REVIEW ARTICLE

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Anesthetic Considerations for Interventional Neuroradiology

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Contents

IT has been over 30 yr since Luessenhop and Spence[70] proposed embolizing cerebral fistulae under radiologic guidance and almost 20 yr since Serbinenko[105] described the use of intravascular balloons for selective vascular occlusions. Especially over the past decade, interventional neuroradiologists have made enormous progress in the treatment of central nervous system disease by the endovascular approach.[7,14,15,34,50,55,69,107] Development of novel materials and techniques has allowed unprecedented access into the distal cerebral and spinal cord vasculature and opened new therapeutic windows, as well as offered the means of gaining greater understanding of central nervous system pathophysiology.

In fact, the enormous amount of development in this discipline has left some disagreement as to what it should be called (some suggestions have been endovascular therapy, surgical neuroangiography, endovascular neurosurgery, and interventional neuroradiology), to reflect the interests of both neuroradiologists and neurosurgeons.[75,121] Formal guidelines for training and certification are being considered by the concerned professional societies.‡ Local practices worldwide vary considerably.[29,69] At some institutions, the interventional neuroradiology (INR) service is an autonomous department, separate from the division of neuroradiology. Many INR services have separate ward and admitting privileges and designated ICU space, and patients are cared for by the INR service from admission to discharge. In some institutions, such as ours, the INR staff have joint neurosurgery and radiology appointments and admitting privileges. Ward coverage is handled by INR staff and neurosurgery residents.

As the frontiers of INR expand, care of these patients will demand more of the anesthesiologist's participation in years to come. Anesthesiologists have an unquestionable role in the management and prevention

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‡ There are both a World Federation of Interventional and Therapeutic Neuroradiology and an American Society of Interventional and Therapeutic Neuroradiology.

Table 1. Interventional Neuroradiologic Procedures and Primary Anesthetic Considerations

Procedure	Anesthetic Considerations
Therapeutic embolization of vascular malformation	
Intracranial AVMs	Deliberate hypotension, post-procedure NPPB
Dural AVM	Deliberate hypercapnia
Extracranial AVMs	Deliberate hypercapnia
CCF	Deliberate hypercapnia, post-procedure NPPB
Cerebral aneurysms	Aneurysmal rupture, blood pressure control*
Sclerotherapy of venous angiomas	Airway swelling, hypoxia, hypoglycemia, intoxication from ethanol
Balloon angioplasty of occlusive cerebrovascular disease	Cerebral ischemia, deliberate hypertension, concomitant coronary artery disease
Balloon angioplasty of cerebral vasospasm secondary to aneurysmal SAH	Cerebral ischemia, blood pressure control*
Therapeutic carotid occlusion for giant aneurysms and skull base tumors	Cerebral ischemia, blood pressure control*
Thrombolysis of acute thromboembolic stroke	Post-procedure ICH, concomitant coronary artery disease, blood pressure control*
Intraarterial chemotherapy of head and neck tumors	Airway swelling, intracranial hypertension
Embolization for epistaxis	Airway control

AVM = arteriovenous malformation; NPPB = normal perfusion pressure breakthrough; CCF = carotid cavernous fistula; SAH = subarachnoid hemorrhage; ICH = intracerebral hemorrhage.

* Blood pressure control refers to deliberate hypo- and/or hypertension.

of morbidity and mortality during INR procedures: Many of the risks encountered in this newer arena are the same as those encountered during traditional operative neurosurgery (*e.g.,* aneurysmal rupture or cerebral ischemia from vascular occlusion) and the anesthesiologist's manipulation of systemic and cerebral hemodynamics (*e.g.,* deliberate hypotension or hypertension). Although many of the risks and responses are, for the most part, conceptually the same, there are many important differences in the working environment. The various types of INR procedures[29,69] and primary areas of anesthetic care interaction are shown in table 1. Tables 1, 2, and 3 illustrate the scope of disease processes, procedures, and complications that might be expected in an INR practice.

Historically, the pioneers of INR provided light intravenous sedation with rudimentary monitoring for their adult patients, although many opted to have an anesthetist in attendance. One recent survey estimated that 75% of INR services had the "anesthesia service monitor procedures in radiology suite or operating room."[69] Some centers have employed anesthesiologists in an "on call" fashion.[96] With certain exceptions,[87] however, there is a conspicuous lack of information on specific anesthetic considerations for INR available in the existing literature. As the complexity of procedures and breadth of patient populations expands, the distinction between the endovascular therapists' angiography suite and the operating room will blur, and the need for sophisticated sedation techniques and monitoring will increase.

This article will review basic concepts in treating the various disease processes and emphasize the synergistic interaction between anesthetist and interventionist. To furnish a perspective on this important area of "out-service" anesthesia care and the new vocabulary that goes along with it, we have reviewed the published literature on the subject and summarized our recent clinical experience. We would like to emphasize at the outset that many of the techniques described are the result of the evolution of the authors' clinical experience. In some instances, recommendations presented are based on clinical intuition, and many remain to be scientifically validated in future clinical trials or studies.

Interventional Neuroradiology Considerations Pertinent to Anesthetic Care

General Comments and Scope of Interventional Neuroradiology Procedures

The procedures performed in INR practice are inherently dangerous. Despite tremendous advances in neuroradiology (cerebral angiography was a *surgical* procedure in the 1950s[30]), standard *diagnostic* cerebral angiography is associated with a low but significant morbidity. In 1984, Earnest *et al.* reported an overall incidence of complications in 8.5% of patients, neurologic complications in 2.6% of patients, and permanent neurologic deficit in 0.33% of patients in a prospective study of 1,517 patients.[31] In 1987, Dion

et al. reported a 1.3% neurologic complication rate in the first 24 h (0.1% permanent) following 1,002 procedures and a 1.8% rate (0.3%) for late cerebral ischemic events between 24 and 72 h after angiography.[24]

Our clinical experience with 243 procedures in 170 patients performed from April 1992 to April 1993 is summarized in table 2. Complications are shown in table 3. Examples of the interaction of anesthesia care with patient management and outcome are discussed in specific sections.

In our series, the total 30-day rate of complications was 33/243 procedures (14%). The total for death and major complications was 3/243 (1.2%). Complications in our series that had significant interaction with anesthetic care are discussed in later sections.

Even in experienced hands, the complication rate may be considerably higher for multifactorial reasons (*e.g.,* patient selection) than noted in our experience. For example, in 1991, Purdy *et al.* reported an incidence of intracranial hemorrhage (ICH) during arteriovenous malformation (AVM) embolization of 11% (7/63), with a poor outcome in 5% (3/63).[95] A primary goal of anesthesia coverage is immediate intervention in the event of catastrophe such as ICH.

Interventional Neuroradiology Treatment Goals

Because INR therapy is relatively new, precise indications and efficacy of many of the treatment modalities require further definition. For detailed discussion of indications and experience, the reader is referred elsewhere.[4,5,7,8,14,15,29,34,50,69,94,107,128,131] However, a basic grasp of the terms, general concepts, and methodology is desirable for optimal anesthesia care and further development of the field.

