Interventional Neuroradiology

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22.1 Introduction

Interventional neuroradiology (INR) has remarkably evolved over recent years and serves as an alternative to many open neurovascular procedures. Endovascular therapy is less invasive and allows for some interventions that simply cannot be performed with open surgery, making it a primary approach for treating a host of thrombo-occlusive and hemorrhagic neurovascular conditions. To that end, INR also poses many challenges for the anesthesiologist from caring for fragile or critically ill patients in an off-site location to having to navigate through various physical obstacles within the interventional suite.

This chapter will (1) explore the challenges met with working in an angiography suite, (2) discuss a basic approach to the anesthetic regimen and management of common INR procedures, (3) review the uses of endovascular therapy in the treatment of various neurological conditions (both elective and emergent), and (4) discuss some of the common complications encountered in INR.

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22.2 Angiography Suite Considerations

The angiography suite itself can be an extreme test for the anesthesiologist. Depending on the institution, these suites are often located off-site with limited accessibility to anesthesia personnel and resources, which can be a particular challenge in emergent situations. A biplanar fluoroscope with two juxtaposed C-arms for anterior-posterior and lateral images occupies a significant amount of room centrally within the suite. All personnel and equipment are situated circumferentially around the fluoroscopy unit, decreasing accessibility to the patient and creating physical obstacles to obtain equipment in a timely manner. Also, the presence of digital imaging screens poses a barrier to communication as it is often located directly between the interventionalist and the anesthesiologist (Figs. 22.1 and 22.2).

The patient is situated such that the head is often positioned in the far end of the room between the two C-arms which must have enough unobstructed room to allow rotational movement. This essentially pushes the anesthesia machine to the distal end of the patient, allowing access to just the lower half of their body. More importantly, this also limits access to the patient's airway and torso both during intubation and during the procedure. The INR table itself poses additional challenges. While it can be maneuvered into Trendelenburg and reverse

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Fig. 22.1 Angiography suite from the side of the interventionalist

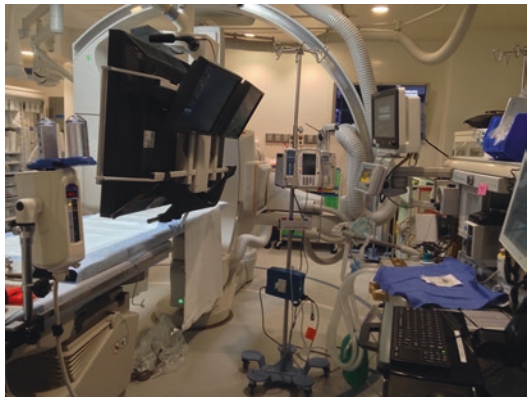

Fig. 22.2 Angiography suite from the side of the anesthesiologist

Trendelenburg positions, more nuanced positions such as head up cannot be obtained. This may make it difficult to place the patient into an optimal sniffing position for intubation. The table also does not have built-in arm boards. Boards such as these (Fig. 22.3) can be used for arterial line placement but must be removed and the arms must be tucked to allow the C-arms necessary access to the patient. Finally, the bed is firmer than a conventional operating room table, which can pose a challenge in keeping a patient comfortable for any cases done under sedation/monitored anesthesia care.

It is for these considerations that precise planning is required. Extension tubing for the circuit and end-tidal gas sampling line is often needed to allow for the added distance between the patient and the anesthesia machine. Care should

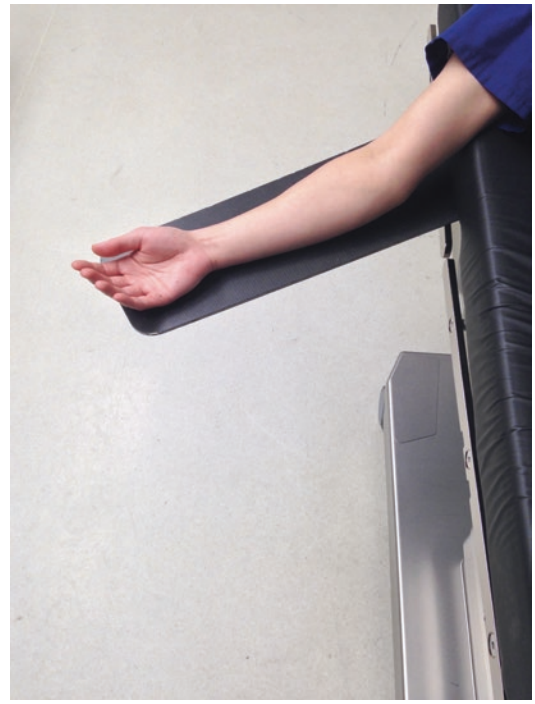

Fig. 22.3 An arm board can be used for arterial line placement; however it must be removed before the procedure begins

be taken to ensure that the circuits, monitor cables, and IV tubing are not hanging in the path of the rotational C-arms. Also, as there is often significant distance between the patient's IV site and the anesthesiologist, IV extension tubing should be utilized with consideration given to the increase in dead space when initiating any vasoactive or anticoagulant infusion therapy [1]. Finally, radiation exposure necessitates the utilization of lead garments and leaded shields and complicates navigating an already restricted environment.

22.3 Radiation Safety

In the neurointerventional suite, radiation exposure is a significant occupational hazard. The four factors that affect occupational radiation exposure are the amount of radiation used, duration of exposure, shielding from the radiation, and distance from the source. Embolizations of cerebral aneurysms and arteriovenous malformations are considered high-dose radiation

procedures due to the interventional technique of digital subtraction angiography requiring more ionizing radiation than standard fluoroscopy. The two controllable methods of protection from radiation for anesthesiologists are appropriate shielding and increased distance from the source. Appropriate personal shielding includes radiation-protection aprons, thyroid collars, and radiation-protection eyewear. Lead-plated glass/plexiglass shields can also be positioned to reduce direct exposure to the fluoroscopy equipment. The concept of increased distance from the radiation source stems from the inverse-square law: radiation concentration is inversely proportional to the square of the distance from the radiation source [2].

