical detail. Optimal medical management of critically ill patients during transport involves understanding the neurological pathophysiology (eg, Is it safe to clamp the ventriculostomy? Should we monitor ICP during transport?), and the hemodynamic and respiratory status, through targeted communication with primary caregivers. Transport may require responding to changes in ICP, continuing and titrating vasoactive and other infused medications, and completing the transfer expeditiously. Attention must be given to ensure that appropriate medications and equipment are available, including vasoconstrictors, inotropes, sedatives, anticonvulsants, paralytics, IV flush solution, intubation equipment, bag valve mask, and adequate tank oxygen supply, particularly in the event of a power failure in the elevator. At least 2 personnel, with at least one certified in advanced cardiovascular life support, should attend the critically ill patient during transport to and from the IR suite.

■ Blood Pressure Management (Including Deliberate Hypertension and Hypotension)

Management of blood pressure within a narrow range is a mainstay of IR anesthetic management. Significant deviations from the autoregulatory cerebral arterial pressure range may adversely impact neurophysiology, neurological outcome, and survival.[19] In addition, specific procedures and/or intraprocedural crises may benefit from deliberate hypotension or hypertension; these considerations are discussed in subsequent sections. It is critical for the anesthesiologist to assess baseline blood pressure and degree of chronic preoperative hypertension to ascertain the likely autoregulatory range and tolerance for various cerebral perfusion pressures. Delay in recognition of hemodynamic perturbation or collapse in the absence of intra-arterial monitoring can be disastrous. Thus, interventional cases involving the spinal or intracranial arterial system

typically require placement of an intra-arterial catheter for beat-to-beat blood pressure monitoring. Diagnostic angiography cases typically do not. In cases where radial artery cannulation is deemed unsafe or not feasible, the side port of the femoral artery introducer sheath can permit intra-arterial pressure monitoring during the procedure. Central venous catheterization is seldom necessary for anesthetic management in the IR suite, the need for prolonged inotropic or pressor administration being the obvious exception.

Deliberate Hypertension

Management of acute intraprocedural arterial occlusion (such as intentional balloon occlusion or inadvertent thrombotic occlusion) may require deliberate hypertension. Mean blood pressures increases of 30% to 40% above baseline may be pharmacologically induced in an attempt to increase cerebral blood flow (CBF) through collaterals or through a partially occluded end-artery. At present, little data exist to suggest improved outcomes resulting from this technique compared with maintenance of normotension; rather this intuitive approach is based on the current understanding of cerebrovascular pathophysiology. Deliberate hypertension may be induced with multiple different vasoactive agents; a pure alpha agonist, such as phenylephrine, is usually chosen due to ease of titration and lack of inotropic effect. Careful electrocardiographic monitoring is advised given the potential increase in afterload, particularly in patients with severe cardiac disease.[18] Risk of acute hemorrhage due to cerebral aneurysm or AVM rupture from deliberate hypertension must be weighed against potential therapeutic benefit on a case-by-case basis.[20] As always, close communication with the endovascular surgeon is essential to accurately assess intraoperative issues and define mutually agreed upon hemodynamic goals, duration of therapy, etc.

Deliberate Hypotension

As with induced hypertension, little data exist to suggest improved outcomes resulting from deliberate hypotension compared with maintenance of normotension. However, indications for induction of deliberate hypotension may be to define cerebrovascular reserve before carotid occlusion procedures and to decrease blood flow into an AVM immediately before intra-arterial liquid embolization (flow arrest).[18] Further, it may be necessary to temporarily decrease systemic blood pressure in the setting of acute iatrogenic intracerebral hemorrhage (usually due to vessel rupture from an endovascular device) in an attempt to limit further bleeding while the endovascular surgeon attempts to treat the injury. Adenosine, esmolol, clevidipine, or

nicardipine may be utilized. An intuitive initial approach would be a propofol bolus to create both hypotension and burst suppression (ie, some pharmacologic neuroprotection).

■ Anticoagulation and Reversal

Thromboembolic complications of IR procedures may be avoided by judicious use of anticoagulant medications. Unfractionated heparin is most commonly used, with an initial dosing of ~50 to 70 U/kg with titration to an activated clotting time (ACT) of 2 to 3× preheparin ACT.[18] Hourly ACT determination guides continuous or bolus heparin dosing. Cases of heparin resistance are commonly attributed to acquired antithrombin III deficiency resulting from consumption and may be treated with fresh-frozen plasma or antithrombin III concentrates.[21] Reversal of heparin is undertaken by administration of protamine (1 mg per 100 units heparin given).[18] Hypotension associated with rapid protamine dosing should be avoided.

Patients with heparin allergy or those at high risk for heparin-induced thrombocytopenia may require the use of direct thrombin inhibitors in cases requiring anticoagulation. Direct thrombin inhibitors inhibit free and clot-bound thrombin. Hemorrhage and anaphylaxis are potential adverse events associated with their use.[22] Lepirudin and bivalirudin undergo plasma metabolism and renal elimination, whereas argatroban undergoes hepatic metabolism and fecal elimination; the latter may be useful in patients with renal dysfunction.[23]

Antiplatelet medications, including aspirin, glycoprotein IIb/IIIa antagonists, and thienopyridines, are often needed in IR cases, particularly those involving stent placement. Success in reducing morbidity and mortality through the use of such drugs in the setting of coronary intervention has resulted in their application to cerebrovascular interventions.[24,25] The thienopyridine drug clopidogrel, in particular, is commonly added to the drug regimen (along with aspirin) for stent placement, or stent-assisted aneurysm coiling, usually in the setting of unruptured aneurysms.[18] Because of pharmacogenomic differences in response to clopidogrel, prasugrel may function as an alternative P2Y12 platelet inhibitor. Some interventionalists request platelet activity assays before the procedure to evaluate the individual response to P2Y12 inhibitors. Finally, intravenous abciximab is commonly used to treat acute thromboembolic complications of endovascular cerebral procedures either intraprocedure or postprocedure.[26]