The goals of INR therapy, as in any surgical or invasive therapy, must be well defined. In general, goals fall into three classes of treatment: 1) *definitive, e.g.,* certain dural and spinal fistulae, 2) *adjunctive* therapy for surgery or radiotherapy, *e.g.,* preoperative embolization of AVM feeding arteries that will be difficult to control during craniotomy, and 3) *palliative, e.g.,* intraarterial chemotherapy for a malignant and inoperable brain tumor.

§ Catheters usually are measured as French, which equals the circumference in millimeters (3 French ≈ 1-mm diameter); wires are measured in "Mils," which is the diameter in thousandths of an inch (040 Mils = 1 mm or 3 French). Gauge, used for needles, is the number of items laid side-to-side that are required to make an inch (27 G ≈ 1.2 French ≈ 016 Mils; 20 G ≈ 2.6 French ≈ 035 Mils; 14 G ≈ 6.3 French ≈ 083 Mils). See Rüfenacht and Latchaw[100] for a detailed discussion of this topic.

Many of the considerations described below also are applicable to certain complex neuroradiologic diagnostic procedures. As discussed in detail below (see imaging technology), *superselective angiography* can define complex angioarchitecture not seen on routine angiography. Proximal carotid or vertebral artery *test occlusion* can be used. Finally, *provocative testing* with deliberate hypotension during test occlusions or superselective anesthesia functional examination (SAFE), such as intraarterial amobarbital administration into vascular territories at risk, fall into this category.

Radiologic Vascular Access and Methods

The INR procedures typically involve placing special catheters into the arterial circulation of the head, neck, or spinal cord. The transfemoral approach is used in most cases, although direct carotid or brachial puncture can be used in special circumstances. As illustrated in figure 1, transfemoral access is accomplished by the placement of a large introducer sheath into the femoral artery, usually 7.5 French.§ The transfemoral puncture site can be infiltrated with a local anesthetic, such as 0.25% bupivicaine. Because of the proximity of the femoral nerve, inadvertent femoral nerve block can result in a motor and sensory deficit that must be differentiated from central nervous system damage. Through this introducer, a 7.0-French coaxial catheter then is

Table 2. Summary of Procedures Performed in Present Series

Procedure	Lesion	Procedures (n)	Patients (n)
Embolization	Intracerebral AVM	144	70
Embolization	Dural AVMs	12	10
Embolization	Spinal AVM	15	10
Embolization	Extremity and trunk AVM	10	4
Embolization	CCF	5	5
Embolization	Cerebral aneurysms	14	14
Embolization	Tumors (brain or head and neck)	5	5
Sclerotherapy	Venous malformations	13	27
Angioplasty	Vasospasm from SAH	6	6
Angioplasty	Carotid stenosis	6	6
Test occlusion	Cerebral aneurysms	2	2
Test occlusion	Brain tumors	5	5
Superselective angiography	AVMs, aneurysms	6	6
Total		243	170

AVM = arteriovenous malformation; CCF = carotid cavernous fistula; SAH = subarachnoid hemorrhage.

positioned by fluoroscopic control into the carotid or vertebral arteries. Finally, a 1.5–2.8-French superselective microcatheter then is introduced into the cerebral circulation. The superselective catheter can be used to deliver drugs, embolic agents, or balloons to the desired location. A typical microcatheter and various balloons are shown in figures 2 and 3.

Transfemoral venous access can be used to reach the dural sinuses and, in some cases, the arterial side of the AVMs as well. Direct percutaneous puncture is used for access to superficial venous malformations.

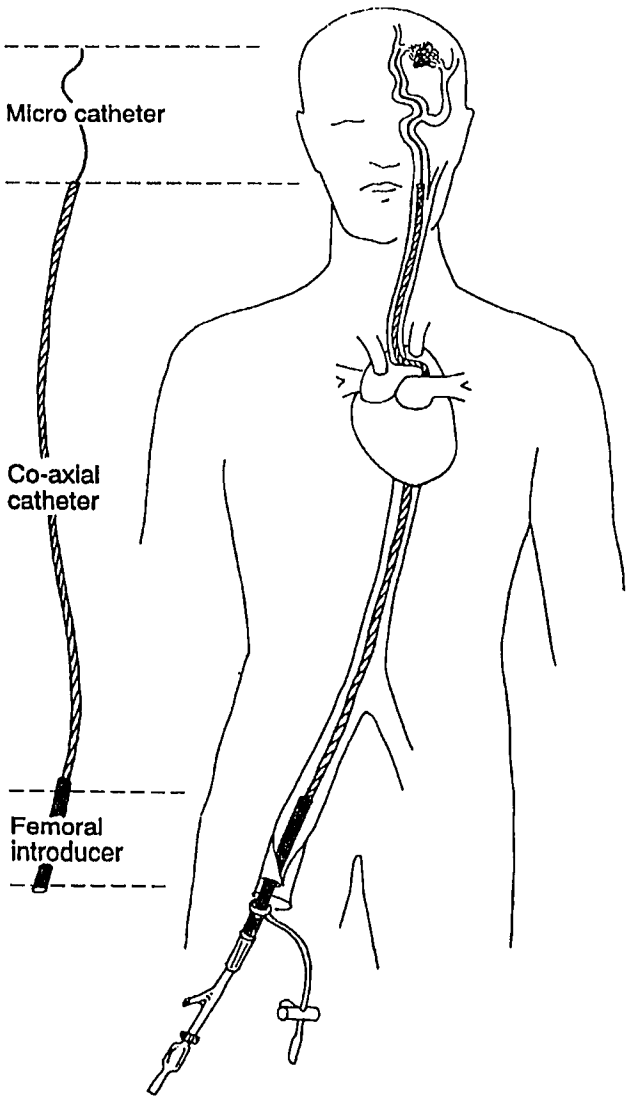

Fig. 1. Representation of a typical arrangement of the transfemoral coaxial catheter system showing the femoral introducer, the coaxial catheter, and the microcatheter (superselective catheter). (Adapted with permission from Young WL: Clinical Neuroscience Lectures. Munster, Cathenart, 1992.)

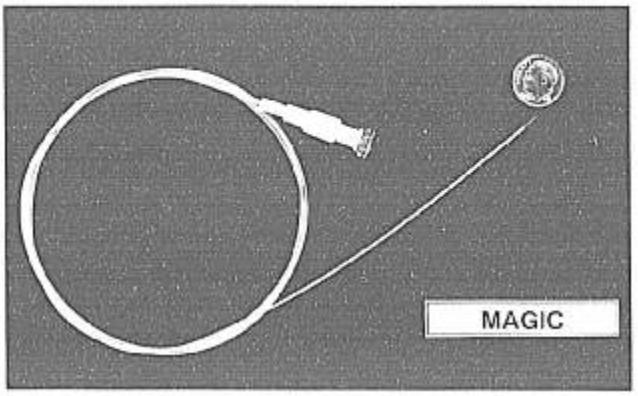

Fig. 2. An illustration of a Magic microcatheter (Balt, Montmorency, France) placed next to a dime (≈ 17 mm) for size comparison.

Materials for Embolization or Infusion

The nature of the disease, purpose of the embolization, size and penetration of emboli and vessels, and

Table 3. Adverse Events

Event	Neurologic (n)	Angiographic (n)	Technical (n)	Total (n)
Death	2	0	0	2
Major	1	0	0	1
Minor	4	0	0	4
Transient	19	2	5	26
Total	26	2	5	33

Adverse events were classified as: Death, cause of death possibly or directly related within 30 days of procedure; Major, neurologic deficit, not able to return to previous level of work or activities of daily life; Minor, neurologic deficit, able to return to previous level of work or activities of daily life; or Transient, any change or complication resolving before discharge.