Radiation exposure can lead to acute and chronic effects. Directed acute effects can cause skin erythema and dermatitis. Generalized whole-body radiation exposure can cause nausea, vomiting, diarrhea, weakness, and possibly death. Chronic effects can lead to skin cancer, bone marrow suppression, and reproductive issues. Dosimeters should be utilized routinely, and exposure to the anesthesiologist should be kept lower than the annual limit for healthcare workers set by the United States Department of Labor's Occupational Safety and Health Administration [3].

22.4 INR Procedure

INR procedures routinely begin by obtaining arterial femoral sheath access, most often with a 6 or 7 French catheter [4]. Alternative access can also be obtained via the radial, brachial, or carotid arteries [4–6]. Through the femoral sheath, a smaller coaxial guide catheter is introduced and advanced to the common carotid artery. This is followed by a diagnostic cerebral angiogram, where contrast medium is bolused to outline the cerebral vasculature. The radiological imaging is produced via high-resolution fluoroscopy and high-speed digital subtraction angiography (DSA) with a "roadmapping" feature (Fig. 22.4a) [4, 7]. While performing live fluoroscopy, a computer will superimpose these live images on those acquired in the "roadmapping" process (inversion of the black vessels on angiography to make them white) to allow visualization of the progress of the radiopaque catheter tip into the target vessel [4, 7].

If the visualized neurovascular lesion is deemed treatable by an interventional route, a microcatheter is advanced into the cerebral vasculature and navigated to the neurovascular lesion. The lesion is then treated with detachable coils, stents, or embolization agents as deemed necessary by the interventionalist (Fig. 22.4b).

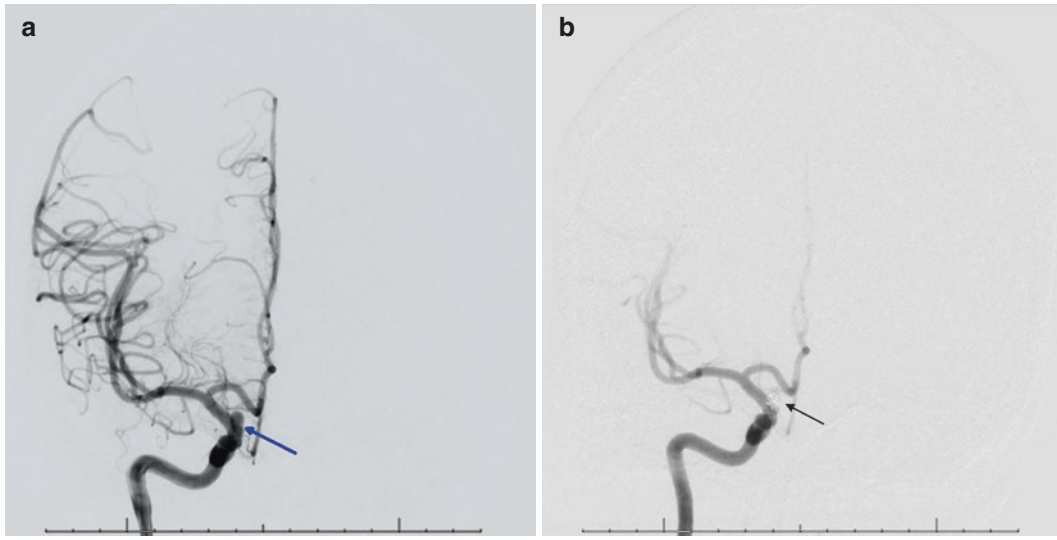

Fig. 22.4 Coil embolization of a type II right ophthalmic artery aneurysm. (**a**) Pre-procedure diagnostic angiography obtained using digital subtraction angiography (DSA). (**b**) Post-procedure DSA angiography performed after successful coil embolization of the aneurysm. Arrow denotes the coil

After the procedure is complete, the interventionalist will obtain hemostasis and may insert an arterial closure device to prevent post-procedural bleeding from the arteriotomy. This process is variable and may require several minutes of direct pressure to the groin. Closure devices work through various mechanisms that may include a disc deployed on the intravascular side of the arteriotomy, a collagen plug or polyethylene glycol sealant applied on the extravascular side of the arteriotomy, a clip, sutures, or some combination of the abovementioned components [8]. Many of these techniques will require the patient to lay flat with the ipsilateral leg to the arteriotomy extended at the hip for a certain period of time, up to several hours. It is important that the anesthesiologist and interventionalist communicate prior to emergence regarding the needs for each patient.

22.5 Anesthetic Management

The anesthesiologist's involvement in INR cases is vital for a number of reasons. (1) The anesthetic can provide partial or complete immobility, which is essential while the interventionalist navigates through delicate cerebrovascular structures. (2) Since the majority of INR procedures involve manipulation of the cerebral vasculature, precise hemodynamic monitoring and control are critical in order to complement the intervention being performed. (3) A rapid emergence is essential to allow for neurological monitoring in the post-procedural period, which is largely determined by the anesthesiologist's choice of drugs.

22.5.1 Lines and Monitors

Neurovascular disease can often call for extremes in the patient's blood pressure parameters. Generally speaking, hemorrhagic disease calls for deliberate hypotension or controlled normotension, while thrombo-occlusive or spastic disease requires deliberate hypertension. Moreover, the anesthesiologist may need to manage intracranial pressure (ICP) and cerebral perfusion pressure (CPP) when a ventriculostomy or other ICP monitor is in place. Communication regarding hemodynamic targets is of the utmost importance between the anesthesiologist and the interventionalist.