Both direct thrombin inhibitors and antiplatelet agents have no specific antidotes. Biological half-life is of paramount importance in drug selection. Intravenous desmopressin (DDAVP) may be used to decrease the effect of antiplatelet drugs, but platelet transfusion remains

Table 1. *Commonly Used Anticoagulants and Their Respective Reversal Agents*

Anticoagulant	1st Line Therapy	2nd Line Therapy
Heparin	Protamine	
Direct thrombin inhibitors	Controversial/unproven[30]	Prothrombin complex concentrate, FFP, recombinant-activated factor VII
Warfarin	4-factor prothrombin complex concentrate	Recombinant-activated factor VII, FFP, vitamin K
Aspirin	Platelets	DDAVP
Clopidogrel	Platelets	DDAVP
IIb/IIIa inhibitors	Platelets	DDAVP

standard therapy for reversal of the effects of direct thrombin inhibitors and other antiplatelet drugs.[27,28] Use of specific clotting factors such as recombinant factor VII may be considered in cases of life-threatening bleeding uncontrolled with platelet transfusion therapy.[18] Intravenous thrombolysis for acute stroke intervention [typically with alteplase; ie, tissue plasminogen activator (tPA)] can be reversed with cryoprecipitate[29] (Table 1).

■ Radiation Exposure Risk and Protection

Ionizing radiation is an occupational hazard of particular concern in the IR suite. The Occupational Health and Safety Administration, among other regulatory agencies, updates maximum allowable exposure limits for ionizing radiation in health care workers; exposure should be measured through the use of an exposure badge and minimized to the extent possible.[31] For this reason, protective lead clothing, including apron (covering front and back), thyroid shield, and leaded eye protection should be worn throughout the procedure.[32] Leaded plexiglass panels are also useful for shielding anesthesia personnel. Radiation sources in the IR suite come directly from the fluoroscopy tube, leakage around and through the collimators' protective shielding, and scatter.[7,18] As exposure decreases proportionally to the inverse of the square of the distance from the radiation source, attempts should be made to remain as great a distance as feasible from the fluoroscopy tube and limit activity near the patient's head during fluoroscopy. DSA entails significantly more ionizing radiation output than fluoroscopy and requires particular attention to the employment of radiation safety measures. Monitoring from behind a leaded glass screen or adjacent console room is preferable in this case.[18]

■ Crisis Management

Intraprocedural complications in IR are often rapid and may be catastrophic, particularly if not managed promptly and effectively. In all such cases the first step is for the anesthesiologist and neurointerventionalist to communicate immediately and efficiently as soon as a complication is suspected. The anesthesiologist must immediately ensure a secure airway (if not already secured) and ventilation with 100% oxygen. Further management will depend on whether the crisis is ischemic or hemorrhagic. In ischemic complications (eg, vessel occlusion, vasospasm), deliberate hypertension should be considered. The neurointerventionalist may attempt to alleviate the occlusion through emergent angioplasty, stenting, mechanical lysis via guidewire, clot extraction, or intra-arterial thrombolysis or vasodilators.[33,34] The degree of hypertension induced must respect patient comorbidity and can be titrated to neurological examination in an awake patient (rarely) or angiographic endpoint.[18] In hemorrhagic complications, immediate reversal of anticoagulation and transient-induced hypotension should be considered. The Cushing response (hypertension and bradycardia) may accompany otherwise difficult to diagnose intracranial hemorrhage. The interventionalist will attempt to coil or balloon-occlude the site of hemorrhage. Emergent placement of a ventriculostomy catheter to alleviate high ICP and provide subsequent monitoring may be necessary. Subsequent CT imaging and possibly emergent craniotomy can be required.[6,7] The anesthesiologist should control hemodynamics to attempt to maintain adequate cerebral perfusion pressure (typically >60 mm Hg). Utilizing hyperventilation, head elevation to 15 to 30 degrees, intravenous mannitol boluses, and burst suppression are other temporizing measures to manage critical ICP elevations and/or herniation syndromes.

Anesthesia for Specific Cerebrovascular Disease States

Endovascular Aneurysm Surgery Long-term follow-up results from the International Subarachnoid Aneurysm Trial published in 2009 demonstrated superior outcomes from endovascular coiling treatment of ruptured anterior and posterior circulation aneurysms compared with neurosurgical clipping in risk of patient death at 5 years (11% vs. 14%, log-rank, $P = 0.03$) (Fig. 2 and Video, Supplemental Digital Content 1, http://links.lww.com/AIA/A21).[35] As a result, endovascular treatment is now considered first-line therapy for many ruptured and unruptured aneurysms.[36]

Anesthetic implications for endovascular aneurysm therapy include maintenance of hemodynamic stability, especially during induction and emergence if GA is used, while maintaining appropriate cerebral