Major complications included the following: One patient suffered an intracerebral hemorrhage (ICH) during AVM embolization and never regained consciousness from deep coma; the patient expired 2 months after the incident. Another patient, who had suffered spontaneous ICH twice previously, had another spontaneous ICH 15 days after AVM embolization (which may have been related to delayed glue-related venous thrombosis or natural history). Another patient with a skull base tumor underwent carotid test occlusion and failed the occlusion test (see specific procedures: carotid test occlusion and therapeutic carotid occlusion). He had a complicated post-procedure course that involved gastric aspiration, sepsis, and a hypotensive episode resulting in a cerebral watershed infarct.

Neurologic problems were defined as an objective neurologic deficit or, in the case of hemorrhage, loss of consciousness. Angiographic complications included two cases of intimal dissection during the course of the procedure that resulted in an angiographically demonstrable lesion but did not result in symptoms. Technical complications included three asymptomatic patients in whom the catheter was glued inadvertently into the vessel, one patient with leg arterial air embolism, and a patient with a femoral nerve block from local infiltration.

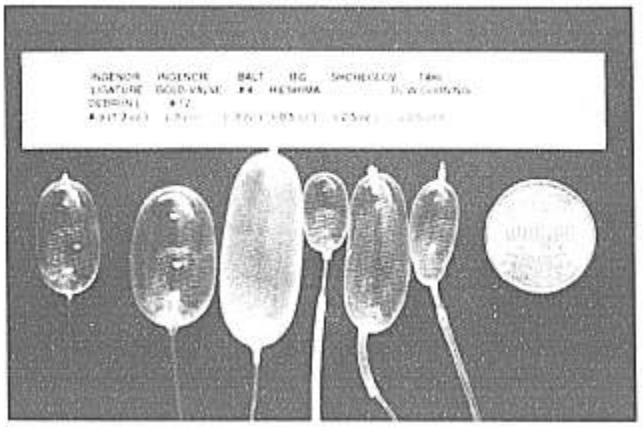

Fig. 3. Various balloons that can be used to occlude vessels temporarily or permanently, placed next to a penny (≈ 18 mm) for size comparison. The numbers in parentheses indicate the balloon volume.

permanency of occlusion are among the factors taken into consideration for agent selection. The ideal choice and combination of agents is controversial.[42,94,130] Table 4 summarizes the various materials in current use.

Glues are of particular use in our experience, the newest being N-butyl cyanoacrylate. It is a low-viscosity liquid monomer that polymerizes to a solid form on contact with ionic solutions, including blood and saline, but not D5W. It is blended with radioopaque materials of tantalum micropowder and an oil-based agent such as ethiodized oil (a contrast agent used for lymphography and hysterosalpingography). Depending on the clinical situation, the radiologist can adjust how fast the glue solidifies once it is injected into the circulation by adjusting the polymerization time. Increasing the amount of oil can vary polymerization time from a few milliseconds to over 5 s.

Imaging Technology

Necessary radiologic imaging methods include high resolution fluoroscopy and high-speed digital subtraction angiography (DSA) with a "roadmapping" function. High-speed DSA‖ can provide as many as 12–30 images per second. To remove bone shadows and other nonvascular structures from the images, a "scout" film is taken before each run. This scout film serves as a "mask". The mask is subtracted by computer from all subsequent images in the run so that only opacified vessels with contrast are visible (figs. 4 and 5).

‖ Sometimes called digital vascular imaging.

Injection of contrast through distally placed superselective catheters yields a level of specificity to delineate vascular anatomy and the pathologic nature of the diseases in detail not obtainable with the proximal (*e.g.*, internal carotid) injection used for conventional angiography. This is particularly pertinent for certain complex AVMs and aneurysms.

To facilitate placement of superselective catheters in the distal circulation, a technique called "roadmapping" is used. To make a roadmap, a bolus of dye is injected into the circulation from the proximal coaxial catheter (*e.g.*, the internal carotid or vertebral artery) to obtain an image that demonstrates the vascular anatomy. The computer then superimposes this image on the live fluoroscopy image so the radiologists can see the progress of the radioopaque catheter (especially the tip) against the roadmap. Any motion during this stage of the procedure profoundly degrades the image. This is illustrated in figure 5.

Anesthetic Considerations

In adults, the basic anesthetic approach is conscious sedation. This allows for intermittent assessment of neurologic function during manipulation of the vasculature. Small children and uncooperative adult patients generally will require general anesthesia with tracheal intubation; general anesthesia also is used for

Table 4. Materials Used in Interventional Neuroradiologic Procedures

Balloons
Detachable
Nondetachable
Solid agents
Polyvinyl alcohol particles
Oxidized cotton
Suture material
Coils
Simple
Detachable
Silastic pellets
Liquid agents
Cyanoacrylates (NBCA)
USP grade 95% ethanol
Thrombolytic agents
Urokinase
Streptokinase
Tissue plasminogen activator
Chemotherapeutic agents for tumors

This list is not meant to be inclusive and is for illustrative purposes only.
NBCA = n-butylcyanoacrylates; USP = U. S. Pharmacopia.