Intra-arterial blood pressure monitoring allows for precise measurement of the arterial pressure during the procedure as well as for post-procedural care in the intensive care unit. For the critically ill, placement of the arterial line should be considered prior to induction (such as for the aneurysmal subarachnoid hemorrhage patient). Note that certain femoral sheath catheters possess a side port which allows a line to be added ("piggy-backed") for blood pressure monitoring, although these ports often provide dampened waveforms. Also, once the sheath is removed, monitoring will not be available for emergence and post-procedural care.

The necessity of vasoactive infusions and the risk of blood loss, should a complication occur, make a second IV line useful in many cases. In the nondiabetic patient, the legs (saphenous vein) and feet are a convenient location as they are easily accessible to the anesthesiologist during the procedure. Central venous access is usually not needed unless the patient is critically ill, such as the aneurysmal SAH patient with vasospasm.

Urinary catheters are usually deemed necessary to monitor the increased urine output that can develop from the contrast-induced diuresis. This also helps the anesthesiologist determine volume status and maintain adequate hydration to help prevent nephropathy induced by contrast medium [9].

22.5.2 Type of Anesthesia

For diagnostic cerebral arteriograms, conscious sedation is typically the preferred approach as this procedure simply entails imaging the cerebral vasculature, assuming the patient is cooperative enough for the procedure. However, for more complicated interventional procedures involving coiling, embolization, stenting, or thrombolysis/clot retrieval, patient movement in the angiography suite can be potentially disastrous, especially

if the motion comes at a critical portion of the case. This can lead to impaired radiographic imaging, perforation of a cerebral vessel, or drastic elevations in intracranial pressure. As a result, immobility is usually preferred [10]. The choice between conscious sedation (CS)/monitored anesthesia care (MAC) and general endotracheal anesthesia (GETA) is often left to the discretion of the anesthesiologist and interventionalist.

CS/MAC offers the option of intra-procedural monitoring of the clinical neurological exam [11], which is preferable in a carotid stenting procedure for example. However, this comes at the expense of potential patient movement, increased procedural difficulty, and motion artifact. Moreover, CS/MAC may be preferable in those presenting with other significant comorbidities which would make them less amenable to GETA [12]. Relieving an obstructed airway with jaw thrust/chin lift can be extremely challenging and can impair the fluoroscopic views as the patient's head/airway is away from the anesthesiologist and in the field of radiation. GETA is a preferable alternative as it can provide complete immobility, especially in lengthy procedures or in patients with an inability to lay flat. It can also be of value in that it provides a protected airway and gives the ability to hyperventilate a patient to reduce ICP or, alternately, hypoventilate to dilate the cerebral vasculature in appropriate scenarios. Moreover, GETA provides more flexibility for the anesthesiologist to focus on other matters such as hemodynamic monitoring during the procedure. On the other hand, being under GETA during an INR procedure, which is minimally stimulating, can lead to hemodynamic lability with drops in blood pressure and/or heart rate, requiring titration of the depth of anesthesia and the addition of vasopressor infusions.

22.5.3 Anesthetic Medications

Induction Induction can be a very labile period, especially in critically ill neurological patients who need to stay within a range of specific hemodynamic parameters. Performing a smooth and controlled induction can be a challenge. Pre-induction arterial access to allow invasive blood pressure monitoring can be of value, although obtaining such access can be a challenge in the setting of time-sensitive conditions such as acute ischemic stroke. Patients typically need to maintain blood pressure with some degree of induced hypertension in conditions such as ischemic stroke and cerebral vasospasm. The use of hemodynamically stable agents such as etomidate and a pre-induction fluid bolus can help reduce lability. The induction in those with hemorrhagic lesions, who need minimal surges in blood pressure, can be accomplished with agents that achieve a deep level of anesthesia in a short period of time such as propofol and remifentanil.

Maintenance A rapid, clear emergence and wake-up are crucial to allow for an adequate neurological examination post-intervention. Intracerebral bleeding or thrombotic complications as a result of INR procedures can be detected by neurological examination and could necessitate, depending on the situation, an emergent head CT, anticoagulation, reversal of anticoagulation, surgical hematoma evacuation, or endovascular thrombectomy. Thus, using short-acting anesthetic agents is vital to allow for more effective surveillance for these complications (Table 22.1). Time is critical in these situations, and the use of longer-acting agents can delay their recognition. Nitrous oxide should be avoided in neurointerventional procedures since this agent will enlarge any gaseous/air microemboli that may have complicated the interventional procedure. If an air embolus does complicate the procedure, hyperoxia is the treatment (1.0 FiO_2 then emergent transfer to a hyperbaric treatment facility) [13].

Most INR procedures do not provide a great deal of intraoperative stimulation or postoperative pain. Manipulation of cerebral vessels can be painful for an awake patient but does not provoke much of a response under general anesthesia. A typical approach is 0.6 MAC (minimum alveolar concentration) halogenated inhalational agent with a remifentanil infusion

Table 22.1 Select anesthetic drugs in the INR suite

Anesthetic drug (drug class, dose range)	Advantage for INR	Disadvantage for INR	Acceptable substitutes	Other aspects
Desflurane (volatile anesthetic, ~0.6 MAC)	Rapid titration and emergence	Potential for airway irritation leading to coughing; Theoretical risk of cerebral steal in certain disease states (such as moyamoya)	Sevoflurane Isoflurane	Avoid nitrous oxide (due to potential for intra-arterial air emboli)
Remifentanil (opioid, 0.1–0.3 mcg/kg/min)	Deep plane of anesthesia, smooth emergence No context sensitivity	Potential for hypertension and tachycardia after emergence	Sufentanil Fentanyl	Bolus dosing effective to blunt the response to intubation (1–2 mcg/kg)
Dexmedetomidine (α-2 agonist, 0.2–0.7 mcg/kg/h)	Sedation and analgesia for procedures	Potential for hypotension, bradycardia, somnolence	Low-dose clonidine	Prevents emergence agitation and hypertension
Midazolam (benzodiazepine, 0.5–5 mg)	Anxiolysis; sedation for procedures	Potential for somnolence, alteration of neurological exam	Propofol bolus or infusion	
Propofol (intravenous anesthetic, 25–200 mcg/kg/min)	Sedation for procedures	Potential for hypotension; loss of anesthetic depth (or awareness) due to IV infiltration		Does not exacerbate ICP; should not cause "cerebrovascular steal"