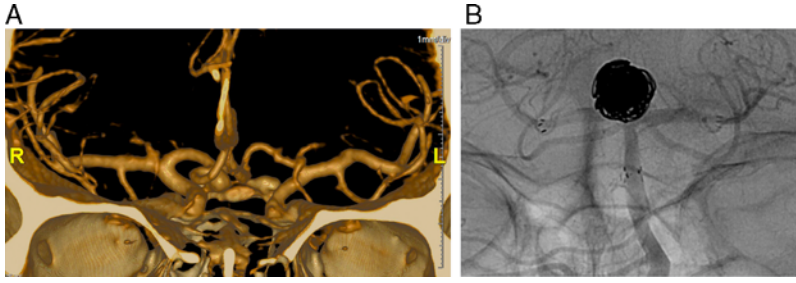

Figure 2. *A, Anterior communicating artery aneurysm (CT angiogram 3D reconstruction). B, Stent-assisted coil embolization: Y-shaped stent extending from the basilar artery into bilateral posterior cerebral arteries and coil-packed basilar tip aneurysm.* full color online

perfusion. Bolus esmolol, laryngotracheal lidocaine, or bolus remifentanil[37] can facilitate this. Use of any volatile anesthetic agent may result in increased CBF, cerebral blood volume, and ICP in patients with poor intracranial compliance or elevated ICP at baseline; use of <1 MAC volatile agent generally limits significant cerebral hyperemia while maintaining the cerebrovascular constrictive response to hypocapnia. Sevoflurane may provide the most favorable emergence profile.[38,39] Propofol TIVA also reduces CBF, ICP, and metabolic demand but often at the cost of a slower emergence compared with sevoflurane, especially for long procedures.[14] Although avoiding GA in favor of sedation may mitigate some undesirable cardiovascular and intracranial hemodynamic effects, some authors report that greater than a quarter of sedation coiling cases may be aborted or associated with medical or technical adverse events.[40]

Keep in mind that the reduction in brain bulk achieved with moderate hypocapnia for open neurosurgical exposure is not required in these procedures. Patients with hydrocephalus [such as in the setting of subarachnoid hemorrhage (SAH)] and elevated ICP should have a preprocedural ventriculostomy catheter placed. ICP control can largely be accomplished with intermittent CSF drainage (target <20 mm Hg) and mild intermittent hypocapnia. As mentioned above, sub-MAC concentrations of volatile agent are usually tolerated, but persistent ICP elevations may require a TIVA.

The anesthesiologist must be attentive to the possibility of aneurysmal rupture (or rerupture) and acute SAH during the intervention.[18] Rupture may be heralded by acute hypertension with or without bradycardia or may have no hemodynamic consequences. Acute treatment involves the measures discussed above under "Crisis Management." Coiling may continue or be abandoned in favor of rescue craniotomy and clipping.

Patients with aneurysmal SAH may present to the endosurgical suite with cranial nerve palsies, seizures, hydrocephalus, cerebral edema, and

dysfunction in non-CNS organ systems. Hypertension is common and may be managed with a parenteral titratable dihydropyridine calcium channel blocker (nicardipine or clevidipine). Target systolic blood pressure (SBP) is typically <160 mm Hg by guideline[41] although this target may be lower based on patient-specific factors. Cardiac dysfunction, termed "stress cardiomyopathy" or "takatsubo cardiomyopathy" is believed to be due to massive catecholamine release around the time of the hemorrhage and can produce symptomatic heart failure.[42] Although rarely seen, neurogenic pulmonary edema can also complicate acute SAH.[43] Common electrolyte disturbances after SAH include hypomagnesemia, hyponatremia due to cerebral salt wasting and/or SIADH, hypokalemia, and hypocalcemia.[44] Poor glucose control worsens overall outcome and serum glucose should generally be kept <180 mg/dL.[45] As discussed above, elevated ICP may be managed by ventricular drainage, while avoiding overdrainage, which may increase the risk of aneurysmal rebleeding by decreasing the transmural pressure across the aneurysm.

AVM Therapy—Cerebral, Dural Cerebral AVMs comprise a nidus of abnormal vessels containing arterial inflow and venous outflow, often in the absence of a capillary component. These AVMs are typically congenital, with a lifetime prevalence of 0.5% to 6%.[46] In contrast, dural AVMs are considered to be acquired, often due to venous dural sinus stenosis or occlusion, with opening or recanalization of a potential fistulous tract due to venous hypertension. Endovascular treatment may be curative in the 20% of cerebral AVMs characterized by only 1 or 2 feeding vessels[7]; however, subsequent surgery or radiotherapy is often necessary and facilitated by endovascular embolization of feeding arteries.[18] Mortality due to embolization is ~1%.[47] Multistaged embolizations may be necessary in complex AVMs with multiple feeding vessels and in the case of dural AVMs, which are commonly fed by multiple meningeal vessels.[7,18] Embolization materials include liquid polymer [most commonly ethylene vinyl alcohol in dimethylsulfoxide (Onyx), EV3, Plymouth, MN] and *N*-butyl cyanoacrylate (Trufill, Depuy Synthes, Warsaw, IN) glue.

GA is generally advisable due to enhanced vessel visualization, lack of patient movement during delicate catheter navigation in friable vessels, and possible need for deliberate hypotension or temporary induced cardiac arrest to counteract venous hypertension in dural fistulae.[18] Normotension is advisable during the majority of cerebral AVM cases; induced hypertension may be advisable in the event of inadvertent vascular occlusion but must be offset by the associated risk of AVM rupture.[48] Short periods of deliberate hypotension using rapidly titratable short-acting agents (eg, volatile anesthetics, propofol, sodium nitroprusside, clevidipine) may be helpful in producing flow arrest through a

cerebral AVM, which allows glue to set rather than be carried off into draining veins; transient asystole produced by adenosine administration or rapid ventricular pacing is an option in selected patients.