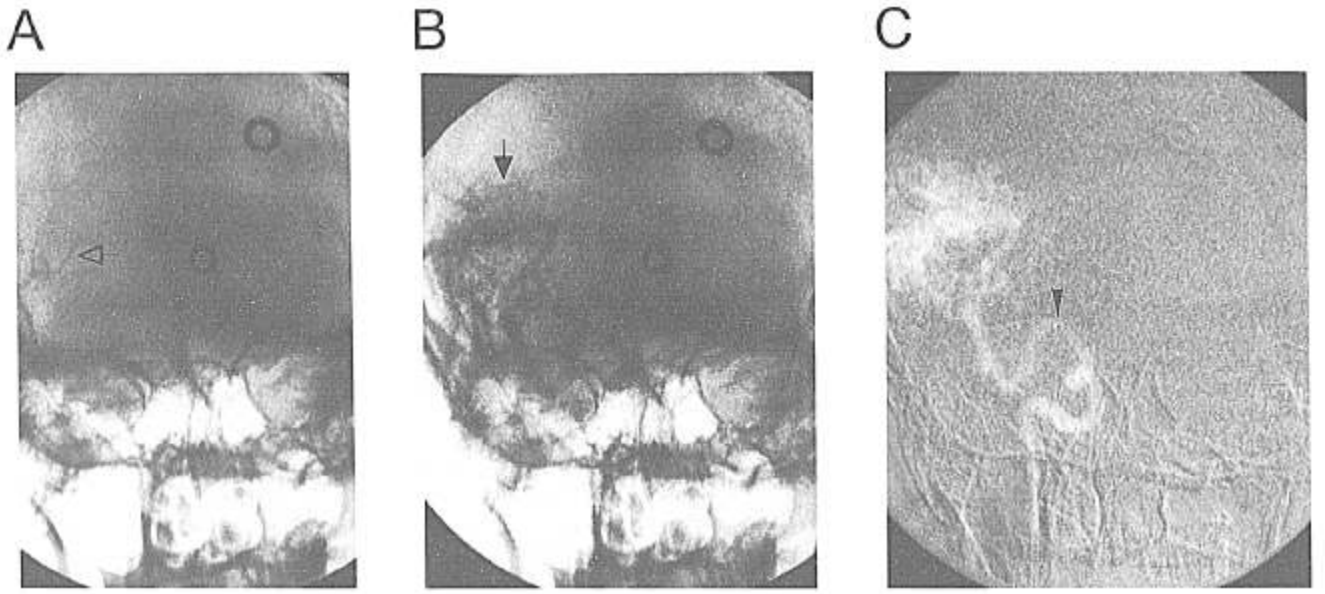

Fig. 4. *(A)* **An anterioposterior scout film showing bone and n-butyl cyanoacrylate (NBCA) glue from previous embolization. The round objects are aluminum washers used to size vessel diameters. The open arrow indicates NBCA from a prior embolization. This image is used as the "mask".** *(B)* **The same view showing contrast injected through the coaxial carotid catheter; the arrow indicates the arteriovenous malformation (AVM)-nidus filling from branches of the middle cerebral artery (MCA).** *(C)* **Subtraction of the mask image of** *A* **from** *B* **results in the "roadmap". This digital image is superimposed on live fluoroscopy and, assuming there is no patient motion, will reflect the course the microcatheter will take as it is advanced distally. The arrow indicates the tip of the microcatheter in the MCA (which is visible in** *A*, *B*, **and** *C*).

certain procedures discussed below (see specific procedures).

Preprocedure Considerations

History. In addition to the usual preanesthetic evaluation of the neurosurgical patient, the physician should note the patient's previous experience with angiography, history of prior anticoagulation or coagulation disorders, protamine allergy (including insulin use, fish allergy, and prior vasectomy), recent steroid use, and contrast reactions (including general atopy and iodine or shellfish allergies). Neck, back, or joint problems can influence the physician's ability to secure the airway and the patient's tolerance to lying supine for several hours. In the population with occlusive cerebrovascular disease, control of preprocedure essential hypertension is critical for perioperative hemodynamic stability, as lucidly reviewed by Gelb and Herrick.[40] The possibility of pregnancy in female patients should be determined.

Physical Examination. Conscious sedation can predispose certain patients to airway obstruction: Checking the patency of the nares will let the anesthetist know which side is less likely to present a problem if the need arises to place a nasal cannula intraoperatively. Of special concern is the patient with a tumor or venous malformation that involves the upper airway (see sclerotherapy of venous angiomas, and intraarterial chemotherapy and embolization of tumors). The potential for postprocedure swelling that might impinge on the airway should be discussed carefully with the INR team.

Preprocedure Laboratory Testing. In addition to general preanesthetic considerations, evaluation of hemostatic function should be considered.

Premedication. An anxiolytic can be given if appropriate to the patient's sensorium. In cases in which deliberate hypotension is to be used, a preoperative regime of a beta-blocker or angiotensin-converting enzyme inhibitor such as captopril may be considered, although intraoperative therapy with an intravenous hypotensive agent without pretreatment is probably adequate in most cases. In patients in whom oral secretions are foreseen to be or previously have been a problem, atropine or glycopyrrolate can be administered intravenously in the angiography suite.

Prophylaxis for cerebral ischemia is in a state of development. We routinely have placed patients on oral

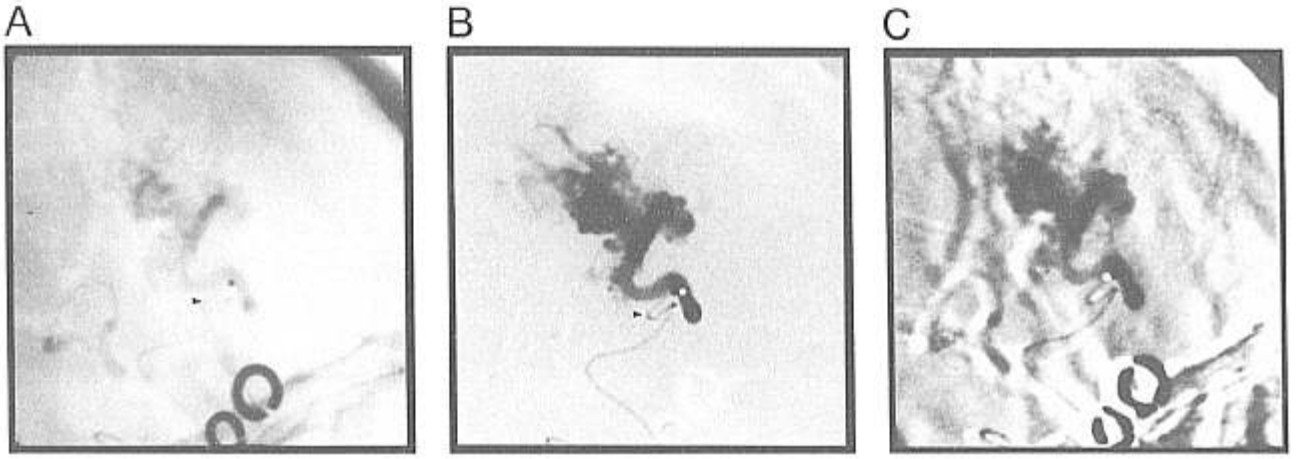

Fig. 5. *(A)* **An example of superselective contrast injection through the microcatheter (small arrow), without bone subtraction.** *(B)* **The same view with bone subtraction.** *(C)* **An example of how patient motion profoundly degrades the image.**

nimodipine in cases that entail an appreciable risk of cerebral ischemia. Because of the data supporting the use of nimodipine for the treatment of cerebral ischemia in the setting of ischemic stroke[41] and subarachnoid hemorrhage,[78,88,92] coupled with the lack of impressive side-effects,[119,133] our personal bias is to treat expectantly with nimodipine. Nimodipine also is thought to lessen the incidence of traumatic vessel spasm during catheter passage; nifedipine is used by some for this purpose.[87] The above notwithstanding, use of nimodipine for such prophylactic purposes has not been documented to be effective in randomized, controlled studies.

Other authors have described additional considerations for premedication, including corticosteroids, anticonvulsants, aspirin, and antibiotics.[8]

Room Preparation. Ideally, the INR suite should be equipped for anesthetic care exactly as a standard operating room. An example is shown in figure 6. Suction, gas evacuation, oxygen, and nitrous oxide should be available from wall outlets. Dedicated 20-A power lines, including emergency circuits, should be available. A dedicated phone line for the anesthesia team for laboratory communication and management of neurologic catastrophes is essential. A refrigerator for drugs should be in the room. Emergency equipment for cardiopulmonary resuscitation, including defibrillator and materials for surgical airway access, should be immediately available. Ideally, the anesthesia machine should have the ability to provide carbon dioxide gas (see deliberate hypercapnia). Adequate spotlighting should be available to maintain the anesthesia record and observe monitors, as the room lights often are dimmed for viewing of the fluoroscopy screens.

Many angiography suites have doors that automatically switch off fluoroscopy when opened. This can be detrimental in the event of emergencies (*i.e.*, if, during a critical manipulation of the intracerebral catheters, someone enters the room). An entry "maze" that allows room access at all times but still provides radiation protection to those outside of the room is preferable (fig. 6).