Table 22.2 Vasoactive medications useful in the INR suite

Pressors/inotropes (drug class, dose range)	Antihypertensives (drug class, dose range)
Phenylephrine (α-1 agonist, 0.1–1 mcg/kg/min)	Nicardipine (calcium channel blocker, 5–15 mg/h)
Ephedrine (indirect sympathomimetic, 5–10 mg IV bolus)	Clevidipine (calcium channel blocker, 4–6 mg/h)
Epinephrine (dose-dependent α and β agonist, 0.01–0.5 mcg/kg/min)	Labetalol (α-1 and β-1,2 blocker, 5–20 mg IV bolus Q 5 min)
Norepinephrine (α-1 and β-1 agonist, 0.02–1 mcg/kg/min)	Esmolol (short-acting β-1 blocker, 10–30 mg IV bolus)
Vasopressin (V1 and V2 receptor agonist, 0.01–0.04 units/min IV)	Hydralazine (vasodilator, 5–10 mg IV Q 20 min)

(0.1–0.3 mcg/kg/min), with or without neuromuscular blockade. For CS/MAC cases, various techniques may be employed depending upon the patient, the need for the interventionalist to communicate with the patient, and the hemodynamic goals of the procedure. A propofol infusion would be a typical approach. Whether the procedure is to be performed under CS/MAC or GETA, vasoactive infusions targeted toward the patient's hemodynamic goals should be available for both bolus and infusion throughout induction, maintenance, and emergence (Table 22.2).

Emergence Special considerations may be involved as the patient emerges from anesthesia. Depending upon the type of closure performed on the groin, the proceduralist may need to hold pressure on the groin for several minutes after the procedure is complete. The patient may also be required to lay flat for a certain period of time to prevent damage to the femoral artery. This may be an uncomfortable position for the patient and may even provide a challenge in achieving extubation criteria. As mentioned before, vasoactive medications via both bolus and infusion should be available to maintain tight adherence to the

agreed-upon hemodynamic goals. A typical approach to emergence is the "remifentanil wake-up" where the inhalational agent is turned off and blown off (to less than 0.25 MAC) prior to stopping the remifentanil. The patient will typically awaken 5–10 min after discontinuation of the remifentanil (and flushing of the IV line).

For patients with an obese body habitus, laying flat may prove detrimental, especially as it relates to their pulmonary mechanics. Discussions should take place between the interventionalist and the anesthesiologist regarding post-procedure positioning and the allowance for a head up or a reverse Trendelenburg position in this subset of patients. Moreover, the use of airway adjuncts such as nasal trumpets should be considered (caution with anticoagulation), to more effectively enable oxygenation and ventilation in patients at risk for suffering from obstructed airways especially in the flat position.

22.5.4 Anticoagulation

A heparinized saline infusion is continuously administered through a side port of the intra-arterial catheters to prevent thrombotic complications from the procedure. Moreover, systemic anticoagulation with heparin is generally required during the procedure, with a dose of ~70 IU heparin/kg being administered upon insertion of the arterial sheath and re-dosing with ~1000 units of IV heparin every hour after the bolus. The goal of this anticoagulation regimen is to achieve an activated clotting time (ACT) of two to three times the patient's normal value [4].

Patients are also often treated with antiplatelet therapy, such as aspirin and clopidogrel, prior to arrival for elective procedures. Adequate antiplatelet activity should be assayed preoperatively. These agents are used most often for procedures involving intra-arterial stents in order to prevent stent thrombosis. They are continued for months postoperatively. Acute intra-procedural thrombi can potentially occur, are platelet rich, and are treated very effectively with intravenous antiplatelet agents (with glycoprotein IIb/IIIa inhibitors such as tirofiban) (Table 22.3).

Table 22.3 Mechanism of action of common anticoagulants

Anticoagulant(s)	Mechanism of action
Aspirin	Inhibitor of cyclooxygenase
Clopidogrel	ADP receptor inhibitor
Abciximab, eptifibatide, tirofiban	Glycoprotein IIb/IIIa inhibitors
Heparin	Binds antithrombin III

22.6 Procedure-Specific Management

22.6.1 Aneurysms

It is estimated that 2–5% of the general population develop cerebral aneurysms [14]. Patients with either unruptured (elective) or ruptured (emergent) aneurysms may present for INR treatment. With spontaneous rupture, about 12% of patients die before arriving to the hospital [15]. For those who survive to hospital admission, the mortality rate is 26–44%, the rate of severe disability is 19%, and the incidence of a favorable outcome is 55% [16]. Aggressive early intervention is standard of care to reduce the risk of rebleed.

The International Subarachnoid Aneurysm Trial has demonstrated the utility of endovascular treatment of cerebral aneurysms [17, 18]. Aneurysms which were historically treated with surgery using clips are now being secured with the utilization of Guglielmi platinum detachable coils, often with stent assistance. While preserving flow through the parent vessel, these coils are introduced into the aneurysm with the goal of inducing stagnant flow in the aneurysm sac and in turn generating a thrombotic reaction to occlude the aneurysm [19]. The thrombogenic fibers of the coils also aid in promoting this reaction [20]. Multiple coils may be required to achieve an adequate coil packing density of at least 20–30% or more depending on the aneurysm size, type, and location [19]. Thrombosis of the parent vessel is a concern when performing an aneurysm coiling. An acute thrombotic event often requires the initiation of intra-procedural antiplatelet therapy such as tirofiban.