Cerebral hyperperfusion with resultant cerebral edema and hemorrhage may result from abrupt restoration of normal perfusion pressure, after AVM treatment, to chronically hypoperfused vascular beds that have lost their autoregulatory capacity.[49] Maintenance of SBP 15% to 20% below baseline in the immediate postoperative period may be protective.[50] Other procedural complications include neurological deficits due to occlusion of normal vessels, intracranial hemorrhage due to vessel perforation, vessel dissection, rupture of aneurysms associated with the AVMs, and regional cerebral hypoperfusion/ischemia (ie, steal phenomenon).[47]

Wada testing involving intra-arterial injection of amobarbital may be used to define eloquent cortex location (ie, language or motor areas) adjacent to the AVM; sedation for these cases must respect the need for minimal impact on concurrent motor and cognitive examinations and may be altogether unnecessary in the setting of appropriate local anesthetic infiltration at the puncture site.[18]

Anesthesia for Ischemic Cerebrovascular Disease

Acute Stroke Interventions IV fibrinolysis with tPA (alteplase) is curative in only half of the 3% to 8.5% of stroke patients who receive it, with particularly poor rates of recanalization in the case of large artery occlusion (see Fig. 3).[51,52] Endovascular treatment of acute large artery cerebro-occlusive disease involves intra-arterial thrombolytic administration or more commonly (in light of superior outcomes) mechanical thrombectomy.[53,54] Mechanical thrombectomy techniques include guidewire clot maceration, clot retrieval with helical or stent-like retrievers, thrombus aspiration, angioplasty, and even stenting.[17] Recent prospective randomized trials have demonstrated the superiority of stent-like clot retrievers over helical retrievers.[55,56] However, a recently published trial[57] suggests IV tPA should remain first-line therapy, and endovascular stroke therapy should be reserved for patients ineligible for or not responsive to IV tPA.

The anesthesiologist's primary goals are to avoid intervention delay, maintain hypertension, and prevent patient movement.[58] Preferential use of hemodynamically stable anesthetics (such as etomidate rather than propofol) at anesthetic induction and maintenance of normocapnia are generally advised.[17] The choice of GA versus sedation in endovascular treatment of acute ischemic stroke remains a matter of significant debate. Retrospective analyses suggest higher risk of early mortality and other complications after use of GA.[59–61] Prospective trial data are not available. If GA is injurious, hypoperfusion, time delay, and initiating a critical care

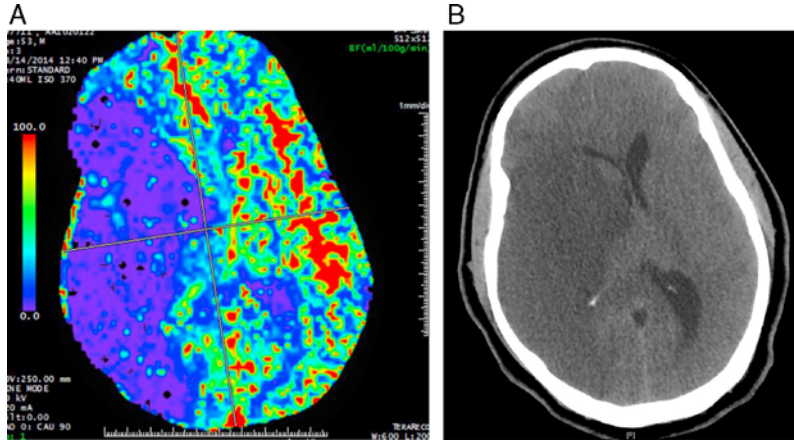

Figure 3. *A, CT perfusion study illustrating a severe perfusion (blood flow) deficit in a right MCA stroke. B, Noncontrast CT image of the same acute right MCA stroke.* full color online

treatment pathway are all hypothesized as potential contributors. Concern for time delay has been reported as a general concern among interventionalists,[62] and retrospective data in multiple studies correlated hypotension (SBP < 130 to 150 mm Hg) with poor outcome.[63–65] In choosing an anesthetic technique, surgeon preference, patient comorbidities, neurological status, and expedience should be considered. GA is preferable when neurological deficits predispose to sedation intolerance, such as bulbar dysfunction causing increased risk of aspiration or loss of airway patency. Sedation may be preferable in the case of milder deficits or tenuous hemodynamic status.[17] Discussion among anesthesia and neurointerventional care providers should be sought to facilitate institution-specific protocolized management of these emergent cases. Recommendations[58] include avoiding time delay, hypoperfusion (SBP target, 140 to 180 mm Hg), hyperthermia (target T, 35 to 37°C), hypocapnia (PCO_2 target, 35 to 40 mm Hg), and hyperglycemia (blood glucose target, 70 to 150).

Intracranial Stenting Results of long-term follow-up of the Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis trial suggest that aggressive medical management (statin therapy, blood pressure control, combined aspirin and clopidogrel for 90 d followed by aspirin alone, and lifestyle modifications with active coaching) of patients with recent TIA or stroke due to intracranial atherosclerotic disease is superior to percutaneous transluminal angioplasty and stent placement (PTAS) (see Fig. 4).[3] Therefore, PTAS is reserved for treatment of patients with symptomatic intracranial atherosclerosis refractory to medical management.

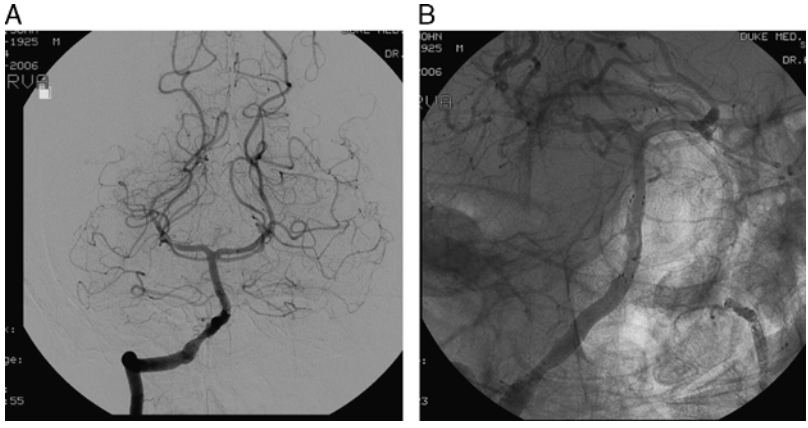

Figure 4. *Stenotic vertebral artery before (A) and after (B) endovascular stent placement.*