Routine equipment and drugs to have at hand are listed in table 5. Because the risk of intracranial hemorrhage or vascular occlusion is ever present, airway, intubation, and induction materials must be prepared for immediate use and remain near the head of the table. Long or extension tubing from the anesthesia circuit is desirable.

Conduct of Anesthesia

Patient Positioning. Because the procedures can take many hours, having the patient as comfortable as possible before beginning sedation is essential. A comfortable air or foam mattress and some type of device for good head and neck positioning are needed. After the femoral introducer sheath has been placed, a pillow can be placed under the patient's knees to obtain a modest amount of flexion to possibly improve tolerance to prolonged periods of lying in a supine position. No amount of conscious sedation can substitute for careful patient positioning. Because patients may return for multiple treatments, continued patient acceptance is important. Because the

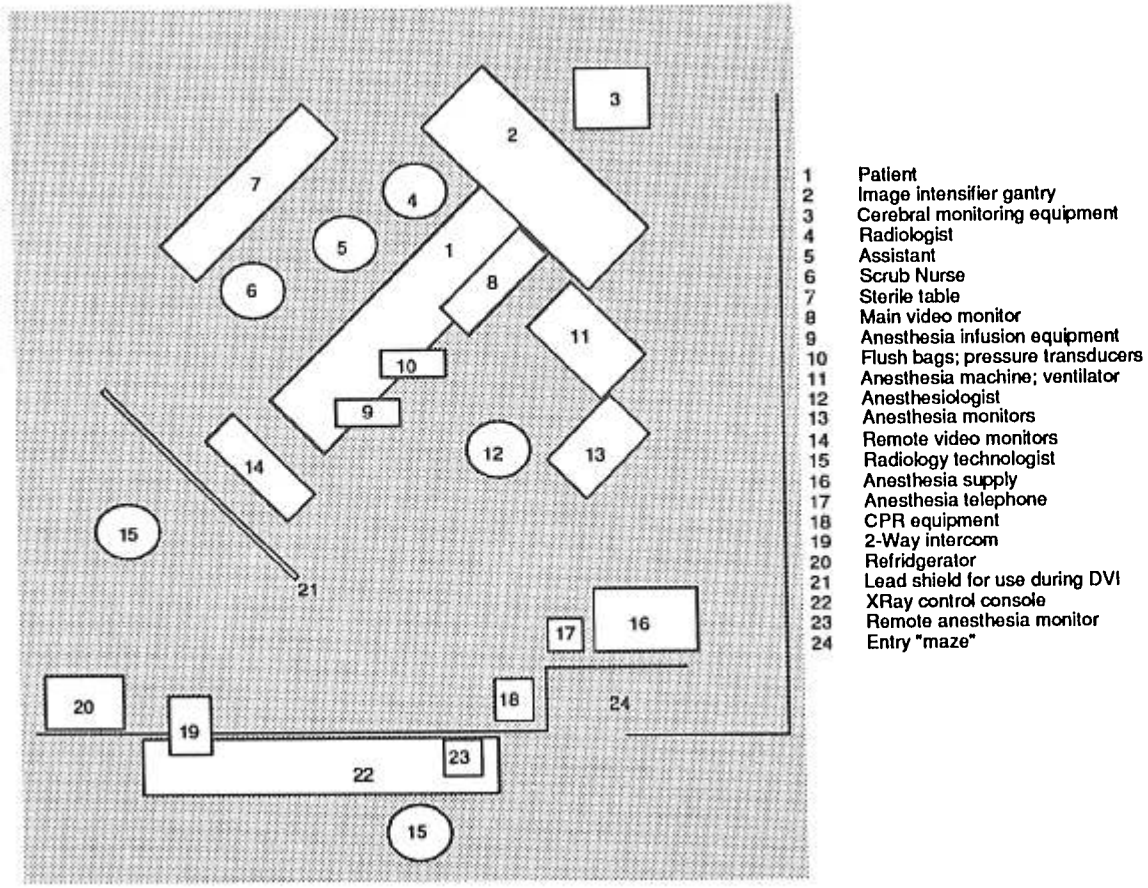

Fig. 6. An idealized example of the interventional neuroradiology (INR) suite and the important components.

head position needs to be maintained constantly, a headrest that discourages movement or paper tape across the patient's forehead is used as a "reminder". Use of rigid fixation should be avoided, because it might increase the likelihood of aspiration if emesis occurs.

Intravenous Vascular Access. For major cases, two intravenous lines should be available, one at least 18-G or greater (in adults). To maximize the distance between the fluoroscopy unit and the anesthetist, intravenous lines are prepared with two anesthesia-extension tubings. In one line, a stopcock or infusion port near the patient can be used for continuous drug infusions. The other line can be used for bolus injections through a port furthest from the image intensifier during fluoroscopy to minimize radiation exposure. When the patient is draped with arms restrained and advanced toward the image intensifier, access to intravenous sites is difficult. Therefore, anesthetics and vasoactive drugs should be in-line and ready to infuse before the patient is moved into final position, and all lines should be labeled clearly.

Arterial Pressure Monitoring. We use direct transduction of arterial pressure in any intracranial or spinal cord procedure: It certainly is indicated whenever the likelihood exists for manipulation of systemic pressure with vasoactive agents, in procedures involving the posterior fossa or upper cervical cord, or when there are mitigating medical considerations. This poses little if any additional risk to the patient, because the arterial system is cannulated as part of the treatment. For the typical intracranial procedure, three arterial pressures are monitored easily, and a pressure measurement setup is shown in figure 7. Pressure transducers and access stopcocks for blood withdrawal and zeroing are mounted, depending on local preferences, either on the sterile field or toward the anesthesia team. The advantage of having the stopcocks and transducers on the field is that the radiology team assumes the care of the various connections as part of their set-up and the like-

Table 5. Anesthesia Setup: Drugs and Equipment Recommended for Typical Conscious-sedation INR Cases

Equipment
Infusion pumps
Portable monitor for transport
Oxygen tank for transport
Laryngoscopy and airway supplies
Source of suction
Anesthesia machine with ventilator
Routine drugs
Midazolam
Fentanyl
Droperidol
Propofol for bolus and infusion
Heparin
Phenylephrine for bolus and infusion
Esmolol for infusion
Labetolol
Emergency drugs
Protamine
Thiopental
Succinylcholine
Nondepolarizing muscle relaxation
Atropine
Lidocaine
Ephedrine
Mannitol

lihood of inadvertently flushing or injecting the wrong catheter is reduced greatly. The disadvantage is that the anesthesia team cannot draw blood samples. We opt for the former.

The first pressure is femoral artery pressure. Although some institutions also perform radial artery catheterizations, the femoral artery introducer sheath is used easily as the "real-time" monitor of arterial pressure. This spares the patient from radial artery cannulation. A disadvantage is that it consistently underestimates the systolic pressure and overestimates the diastolic pressure, because of the coaxial catheter passing through it. This effect can be minimized by using a femoral sheath ½-French larger than will accommodate the coaxial catheter. However, the mean pressures are reliable and can be used to safely monitor the induction of either hyper- or hypotension. In addition, the disparity between femoral artery values can be compared with the readings of the blood pressure cuff. The femoral catheter is flushed continually with an intraflow device with 3 ml/h heparinized saline, which does not appreciably influence the mean pressure recording.