These techniques were initially used in the treatment of berry aneurysms as the narrow neck would retain the coils. However, stent-assisted coiling now allows for coil placement in wider-necked aneurysms using the stent to keep the coils within the sac. Some types of aneurysm morphology still require open surgical treatment including wide-necked and fusiform aneurysms or those with proximal vessels that are occluded.

A new approach to aneurysm management has emerged which involves endoluminal reconstruction of the cerebral aneurysm with a flow-diverting stent (such as the Pipeline™ stent or Pipeline™ embolization device [PED]) (Fig. 22.5). This technique allows interventionalists to treat giant aneurysms and those with complex morphologies that were previously only amenable to vessel occlusion followed by extracranial to intracranial bypass. This is a considerably different approach to the classic coiling procedure. A low-porosity self-expanding tubular mesh stent promotes blood flow along the typical course of the parent artery, thus diminishing the flow within the aneurysm [21]. Over time the aneurysm thromboses around the stent, and the flow-diverter stent slowly incorporates into the parent artery with neointimal overlay; endoluminal reconstruction takes place

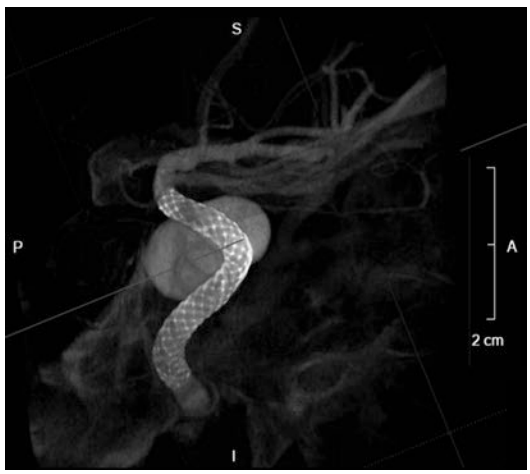

Fig. 22.5 Pipeline TM embolization device (PED) deployed across a large cavernous left internal carotid artery aneurysm

[21]. Dual antiplatelet therapy is required (for ~6 months) until the stent is fully endothelialized.

Potential complications are inherent to all of these procedures. Not filling the aneurysm with enough coils, or with a low packing density, can lead to coil compaction and a residual aneurysm with risk of future rupture [19, 22]. Thus, serial follow-up with surveillance angiography is required to monitor for these issues. Vessel perforation with a catheter or overpacking an aneurysm with coils can lead to aneurysm rupture during the procedure [19]. The risk of thrombosis in the parent vessel is also of concern.

22.6.2 Angioplasty and Stenting

Treatment of intracerebral and carotid atherosclerotic disease can be accomplished with angioplasty and/or stenting procedures. For carotid stents, activation of the carotid body can cause a bradycardic or asystolic response (with hypotension) for which the anesthesiologist should be prepared. Chronotropic agents should certainly be available, and the preemptive placement of transcutaneous pacing pads should also be considered as a precaution. Strict blood pressure control should also be maintained to promote flow through any stenotic regions. As with carotid endarterectomies, the blood pressure should be kept at or above baseline pressures prior to stent deployment (i.e., prior to expanding the stenotic lesion) and then controlled at or below baseline following stent expansion to avoid a cerebral hyperperfusion syndrome (which could cause cerebral edema or hemorrhage). To allow for intraoperative neuromonitoring, these procedures are frequently performed under CS/MAC. This provides the added benefit of curtailing some of the hypotension that may be associated with GETA. Complications of carotid stents include thrombosis, vessel dissection, dysrhythmias, vasospasm, vessel perforation, cerebral hemorrhage from hyperperfusion, and cerebral embolism (ischemic stroke) [1].

22.6.3 Cerebral Vasospasm

Patients may present to INR for treatment of cerebral vasospasm following aneurysmal subarachnoid hemorrhage. The onset of vasospasm is typically about 5 days after the initial hemorrhage, and the risk of its occurrence is proportionate to the subarachnoid blood burden. Anesthetic management largely entails *hypertensive euvolemia* (no longer the classic "triple H" therapy). Hypervolemia has no proven benefit and leads to a myriad of complications such as pulmonary edema and heart failure. The endovascular treatment of cerebral vasospasm consists of either balloon angioplasty or intra-arterial injection of vasodilators including calcium channel blockers, milrinone, nitrates, or papaverine [23]. Arterial injection of such vasodilators can dramatically lower the blood pressure, for which the anesthesiologist should be prepared. These patients are at high risk of ischemic stroke due to the vasospasm, and the anesthesiologist must maintain cerebral perfusion pressure. Typical goals are systolic blood pressure (SBP) 160–180 mmHg. These patients frequently have ventriculostomies and require ICP monitoring and occasional cerebrospinal fluid drainage intraoperatively.

22.6.4 Arteriovenous Malformation/ Fistula/Tumor Embolization

Embolization therapy can be used to treat a variety of neurovascular lesions including arteriovenous malformations (AVM), fistulas (dural AV, cavernous-carotid), and tumors. Embolization can be done with a variety of agents including polyvinyl alcohol particles, liquid agents, cyanoacrylate glues (*N*-butyl cyanoacrylate or NBCA), or nonadhesive polymerizing agents (ethylene-vinyl alcohol copolymer in a dimethyl sulfoxide solvent known as Onyx®) [20, 24]. However, with any embolization therapy, there are potential complications including vessel rupture, inadvertent passage of the embolization material into the systemic circulation or vessels supplying normal brain, potential to glue the catheter to the injected polymer, and even injection of particulate matter into the pulmonary vessels leading to a pulmonary embolism [20]. Injection of embolic material into the pulmonary vessels is more likely to occur with embolization of the great vein of Galen or larger fistulas/AVMs [20]. For these reasons, deliberate hypotension may be required to help lower the risk of these events [1].