This high-risk population provides significant anesthetic challenges in the IR suite. The primary concern is to prevent cerebral ischemia due to hypoperfusion. Blood pressure should be kept at preinduction values. Balloon inflation during angioplasty is commonly associated with bradycardia, which may devolve into asystole in certain individuals. Balloon angioplasty can also be painful and may require transiently increased analgesia or anesthetic depth. Potential procedural complications include distal thromboembolism, vessel perforation, dissection, spasm, TIA, stroke, or hemorrhage from vessel rupture. GA is preferred in cases of basilar artery PTAS due to the risk of loss of consciousness and apnea during balloon inflation in this vascular territory. Deliberate hypertension may be useful to avoid cerebral hypoperfusion during balloon inflation and stent deployment but should be used judiciously as it may also predispose to hemorrhage.[18]

Emergent Therapy for Cerebral Vasospasm The incidence of aneurysmal subarachnoid hemorrhage is estimated to be 14.5 per 100,000 adults with rates relatively stable since the 1970s (see Fig. 5).[66,67] At least 25% of patients with aneurysmal subarachnoid hemorrhage will develop vasospasm, which commonly leads to delayed cerebral ischemia. Endovascular treatment of cerebral vasospasm is high risk, and induced hypertension (ie, hypertensive euvolemia) remains the first-line therapy.[68] For patients refractory to medical therapy, urgent endovascular intervention is performed and may often require more than 1 procedure. Endovascular treatment consists of balloon angioplasty or intra-arterial vasodilator injection with nicardipine, papaverine, nitroglycerin, or verapamil.[69] Angioplasty may result in more sustained effect but carries risk of vessel rupture and can only be utilized in larger more proximal cerebral vessels.[18]

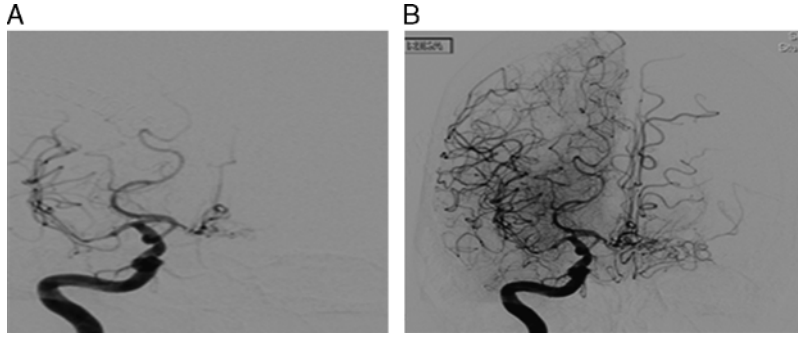

Figure 5. *Acute vasospasm. Early (A) and later (B) phase angiography after a right internal carotid artery contrast injection in a 46-year-old patient 7 days after aneurysmal subarachnoid hemorrhage.*

Anesthetic management should focus on maintaining hypertension at preoperative levels (SBP usually in the 160 to 200 mm Hg range unless the patient's aneurysm remains unsecured). In fact, induced hypotension may result in early and delayed neurological deficits.[70,71] Many patients are on vasopressor infusions that will need to be continued throughout the procedure. Transient decreases in blood pressure after intra-arterial vasodilator therapy should be anticipated.

Carotid Stenting Although carotid endarterectomy (CEA) has historically been the standard of care for patients with symptomatic carotid stenosis, carotid arterial stenting (CAS) has now become widely utilized. The CREST trial randomized over 2500 patients with symptomatic or asymptomatic stenosis to CEA or CAS and found no difference in the primary endpoint (a composite of stroke, myocardial infarction, and death) over a median follow-up of 2.5 years.[72] Interestingly, CAS patients did suffer more strokes, whereas CEA patients suffered more myocardial infarctions. Carotid stenting is typically accomplished by a femoral artery approach, and cerebral thromboemboli and vessel dissection are the predominant risks in CAS, despite the use of distal filters.[73] Aspirin and clopidogrel premedication are commonly administered, along with intraprocedural heparin to minimize distal embolization.

Most cases can be safely completed under mild to moderate sedation.[74] Blood pressure management with vasoactive drugs may be required to assure normotension and prevent cerebral hypoperfusion from hypotension or increased risk of migration of distal microthrombi with hypertension. Significant hypotension is associated with neurological complications; mild hypotension is usually well tolerated.[19,75] Profound or symptomatic bradycardia during stent deployment may be treated with glycopyrrolate or atropine.

■ Vessel Closure Device Review for the Anesthesiologist

The vast majority of neurovascular procedures are performed via access of the femoral artery. It is preferred mainly due to its large diameter. Most cases are performed through 5- or 6-Fr sheaths. At the conclusion of the procedure, the sheath is removed and hemostasis is obtained. This was initially accomplished by manually applying pressure to the groin puncture site, followed by maintaining supine position for 2 to 4 hours. This method, known as the Seldinger technique, was developed by Dr Sven Seldinger, a Swedish radiologist in the 1950s.

Arterial closure devices, first introduced in 1994, rapidly obtain hemostasis by mechanically closing the arterotomy, allowing patients to ambulate sooner after their procedure. These devices function by one of 3 fundamental mechanisms: suture-mediated, vascular clip, and collagen plug. Suture-mediated closure devices deliver a stitch down the tract of the sheath. Metal clips close the arterotomy on the external aspect of the artery and collagen plugs are delivered onto the extravascular surface and typically dissolve over 60 to 90 days. Although no double-blind, randomized, controlled clinical trials have been performed on arterial closure devices, observational studies have demonstrated complication rates of these devices between 1.2% and 2.9%.[76] However, manual compression remains the gold standard and is the method used when an arterial closure device fails.