The second pressure is derived from the coaxial catheter in the carotid or the vertebral artery. There are several reasons for monitoring this pressure. Thrombus formation and vascular spasm at the catheter tip or migration of the catheter can be diagnosed by damping of the waveform. In addition, leaks in the introducer collar for the superselective catheter appear as a loss of pressure in the coaxial tracing. A high volume (100 ml/h) of continuous heparinized flush is passed through the coaxial tip to discourage thrombus formation (this infusion characteristically results in a 20-mmHg increase in the coaxial pressure and should be turned off for quantitative readings). A flush system malfunction can be detected readily by a change in or loss of the waveform.

Finally, the pressure at the tip of the superselective or balloon catheter can be monitored. This is useful during intracranial AVM embolization (see therapeutic embolization of intracranial arteriovenous malformations) and for measuring "stump" pressures during balloon occlusions (see carotid test occlusion and therapeutic carotid occlusion). The use of microcatheters for mean pressure measurements has been validated by Duckwiler *et al.*[28]

Other Systemic Monitoring. Other monitors should include 5-lead electrocardiogram (ideally with automated ST-segment trending) and automatic blood pressure cuff. In patients at risk for myocardial ischemia, a baseline recording of the electrocardiogram can be helpful for later comparisons during hemodynamic manipulation. A pulse oximeter probe is placed on the great toe of the leg that will receive the femoral catheters. This can give an early warning of femoral artery obstruction or distal thromboembolism. It also is useful when the femoral sheath must be removed and the site compressed for hemostasis, particularly in smaller children where overly vigorous compression can lead to permanent occlusion of the vessel.

Oxygen (2–4 l/min) is given by nasal cannula with a plastic catheter in place to monitor the partial pressure of end-tidal carbon dioxide ($P_{ET_{CO_2}}$). In our practice, this is accomplished with a "suction" type end-tidal monitor (Ohmeda RGM) and a truncated 14-G catheter placed in one of the prongs of the nasal cannula. For the spontaneously breathing patient, an indicator of respiratory rate is recommended if $P_{ET_{CO_2}}$ is not available and would be useful for detecting abnormal respiratory patterns during procedures involving the posterior fossa. Peripheral temperature can be monitored in a number of ways, such as with skin sensors or an axillary probe. In our experience, tympanic temperatures correlate best with brain temperatures.[118] Shivering is a troublesome problem because it results in patient motion and imaging degradation (see materials for embolization or infusion),

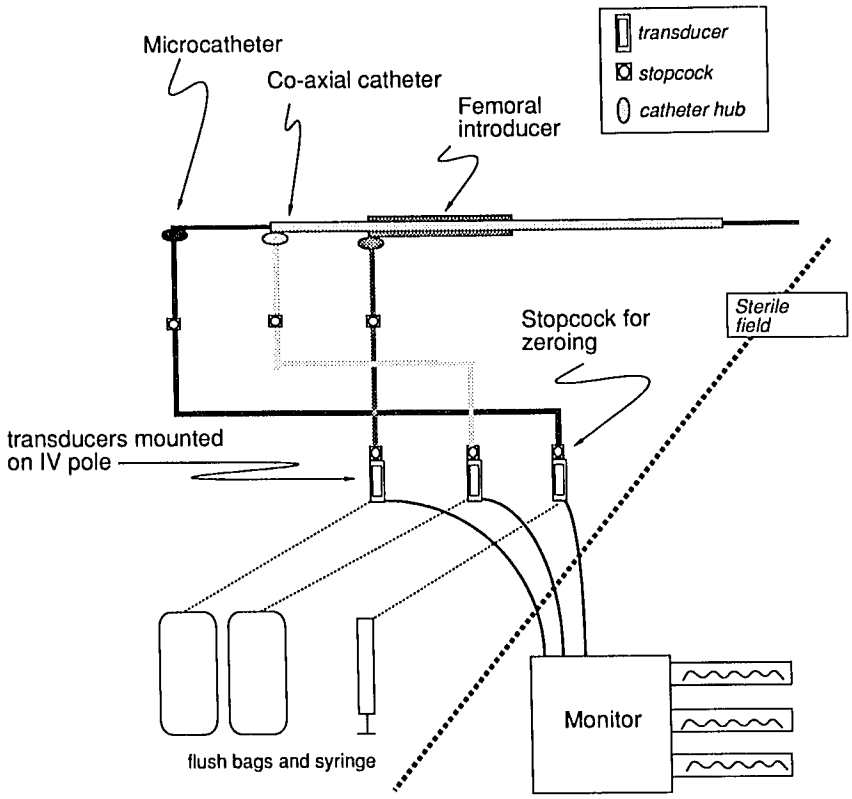

Fig. 7. Schematic drawing of pressure monitoring and continuous flush system.

and every effort should be made to keep the patient's temperature near normal (except in the case of a neurologic catastrophe, table 6).

All patients undergoing transfemoral procedures should receive bladder catheters to assist in fluid management, as well as to provide patient comfort. A large volume of heparinized flush solution may be necessary over the course of the procedure. Radiographic contrast is an osmotic diuretic and administration of diuretics such as mannitol or furosemide may be required.

After the monitors have been placed, sufficient slack is needed on all monitoring lines, intravenous lines, and airway connections to advance the patient toward the image intensifier. The timing and amount of contrast material administered should be noted by the anesthesia team.

When the patient's condition warrants placement of a central venous or pulmonary artery catheter, central catheters can be positioned by taking advantage of fluoroscopy. Similarly, the tracheal tube position for general anesthesia cases is verified easily by the fluoroscopy of the chest during passage of the coaxial catheters.

Central Nervous System Monitoring. During many procedures, the neurologic examination provides adequate monitoring of central nervous system integrity, as the INR team will follow the neurologic examination as an index of distal ischemia. Adjuncts, especially useful during general anesthesia or planned proximal occlusions, include electroencephalogram,[9,98] somatosensory[10] and motor-evoked potentials,# transcranial Doppler ultrasound (TCD),[43] and 133-Xenon (133 = Xe) cerebral blood-flow (CBF) monitoring.[4]

Other methods of determining the cerebral hemodynamic effect of proximal carotid or vertebral occlusion that can be used during the period of anesthesia care include CBF measurement with stable Xenon (Xe) computerized axial tomography scanning or single-photon emission computerized tomography (SPECT)[32,79,80,143] (see carotid test occlusion and therapeutic carotid occlusion). There is still debate as to which of the physiologic imaging procedures yields the most appropriate information for a given clinical setting.[93]

Anesthetic Techniques.

Conscious Sedation. Primary goals of anesthetic choice for conscious sedation include alleviating pain

Adams DC: Personal communication, 1993.