AVMs are a confluence of several feeding arteries into a tangled nidus that is drained by one or more veins. The goal of INR management of AVMs is to occlude as many of the fistulous arteries as possible. This is usually done adjunctively with surgical resection or radiotherapy (gamma knife). AVMs can vary in size and can have a high propensity to bleed during surgical resection. INR is increasingly being used to treat intracerebral AVMs both as a primary treatment and to embolize feeding vessels in hopes of helping minimize blood loss prior to surgical resection [25]. During AVM embolization in the angiography suite, some degree of induced hypotension may be desirable in order to prevent flow through the AVM. This can be accomplished with anesthetics, short-acting vasodilators (nitroglycerin, nicardipine, clevidipine), or even adenosine. A smooth, hemodynamically controlled emergence is also paramount in these patients as the AVM is usually not fully secured on the first treatment and often requires multiple interventions. This keeps the patient at ongoing risk of intracerebral hemorrhage [1].

Dural arteriovenous fistulas (AVF) are lesions that are typically acquired due to opening of potential arteriovenous shunts or stenosis of the dural sinuses [26]. Symptoms vary depending on the involved vessels. Dural AVFs can increase venous pressure, and it should be noted that this can impact cerebral perfusion pressure when determining the blood pressure goals [1].

Craniofacial venous malformations are often congenital and are typically treated with sclerotherapy either for cosmetic reasons or to ablate lesions which impede on the airway or oropharynx. It should be noted that the vascular deformities can enlarge after the injection, with potential to impact the airway [1].

22.6.5 Strokes

Anesthesiologists are encountering increasing numbers of patients presenting for endovascular treatment of acute ischemic stroke (AIS). In 2015, multiple prospective randomized controlled trials were published, all demonstrating the superiority of endovascular thrombectomy over intravenous alteplase (TPA) for patients with acute anterior circulation large vessel occlusion (LVO), less than 6 h from onset of symptoms [27–30]. This success is largely credited to the implementation of a new "stent retriever" technology (Fig. 22.6).

The choice of anesthetic type (CS/MAC vs. GETA) has been a hotly debated topic. Because of the extreme urgency in achieving recanalization, and need to minimize door-to-groin and groin-to-reperfusion times, the initiation of the anesthetic must be done in a rapid manner regardless of the type. Multiple *retrospective* studies of patients in the interventional stroke trials demonstrated an association between the use of GETA and increased mortality and/or neurological disability [31–33]. However, concern for selection bias was high as the neurologically "sicker" patient often does not meet criteria for CS/MAC. Fortunately, we now have data from three *prospective* trials conducted in Europe (SIESTA, ANSTROKE, and GOLIATH) [34–37]. These trials randomized patients with acute anterior circulation LVO to GETA or CS/MAC and demonstrated *no* difference in neurological outcome at 24 h or 3 months post-stroke. Although there was a slight delay in the time to initiate GETA compared to CS/MAC, there was a shorter procedural time in the GETA group, presumably from providing more optimal procedural conditions, less patient movement, and less motion artifact, thus resulting in no overall time delay.

At present, the anesthetic management of the patient is an individualized decision to be made between the anesthesiologist and the interventionalist. GETA is considered a safe option for the patient presenting for acute stroke intervention. Maintenance of cerebral perfusion pressure (SBP 140–180 mmHg) is of the utmost importance. Intravenous or inhalational anesthetics are acceptable (both were

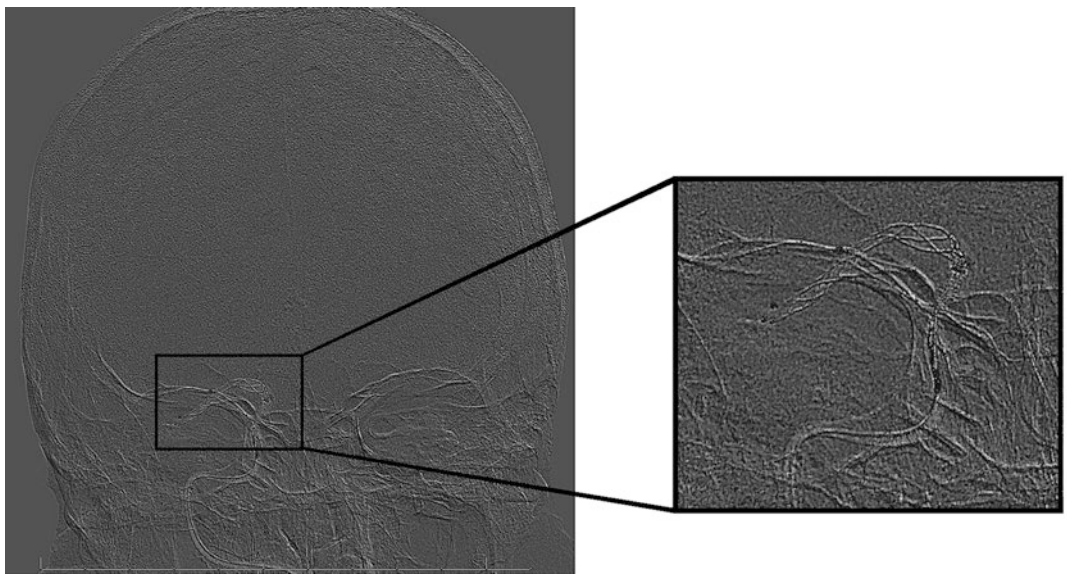

Fig. 22.6 Stent retriever technology at the right internal carotid artery terminus

used in the different prospective trials). Looking ahead, new evidence from the DAWN trial suggests that some patients with small core infarcts and large penumbral regions based on CT or MR perfusion imaging can be treated with endovascular thrombectomy up to 24 h post-ictus [38]. This will continue to increase the number of endovascular stroke thrombectomy patients that anesthesiologists will encounter worldwide as we move into the future.