■ Conclusions

Endovascular therapeutics for cerebral and spinal disease continue to expand and evolve. The anesthesiologist must be prepared to handle these cases on elective and emergent bases. Solid understanding of the cerebrovascular disease, urgency, and potential for injury from hypotension or hypertension is essential for the optimal management of this patient population. Much of the care is subjective and/or institution specific. Optimal care mandates open communication between anesthesia and interventional teams regarding blood pressure goals, anticoagulants, case urgency, procedural variables, and complications. Finally, evidence regarding the impact of anesthetic type on outcome is growing and must be incorporated into treatment plans.

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■ References

1. Andaluz N, Zuccarello M. Recent trends in the treatment of cerebral aneurysms: analysis of a nationwide inpatient database. *J Neurosurg*. 2008;108:1163–1169.
2. Ogilvy CS. Neurosurgical clipping versus endovascular coiling of patients with ruptured intracranial aneurysms. *Stroke*. 2003;34:2540–2542.
3. Chimowitz MI, Lynn MJ, Derdeyn CP, et al. Stenting versus aggressive medical therapy for intracranial arterial stenosis. *N Engl J Med*. 2011;365:993–1003.
4. Mohr JP, Parides MK, Stapf C, et al. Medical management with or without interventional therapy for unruptured brain arteriovenous malformations (ARUBA): a multicentre, non-blinded, randomised trial. *Lancet*. 2014;383:614–621.
5. Derdeyn CP, Chimowitz MI, Lynn MJ, et al. Aggressive medical treatment with or without stenting in high-risk patients with intracranial artery stenosis (SAMMPRIS): the final results of a randomised trial. *Lancet*. 2014;383:333–341.
6. Young WL. Anesthesia for endovascular neurosurgery and interventional neuroradiology. *Anesthesiol Clin*. 2007;25:391–412; vii.
7. Varma MK, Price K, Jayakrishnan V, et al. Anaesthetic considerations for interventional neuroradiology. *Br J Anaesth*. 2007;99:75–85.
8. Thal GD, Szabo MD, Lopez-Bresnahan M, et al. Exacerbation or unmasking of focal neurologic deficits by sedatives. *Anesthesiology*. 1996;85:21–25; discussion 29A–30A.
9. Lazar RM, Fitzsimmons BF, Marshall RS, et al. Midazolam challenge reinduces neurological deficits after transient ischemic attack. *Stroke*. 2003;34:794–796.
10. Otite F, Mink S, Tan CO, et al. Impaired cerebral autoregulation is associated with vasospasm and delayed cerebral ischemia in subarachnoid hemorrhage. *Stroke*. 2014;45:677–682.
11. Strandgaard S. Autoregulation of cerebral blood flow in hypertensive patients. The modifying influence of prolonged antihypertensive treatment on the tolerance to acute, drug-induced hypotension. *Circulation*. 1976;53:720–727.
12. Wakhloo AK, Linfante I, Silva CF, et al. Closed-cell stent for coil embolization of intracranial aneurysms: clinical and angiographic results. *AJNR Am J Neuroradiol*. 2012;33:1651–1656.
13. Price MJ, Berger PB, Teirstein PS, et al. Standard- vs high-dose clopidogrel based on platelet function testing after percutaneous coronary intervention: the GRAVITAS randomized trial. *JAMA*. 2011;305:1097–1105.
14. Castagnini HE, van Eijs F, Salevsky FC, et al. Sevoflurane for interventional neuroradiology procedures is associated with more rapid early recovery than propofol. *Can J Anaesth*. 2004;51:486–491.
15. Kapila A, Glass PS, Jacobs JR, et al. Measured context-sensitive half-times of remifentanil and alfentanil. *Anesthesiology*. 1995;83:968–975.
16. Pajewski TN, Arlet V, Phillips LH. Current approach on spinal cord monitoring: the point of view of the neurologist, the anesthesiologist and the spine surgeon. *Eur Spine J*. 2007;16(suppl 2):S115–S129.
17. Froehler MT, Fifi JT, Majid A, et al. Anesthesia for endovascular treatment of acute ischemic stroke. *Neurology*. 2012;79(suppl 1):S167–S173.
18. Lee CZ, Young WL. Anesthesia for endovascular neurosurgery and interventional neuroradiology. *Anesthesiol Clin*. 2012;30:127–147.
19. Howell M, Krajcer Z, Dougherty K, et al. Correlation of periprocedural systolic blood pressure changes with neurological events in high-risk carotid stent patients. *J Endovasc Ther*. 2002;9:810–816.
20. Szabo MD, Crosby G, Sundaram P, et al. Hypertension does not cause spontaneous hemorrhage of intracranial arteriovenous malformations. *Anesthesiology*. 1989;70:761–763.
21. Spiess BD. Treating heparin resistance with antithrombin or fresh frozen plasma. *Ann Thorac Surg*. 2008;85:2153–2160.