Table 6. Management of Neurologic Catastrophes

Initial resuscitation
Communicate with radiologists
Call for assistance
Secure the airway and hyperventilate with 100% O_2
Determine if problem is hemorrhagic or occlusive
Hemorrhagic: immediate heparin reversal (1 mg protamine for each 100 units heparin given) and low normal pressure
Occlusive: deliberate hypertension, titrated to neurologic examination, angiography, or physiologic imaging studies (*e.g.*, TCD, CBF)
Further resuscitation
Head up 15° in neutral position
Titrate ventilation to a Pa_{CO_2} of 26–28 mmHg
0.5 g/kg mannitol, rapid intravenous infusion
Anticonvulsants: dilantin (give slowly, 50 mg/min) and phenobarbitol
Titrate thiopental infusion to electroencephalogram burst suppression
Allow body temperature to fall as quickly as possible to 33–34° C
Consider dexamethasone 10 mg*

These are only general recommendations, and drug doses must be adapted to specific clinical situations and in accordance with a patient's preexisting medical condition. In some cases of asymptomatic or minor vessel puncture or occlusion, less aggressive management may be appropriate.

* Steroids are of dubious value in the treatment of focal cerebral ischemia,[101] but may have a place for reducing mass effect from a hemorrhage, if clinically appropriate.

TCD = transcranial Doppler; CBF = cerebral blood flow.

or discomfort, anxiolysis, and patient immobility, but at the same time one must allow for a rapid decrease in the level of sedation when neurologic testing is required.

The procedures generally are not painful, exceptions being sclerotherapy and chemotherapy. There is an element of pain (frequently described as burning) associated with injection of contrast into the cerebral arteries and by distention or traction on them. Discomfort from long periods of lying still, however, is the rule. Insertion of the bladder catheter and, to a lesser extent, the initial groin puncture for the femoral cannulation are two notable causes of discomfort.

The procedure also is stressful psychologically. There is a risk of serious stroke or death. This may be particularly important in a patient who already has suffered a preoperative hemorrhage or stroke.

Movement by the patient will decrease the usefulness of the roadmapping techniques described above (see imaging technology) and could result in a complication (fig. 5). For example, the catheter could penetrate a vessel wall and still appear to reside within the lumen.

Anesthetic agents are selected to meet the above goals. Our primary approach to conscious sedation is to establish a base of neuroleptanesthesia by titration of 2–4 μg/kg fentanyl, 2.5–5 mg droperidol, and 3–5 mg midazolam after intravenous access and oxygen administration have been established. The goal of this initial drug titration is to render the patient immobile and generally unaware of the surroundings, but still arousable with adequate spontaneous ventilation. A small bolus of propofol can be useful just as a (well lubricated) bladder catheter is passed in males.

When the patient is in final position and draping begins, a propofol infusion is started at very low levels (10–20 $\mu g \cdot kg \cdot min$) and then titrated slowly to result in an unconscious patient with a patent airway. The use of propofol gives the anesthetist some degree of control when a rapid return to consciousness is needed for neurologic assessment.

All patients should receive supplemental oxygen during conscious sedation techniques. Placement of nasopharyngeal airways can cause troublesome bleeding in anticoagulated patients and generally is avoided. If the need for a nasopharyngeal airway is expected, it is prudent to place it before anticoagulation begins and observe meticulous hemostasis.

We feel that droperidol is an excellent addition to the neurolept technique because of its antiemetic effect and alpha-adrenergic blockade and our impression that it renders a calmer, more motionless patient than do benzodiazepines alone. Postprocedure dysphoria is a theoretic concern, and dopadrenergic blockade can result in extrapyramidal symptoms in normal patients as well as in those with Parkinson's disease.[90] In the present series, we had one patient with a basal ganglia AVM who developed mild dyskinetic movements after droperidol administration that resolved within several hours after the procedure was finished.

A variety of other sedation regimens and variations are certainly possible[12,34,35,44,87] and must be based on the experience of the practitioner and the goals of anesthetic management for a particular procedure. In our experience and in that of others, chloral hydrate and ketamine-based techniques have little to offer.[35] A predominantly propofol-based technique is possible and intuitively appealing. Our enthusiasm for this method, however, quickly waned because of an unacceptably high incidence of upper airway obstruction, which led to nasopharyngeal airway placement in an anticoagulated patient and enough epistaxis to complicate airway control. In addition, troublesome behavioral disinhibition seemed to occur frequently.

General Anesthesia with Tracheal Intubation. Small children and uncooperative adult patients require general anesthesia with tracheal intubation. Although deep intravenous anesthesia without tracheal intubation has been proposed in such patients,[44] prolonged periods on the procedure table may result in patient motion. General anesthesia is used also for certain specific procedures such as aneurysm ablation, sclerotherapy, and certain cases of chemotherapy. Of the 170 patients in our series, 17 (10%) were pediatric, ages ranging from 3 to 17 yr (mean 10 ± 4 yr). Children between 12 and 15 usually will tolerate conscious sedation if the procedure is explained carefully to them and their parents preoperatively and they are well coached on what to expect during treatment.

There is no evidence or suggestion that general anesthesia with tracheal intubation should differ in the INR suite from its usual intraoperative application, be it for adult or pediatric cases. Anesthetic choice and cerebral protection during neurosurgical procedures is reviewed extensively elsewhere.[27] A theoretic argument could be made for eschewing the use of nitrous oxide because of the possibility of introducing air emboli into the cerebral circulation, but there are no data to support this theory.

When general anesthesia is used, it is frequently to obtain a motionless patient to improve the quality of the images. This is especially pertinent to INR treatment of spinal pathology, in which sometimes exhaustive multileveled angiography must be performed. Because chest excursion during positive-pressure ventilation can interfere with roadmapping, radiologists frequently request apnea for DSA in spinal procedures. An effective alternative to apnea is to adjust the ventilator to a relatively rapid rate and small tidal volume. Adequate gas exchange can be maintained during brief periods without degrading the image quality by excessive chest excursion. If available, this may be an ideal application for high-frequency jet ventilation.

Anticoagulation. Careful management of coagulation is required to prevent thromboembolic complications during and after the procedures, although algorithms for anticoagulation remain controversial.[34,95,131] It certainly is indicated whenever permanent or test occlusion is performed. Distal thromboembolism and clot propagation can be major sources of complications after major vascular occlusion.

Whether heparinization should be used for every case of intracranial catheterization is not clear. Some would argue that anticoagulation increases the risk of intracranial hemorrhage. We feel strongly that heparinization should be performed routinely during any superselective catheterization. In addition to thrombus formation from foreign bodies in the circulation, a considerable amount of thrombogenic, endothelial damage may be done by the passage of the superselective catheter.

After placement of the femoral introducer catheter, a baseline activated clotting time (ACT) is obtained. Heparin (5,000 U/70 kg) is given, and another ACT is checked. The target is at least 2–3 times the baseline value. ACT is monitored at least every hour. If an ACT is not drawn on schedule because of some extenuating circumstance, heparin (2,000 U) is given empirically every hour. The risk of overdosing the patient on heparin in this fashion is minimal compared to the risk of inadvertent thrombus formation. Heparin dose and ACT can be entered in a graphic manner on the anesthesia record so that it is easier to follow trends at a glance.

In our practice, heparin is continued through the first postprocedure night. The rationale for postprocedure anticoagulation is to protect against both the thrombogenic effects of endothelial trauma and the inherently thrombogenic nature of the materials instilled, such as glue or coils, which can cause retrograde thrombosis in embolized vessels. A period of 24 h is felt to be sufficient for a "pseudo-endothelial" layer to form and prevent either retrograde or antegrade thrombus formation that may propagate along the arterial tree (and the venous system in AVMs) with potentially disastrous results. The heparin effect then is allowed to wane on the first postprocedure day. Because the patient is under the effects of heparin, the large introducer sheath in the groin is left in place the first postprocedure night and removed before discharge of the patient to the floor on the following morning.