22.7 INR Complications

The two primary neurologic complications of INR procedures are intracerebral hemorrhage and thromboembolic complications [20], as discussed above.

As with any medication administration, anaphylaxis is possible from medications administered by the anesthesiologist or from contrast given intra-arterially by the interventionalist during the procedure. Epinephrine, inhaled β-2 agonists, steroids, and antihistamines should be available in the INR suite should they become necessary. Pretreatment with corticosteroids and/or antihistamines to prevent such reactions remains controversial [39].

Administration of contrast media can also lead to contrast-induced nephropathy. Patients considered to be at the highest risk include those with pre-existing renal insufficiency (serum creatinine >1.5 mg/dl), diabetes mellitus, volume depletion, myeloma, hypertension, hyperuricemia, advanced age (>70 years), cardiovascular disease, and the use of diuretics. Patients at high risk should be considered for preventative measures such as hydration with 0.9 or 0.45% saline at 100 mL/h for 6–12 h pre-procedure and continuing for 4–12 h afterward. The minimum necessary dose of contrast media should also be utilized in these patients [39].

Groin hematoma, retroperitoneal hematoma, and femoral pseudoaneurysm are also potential complications from arteriotomy. Closure devices, groin pressure, lying flat, and strict hemodynamic control are typically used to help prevent such complications from occurring.

Finally, intra-arterial cerebral air emboli are always a risk. Nitrous oxide should be avoided. Clinically significant air emboli (i.e., large enough to cause neurologic symptoms on emergence) should be treated with 100% inspired oxygen followed by emergent hyperbaric oxygen therapy in a hyperbaric chamber [13, 40, 41].

Ongoing communication with the neurointerventionalist is essential in order to be alerted early to complications, provide timely intervention, and plan for post-procedural care.

22.8 Conclusion

We will continue to see a steady increase in neurointerventional procedures as imaging technology and endovascular devices evolve and improve. Similarly, we will see increasing endovascular stroke interventions as we are able to expand the treatment window using advanced neuroimaging.

Anesthesiologists should keep the following five points in mind when approaching INR cases:

1. Cerebrovascular pathology varies widely between patients. Discuss the specific pathology and planned procedure with the interventionalist.
2. Agree on hemodynamic goals (with the interventionalist) so that everyone shares the same game plan. This is true for both elective and emergent cases.
3. Discuss the degree of urgency of emergent cases and the planned intervention. There is tremendous variability between patients.
4. Discuss the risk for central nervous system injury from *hypotension AND/OR hypertension*. In other words, how fragile is the individual patient's cerebrovascular condition?
5. Finally, plan for the postoperative period in terms of airway management and hemodynamic goals.

Key Points

- Cerebrovascular pathology varies widely between patients. Discuss the specific pathology and planned procedure with the interventionalist.
- Agree on hemodynamic goals (with the interventionalist) so that everyone shares the same game plan. This is true for both elective and emergent cases.
- Discuss the degree of urgency of emergent cases and the planned intervention. There is tremendous variability between patients.
- Discuss the risk for central nervous system injury from hypotension AND/OR hypertension. In other words, how fragile is the individual patient's cerebrovascular condition?
- Finally, plan for the postoperative period in terms of airway management and hemodynamic goals.

References

1. Young WL, Pile-Spellman J. Anesthetic considerations for interventional neuroradiology. Anesthesiology. 1994;80(2):427–56.
2. Tonnessen BH, Pounds L. Radiation physics. J Vasc Surg. 2011;53(1 Suppl):6S–8S.
3. United States Department of Labor Occupational Safety and Health Administration. Radiology OSHA safety and health topics. Washington, DC: United States Department of Labor Occupational Safety and Health Administration; 2018.
4. Varma MK, Price K, Jayakrishnan V, Manickam B, Kessell G. Anaesthetic considerations for interventional neuroradiology. Br J Anaesth. 2007;99(1):75–85.
5. Eskioglu E, Burry MV, Mericle RA. Transradial approach for neuroendovascular surgery of intracranial vascular lesions. J Neurosurg. 2004;101(5):767–9.
6. Ross IB, Luzardo GD. Direct access to the carotid circulation by cut down for endovascular neurointerventions. Surg Neurol. 2006;65(2):207–11.. discussion 211
7. Ruijters D, Homan R, Mielekamp P, van de Haar P, Babic D. Validation of 3D multimodality roadmapping in interventional neuroradiology. Phys Med Biol. 2011;56(16):5335–54.
8. Schwartz BG, Burstein S, Economides C, Kloner RA, Shavelle DM, Mayeda GS. Review of vascular closure devices. J Invasive Cardiol. 2010;22(12):599–607.
9. Barrett BJ, Parfrey PS. Clinical practice. Preventing nephropathy induced by contrast medium. N Engl J Med. 2006;354(4):379–86.
10. McDonagh DL, Olson DM, Kalia JS, Gupta R, Abou-Chebl A, Zaidat OO. Anesthesia and sedation practices among neurointerventionalists during acute ischemic stroke endovascular therapy. Front Neurol. 2010;1:118.
11. Luginbuhl M, Remonda L. Interventional neuroradiology. Recent developments and anaesthesiologic aspects. Minerva Anestesiol. 1999;65(6):445–54.
12. Wang LP, Wolff J. Anesthetic management of severe chronic cardiopulmonary failure during endovascular embolization of a PICA aneurysm: a case report. J Neurosurg Anesthesiol. 2000;12(2):120–3.
13. Powers WJ, Rabinstein AA, Ackerson T, et al. 2018 Guidelines for the early management of patients with acute ischemic stroke: a guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2018;49(3):e46–e110.
14. Steiner T, Juvela S, Unterberg A, et al. European Stroke Organization guidelines for the management of intracranial aneurysms and subarachnoid haemorrhage. Cerebrovasc Dis. 2013;35(2):93–112.
15. Huang J, van Gelder JM. The probability of sudden death from rupture of intracranial aneurysms: a meta-analysis. Neurosurgery. 2002;51(5):1101–5.. discussion 1105–7.
16. Nieuwkamp DJ, Setz LE, Algra A, Linn FH, de Rooij NK, Rinkel GJ. Changes in case fatality of aneurysmal subarachnoid haemorrhage over time, according to age, sex, and region: a meta-analysis. Lancet Neurol. 2009;8(7):635–42.
17. Molyneux A, Kerr R, Stratton I, et al. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised trial. Lancet. 2002;360(9342):1267–74.
18. Li H, Pan R, Wang H, et al. Clipping versus coiling for ruptured intracranial aneurysms: a systematic review and meta-analysis. Stroke. 2013;44(1):29–37.
19. Otani T, Nakamura M, Fujinaka T, et al. Computational fluid dynamics of blood flow in coil-embolized aneurysms: effect of packing density on flow stagnation in an idealized geometry. Med Biol Eng Comput. 2013;51(8):901–10.
20. Osborn IP. Anesthetic considerations for interventional neuroradiology. Int Anesthesiol Clin. 2003;41(2):69–77.
21. Nelson PK, Lylyk P, Szikora I, Wetzel SG, Wanke I, Fiorella D. The pipeline embolization device for the intracranial treatment of aneurysms trial. AJNR Am J Neuroradiol. 2011;32(1):34–40.
22. Roy D, Milot G, Raymond J. Endovascular treatment of unruptured aneurysms. Stroke. 2001;32(9):1998–2004.