22. Keeling D, Davidson S, Watson H. The management of heparin-induced thrombocytopenia. *Br J Haematol*. 2006;133:259–269.
23. Linkins LA, Dans AL, Moores LK, et al. Treatment and prevention of heparin-induced thrombocytopenia: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest*. 2012;141(suppl):e495S–e4530.
24. Tcheng JE. Platelet integrin glycoprotein IIb/IIIa inhibitors: opportunities and challenges. *J Invasive Cardiol*. 1996;8(suppl B):8B–14B.
25. Fiorella D, Albuquerque FC, Han P, et al. Strategies for the management of intraprocedural thromboembolic complications with abciximab (ReoPro). *Neurosurgery*. 2004;54:1089–1097; discussion 1097–1088.
26. Aggour M, Pierot L, Kadziolka K, et al. Abciximab treatment modalities for thromboembolic events related to aneurysm coiling. *Neurosurgery*. 2010;67:503–508.
27. Schulman S, Bijsterveld NR. Anticoagulants and their reversal. *Transfus Med Rev*. 2007;21:37–48.
28. Gordon JL, Fabian TC, Lee MD, et al. Anticoagulant and antiplatelet medications encountered in emergency surgery patients: a review of reversal strategies. *J Trauma Acute Care Surg*. 2013;75:475–486.
29. Goldstein JN, Marrero M, Masrur S, et al. Management of thrombolysis-associated symptomatic intracerebral hemorrhage. *Arch Neurol*. 2010;67:965–969.
30. Levine M, Goldstein JN. Emergency reversal of anticoagulation: novel agents. *Curr Neurol Neurosci Rep*. 2014;14:471.
31. Administration OSaH. Ionizing Radiation.1910.1096. 39 FR 23502, June 27, 1974, as amended at 43 FR 49746, October 24, 1978; 43 FR 51759, November 7, 1978; 49 FR 18295, April 30, 1984; 58 FR 35309, June 30, 1993; 61 FR 5507, February 13, 1996; 61 FR 31427, June 20, 1996.
32. Anastasian ZH, Strozyk D, Meyers PM, et al. Radiation exposure of the anesthesiologist in the neurointerventional suite. *Anesthesiology*. 2011;114:512–520.
33. Brothers MF, Holgate RC. Intracranial angioplasty for treatment of vasospasm after subarachnoid hemorrhage: technique and modifications to improve branch access. *AJNR Am J Neuroradiol*. 1990;11:239–247.
34. Hahnel S, Schellinger PD, Gutschalk A, et al. Local intra-arterial fibrinolysis of thromboemboli occurring during neuroendovascular procedures with recombinant tissue plasminogen activator. *Stroke*. 2003;34:1723–1728.
35. Molyneux AJ, Kerr RS, Birks J, et al. Risk of recurrent subarachnoid haemorrhage, death, or dependence and standardised mortality ratios after clipping or coiling of an intracranial aneurysm in the International Subarachnoid Aneurysm Trial (ISAT): long-term follow-up. *Lancet Neurol*. 2009;8:427–433.
36. Fiorella D, Albuquerque FC, Han P, et al. Preliminary experience using the Neuroform stent for the treatment of cerebral aneurysms. *Neurosurgery*. 2004;54:6–16; discussion 16–17.
37. Berger M, Philips-Bute B, Guercio J, et al. A novel application for bolus remifentanil: blunting the hemodynamic response to Mayfield skull clamp placement. *Curr Med Res Opin*. 2014;30:243–250.
38. Scheller MS, Tateishi A, Drummond JC, et al. The effects of sevoflurane on cerebral blood flow, cerebral metabolic rate for oxygen, intracranial pressure, and the electroencephalogram are similar to those of isoflurane in the rabbit. *Anesthesiology*. 1988;68:548–551.
39. Scheller MS, Nakakimura K, Fleischer JE, et al. Cerebral effects of sevoflurane in the dog: comparison with isoflurane and enflurane. *Br J Anaesth*. 1990;65:388–392.
40. Ogilvy CS, Yang X, Jamil OA, et al. Neurointerventional procedures for unruptured intracranial aneurysms under procedural sedation and local anesthesia: a large-volume, single-center experience. *J Neurosurg*. 2011;114:120–128.

41. Haley EC Jr, Kassell NF, Torner JC. A randomized controlled trial of high-dose intravenous nicardipine in aneurysmal subarachnoid hemorrhage. A report of the Cooperative Aneurysm Study. *J Neurosurg*. 1993;78:537–547.
42. Wittstein IS, Thiemann DR, Lima JA, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. *N Engl J Med*. 2005;352:539–548.
43. Davison DL, Terek M, Chawla LS. Neurogenic pulmonary edema. *Crit Care*. 2012;16:212.
44. Wartenberg KE, Mayer SA. Medical complications after subarachnoid hemorrhage. *Neurosurg Clin N Am*. 2010;21:325–338.
45. Solenski NJ, Haley EC Jr, Kassell NF, et al. Medical complications of aneurysmal subarachnoid hemorrhage: a report of the multicenter, cooperative aneurysm study. Participants of the Multicenter Cooperative Aneurysm Study. *Crit Care Med*. 1995;23:1007–1017.
46. Meyers PM, Schumacher HC, Higashida RT, et al. Indications for the performance of intracranial endovascular neurointerventional procedures: a scientific statement from the American Heart Association Council on Cardiovascular Radiology and Intervention, Stroke Council, Council on Cardiovascular Surgery and Anesthesia, Interdisciplinary Council on Peripheral Vascular Disease, and Interdisciplinary Council on Quality of Care and Outcomes Research. *Circulation*. 2009;119:2235–2249.
47. Ledezma CJ, Hoh BL, Carter BS, et al. Complications of cerebral arteriovenous malformation embolization: multivariate analysis of predictive factors. *Neurosurgery*. 2006;58:602–611; discussion 602–611.
48. Ogilvy CS, Stieg PE, Awad I, et al. AHA Scientific Statement: Recommendations for the management of intracranial arteriovenous malformations: a statement for healthcare professionals from a special writing group of the Stroke Council, American Stroke Association. *Stroke*. 2001;32:1458–1471.
49. Kim LJ, Albuquerque FC, Spetzler RF, et al. Postembolization neurological deficits in cerebral arteriovenous malformations: stratification by arteriovenous malformation grade. *Neurosurgery*. 2006;59:53–59; discussion 53–59.
50. Spetzler RF, Wilson CB, Weinstein P, et al. Normal perfusion pressure breakthrough theory. *Clin Neurosurg*. 1978;25:651–672.
51. Reeves MJ, Arora S, Broderick JP, et al. Acute stroke care in the US: results from 4 pilot prototypes of the Paul Coverdell National Acute Stroke Registry. *Stroke*. 2005;36:1232–1240.
52. Rha JH, Saver JL. The impact of recanalization on ischemic stroke outcome: a meta-analysis. *Stroke*. 2007;38:967–973.
53. Smith WS, Sung G, Starkman S, et al. Safety and efficacy of mechanical embolectomy in acute ischemic stroke: results of the MERCI trial. *Stroke*. 2005;36:1432–1438.
54. Penumbra Clinical Stroke Trial Investigators. The penumbra pivotal stroke trial: safety and effectiveness of a new generation of mechanical devices for clot removal in intracranial large vessel occlusive disease. *Stroke*. 2009;40:2761–2768.
55. Deshaies EM, Singla A, Villwock MR, et al. Early experience with stent retrievers and comparison with previous-generation mechanical thrombectomy devices for acute ischemic stroke. *J Neurosurg*. 2014;121:12–17.
56. Nogueira RG, Lutsep HL, Gupta R, et al. Trevo versus Merci retrievers for thrombectomy revascularisation of large vessel occlusions in acute ischaemic stroke (TREVO 2): a randomised trial. *Lancet*. 2012;380:1231–1240.
57. Broderick JP, Palesch YY, Demchuk AM, et al. Endovascular therapy after intravenous t-PA versus t-PA alone for stroke. *N Engl J Med*. 2013;368:893–903.
58. Talke PO, Sharma D, Heyer EJ, et al. Society for Neuroscience in Anesthesiology and Critical Care Expert consensus statement: anesthetic management of endovascular treatment for acute ischemic stroke*: endorsed by the Society of NeuroInterventional Surgery and the Neurocritical Care Society. *J Neurosurg Anesthesiol*. 2014;26:95–108.