23. Lee KH, Lukovits T, Friedman JA. "Triple-H" therapy for cerebral vasospasm following subarachnoid hemorrhage. Neurocrit Care. 2006;4(1):68–76.
24. Katsaridis V, Papagiannaki C, Aimar E. Curative embolization of cerebral arteriovenous malformations (AVMs) with Onyx in 101 patients. Neuroradiology. 2008;50(7):589–97.
25. Jafar JJ, Davis AJ, Berenstein A, Choi IS, Kupersmith MJ. The effect of embolization with N-butyl cyanoacrylate prior to surgical resection of cerebral arteriovenous malformations. J Neurosurg. 1993;78(1):60–9.
26. Ahmed AMC, Fistulas A. In: Michael J, RBD A, editors. Encyclopedia of the neurological sciences. 2nd ed. San Diego: Elsevier; 2014. p. 277–80.
27. Berkhemer OA, Fransen PS, Beumer D, et al. A randomized trial of intraarterial treatment for acute ischemic stroke. N Engl J Med. 2015;372(1):11–20.
28. Campbell BC, Mitchell PJ, Kleinig TJ, et al. Endovascular therapy for ischemic stroke with perfusion-imaging selection. N Engl J Med. 2015;372(11):1009–18.
29. Goyal M, Demchuk AM, Menon BK, et al. Randomized assessment of rapid endovascular treatment of ischemic stroke. N Engl J Med. 2015;372(11):1019–30.
30. Saver JL, Goyal M, Bonafe A, et al. Stent-retriever thrombectomy after intravenous t-PA vs. t-PA alone in stroke. N Engl J Med. 2015;372(24):2285–95.
31. 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(6):1175–9.
32. 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(6):1180–4.
33. Nichols C, Carrozzella J, Yeatts S, Tomsick T, Broderick J, Khatri P. Is periprocedural sedation during acute stroke therapy associated with poorer functional outcomes? J Neurointerv Surg. 2010;2(1):67–70.
34. Schonenberger S, Uhlmann L, Hacke W, et al. Effect of conscious sedation vs general anesthesia on early neurological improvement among patients with ischemic stroke undergoing endovascular thrombectomy: a randomized clinical trial. JAMA. 2016;316(19):1986–96.
35. Lowhagen Henden P, Rentzos A, Karlsson JE, et al. General anesthesia versus conscious sedation for endovascular treatment of acute ischemic stroke: the anstroke trial (anesthesia during stroke). Stroke. 2017;48(6):1601–7.
36. Goyal M, Menon BK, van Zwam WH, et al. Endovascular thrombectomy after large-vessel ischaemic stroke: a meta-analysis of individual patient data from five randomised trials. Lancet. 2016;387(10029):1723–31.
37. Simonsen CZ, Yoo AJ, Sørensen LH, Juul N, Johnsen SP, Andersen G, Rasmussen M. Effect of general anesthesia and conscious sedation during endovascular therapy on infarct growth and clinical outcomes in acute ischemic stroke: a randomized clinical trial. JAMA Neurol. 2018;75(4):470–7. https://doi.org/10.1001/jamaneurol.2017.4474.
38. Nogueira RG, Jadhav AP, Haussen DC, et al. Thrombectomy 6 to 24 hours after stroke with a mismatch between deficit and infarct. N Engl J Med. 2018;378:11–21.
39. Namasivayam S, Kalra MK, Torres WE, Small WC. Adverse reactions to intravenous iodinated contrast media: a primer for radiologists. Emerg Radiol. 2006;12(5):210–5.
40. Trytko BE, Bennett MH. Arterial gas embolism: a review of cases at Prince of Wales Hospital, Sydney, 1996 to 2006. Anaesth Intensive Care. 2008;36(1):60–4.
41. Moon REGD. The physiology and medicine of diving. New York: Elsevier Science; 2003.