59. Nichols C, Carrozzella J, Yeatts S, et al. Is periprocedural sedation during acute stroke therapy associated with poorer functional outcomes? *J Neurointerv Surg*. 2010;2:67–70.
60. Jumaa MA, Zhang F, Ruiz-Ares G, et al. Comparison of safety and clinical and radiographic outcomes in endovascular acute stroke therapy for proximal middle cerebral artery occlusion with intubation and general anesthesia versus the nonintubated state. *Stroke*. 2010;41:1180–1184.
61. Abou-Chebl A, Lin R, Hussain MS, et al. Conscious sedation versus general anesthesia during endovascular therapy for acute anterior circulation stroke: preliminary results from a retrospective, multicenter study. *Stroke*. 2010;41:1175–1179.
62. McDonagh DL, Olson DM, Kalia JS, et al. Anesthesia and sedation practices among neurointerventionalists during acute ischemic stroke endovascular therapy. *Front Neurol*. 2010;1:118.
63. Leonardi-Bee J, Bath PM, Phillips SJ, et al. Blood pressure and clinical outcomes in the International Stroke Trial. *Stroke*. 2002;33:1315–1320.
64. Vemmos KN, Tsivgoulis G, Spengos K, et al. U-shaped relationship between mortality and admission blood pressure in patients with acute stroke. *J Int Med*. 2004;255:257–265.
65. Okumura K, Ohya Y, Maehara A, et al. Effects of blood pressure levels on case fatality after acute stroke. *J Hypertens*. 2005;23:1217–1223.
66. Shea AM, Reed SD, Curtis LH, et al. Characteristics of nontraumatic subarachnoid hemorrhage in the United States in 2003. *Neurosurgery*. 2007;61:1131–1137; discussion 1137–1138.
67. Feigin VL, Lawes CM, Bennett DA, et al. Worldwide stroke incidence and early case fatality reported in 56 population-based studies: a systematic review. *Lancet Neurol*. 2009;8:355–369.
68. Egge A, Waterloo K, Sjoholm H, et al. Prophylactic hyperdynamic postoperative fluid therapy after aneurysmal subarachnoid hemorrhage: a clinical, prospective, randomized, controlled study. *Neurosurgery*. 2001;49:593–605; discussion 605–596.
69. Newell DW, Eskridge JM, Mayberg MR, et al. Angioplasty for the treatment of symptomatic vasospasm following subarachnoid hemorrhage. *J Neurosurg*. 1989;71(pt 1): 654–660.
70. Farrar JK, Gamache FW Jr, Ferguson GG, et al. Effects of profound hypotension on cerebral blood flow during surgery for intracranial aneurysms. *J Neurosurg*. 1981;55:857–864.
71. Hitchcock ER, Tsementzis SA, Dow AA. Short- and long-term prognosis of patients with a subarachnoid haemorrhage in relation to intra-operative period of hypotension. *Acta Neurochir*. 1984;70:235–242.
72. Brott TG, Hobson RW II, Howard G, et al. Stenting versus endarterectomy for treatment of carotid-artery stenosis. *N Engl J Med*. 2010;363:11–23.
73. Tedesco MM, Lee JT, Dalman RL, et al. Postprocedural microembolic events following carotid surgery and carotid angioplasty and stenting. *J Vasc Surg*. 2007;46:244–250.
74. Steib A, Collange O. Anesthesia for other endovascular stenting. *Curr Opin Anaesthesiol*. 2008;21:519–522.
75. Dangas G, Laird JR Jr, Satler LF, et al. Postprocedural hypotension after carotid artery stent placement: predictors and short- and long-term clinical outcomes. *Radiology*. 2000;215:677–683.
76. Applegate RJ, Sacrinty MT, Kutcher MA, et al. Propensity score analysis of vascular complications after diagnostic cardiac catheterization and percutaneous coronary intervention 1998-2003. *Catheter Cardiovasc Interv*. 2006;67:556–562.