Sometimes the procedure can be aborted before any foreign material is deposited or significant endothelial trauma has taken place. For example, superselective angiography or provocative testing may reveal that a lesion is not amenable to treatment (see superselective anesthesia functional examination). In this event, heparin is reversed electively with protamine at the conclusion of the procedure and the femoral catheter is removed in the angiography suite.

An occasional patient may be refractory to attempts to obtain adequate anticoagulation. A switch from bovine to porcine heparin or vice versa may be of use. If antithrombin III deficiency is suspected, administration of fresh-frozen plasma may be necessary.

Other Laboratory Tests to Monitor. A baseline ABG at the time of the first ACT is useful to determine a baseline Pa_{O_2}–Sa_{O_2} gradient as well as the Pa_{CO_2}–PET_{CO_2} gradient. Although the correlation between Pa_{CO_2} and PET_{CO_2} is usually good during general anesthesia,[142] monitoring PET_{CO_2} through the nasal cannula is less precise and the discrepancy between end-tidal and arterial values is greater. In a subset of 33 patients anesthetized with the neurolept and propofol technique described previously (see conscious sedation; who happened to be enrolled in clinical studies), the mean ± SD PET_{CO_2} was 32 ± 9 mmHg when Pa_{CO_2} was 46 ± 7 mmHg, with an average gradient of 14 ± 9 mmHg. There was no relationship to age or type of procedure.

The patients are given large quantities of fluid and dye and can diurese considerably, and a baseline hematocrit determination is helpful. The issue of optimal hematocrit in the brain-injured patient is controversial,[65,125] especially in the setting of aneurysmal subarachnoid hemorrhage, and is beyond the scope of this review. Based on available evidence, both extremes of hemodilution and hemoconcentration should be avoided. Because intravenous ethanol administration can result in hypoglycemia,[106] monitoring blood glucose can be useful during sclerotherapy (see sclerotherapy of venous angiomas).

Superselective Anesthesia Functional Examination. SAFE is performed to determine, before therapeutic embolization, if the tip of the catheter has been inadvertently placed proximally to the origin of nutritive vessels to eloquent regions, either in the brain or spinal cord.[91,96,98] Such testing is really a variation and extension of the Wada and Rasmussen test,[132] in which amobarbital is injected into the internal carotid artery to determine hemispheric dominance and language function.

Before testing, the level of sedation should be decreased, *e.g.*, by stopping the propofol infusion. In rare instances (to be avoided), it may be necessary to use naloxone or flumazenil to antagonize other intravenous agents. A baseline, focused neurologic examination under residual light sedation is performed by the INR team. Sodium amobarbital (30 mg) or lidocaine (30 mg), mixed with contrast, then is given *via* the superselective catheter, and an angiogram of the distribution of the drug/contrast mixture is obtained. The doses and volume (0.5–3 ml) of agent can be altered to fit the clinical situation. Sodium amobarbital is used for investigating gray matter areas. Lidocaine can be used to evaluate the integrity of white matter tracts, especially in the spinal cord.[25,57] An injection of lidocaine in the brain can result in seizures, particularly when injected in areas such as motor strip. In addition to being disquieting to the patient and increasing the risk of aspiration, seizure activity can result in a transient focal neurologic deficit. A postictal paralysis, for example, can confuse interpretation of the test. For this reason, the barbiturate usually is given first, followed by lidocaine. If the amobarbital is negative, it can protect against cortical seizure but it will not interfere significantly with the assessment of lidocaine's effect on white-matter tracts. Not all authors agree on the use of lidocaine for intracerebral testing.[97]

After drug injection, the neurologic examination is repeated. Attention is directed to areas at risk as well as "quiet areas", in which a deficit might be missed if only a motor or sensory examination is performed, such as that of lesions involving dominant parietal lobe.

SAFE generally is reliable, but false-positive tests can occur with overinjection and reflux into normal vessels. Underinjection, or a "sump" effect from an AVM, can lead to false-negative results.[7] Systemic recirculation of the anesthetic can result, in some cases, in generalized sedation. Rauch *et al.*[97] described the use of electroencephalogram monitoring, coupled with a clinical examination, to enhance the sensitivity of SAFE.

Deliberate Hypotension. The two primary indications for elective deliberate hypotension are to slow the flow in an AVM feeding artery before the injection of glue and to test the cerebrovascular reserve in patients undergoing carotid occlusion. In most cases, the level of sedation is decreased so that neurologic examinations can be followed during the period of deliberate hypotension. In awake patients, nausea and vomiting can be a major problem. It is for this reason that droperidol is an attractive choice as part of the sedative regime. An additional dose of droperidol (1.25 mg) can be given for antiemesis just before the initiation of hypotension (which usually begins at least 2 h after the initial dose). Before beginning hypotension, one should confirm that the patient is fully oxygenated and the airway is unobstructed.

Most of the AVM patients treated are relatively young and fit. Most importantly, they are not under general anesthesia, and the adjunctive hypotensive effect of general anesthesia is absent during conscious sedation. Therefore, it may be considerably more challenging to induce hypotension in this setting: Sometimes surprisingly large doses of hypotensive agents are necessary.

Our first-line agent is usually esmolol, given as a 1 mg/kg bolus and titrated to target systemic blood pres-

sure at an infusion rate beginning at 0.5 mg·kg·min. High levels of infusion often are needed, and boluses of labetolol (≈50–100 mg) are useful as an adjunct. Adrenergic blockers have the advantage of not directly affecting cerebral blood flow[103] and have the theoretic advantage of shifting the autoregulatory curve to the left.[36] Trimethaphan probably also shares such an effect.[135] (A disadvantage is that the large doses needed for awake patients frequently cause pupillary dilation, and this may confound the neurologic examination.)

Sodium nitroprusside and nitroglycerin have been described for use during INR procedures.[87] We tend to avoid using these cerebral vasodilators, including dihydralazine, because of the theoretic potential for cerebral steal unless there is some specific indication (*e.g.*, coronary artery disease for nitroglycerin). The greatest disadvantage of nitroglycerin and sodium nitroprusside is that it is easy to overshoot and render the patient momentarily severely hypotensive. Although this can be treated without incident in the patient under general anesthesia with tracheal intubation, the onset of hypotension-induced emesis and nausea in an awake patient can be disastrous in the INR setting from several standpoints. It can decrease the total amount of time available to the team for the procedure because of continued discomfort, and it can interfere with angiographic visualization because of motion artifact. The nausea can be confused with acute intracranial hypertension from vascular perforation. Retching can cause migration of the intracranial catheters from the desired locations, further endothelial damage, or produce vessel perforation.

Blood Pressure Augmentation (Deliberate Hypertension). Not infrequently, a situation will arise in which the patient will experience cerebral ischemia from either a planned or inadvertent vascular occlusion. As reviewed recently by Young and Cole,[138] the systemic blood pressure should be increased to drive adequate flow *via* collaterals to the area of ischemia as a temporizing measure. The primary routes of collateral circulation are the Willisian channels (anterior communicating artery and posterior communicating artery and the ophthalmic *via* the external carotid artery).

The second main recourse for collateral flow in the hemispheres is the surface connections between pial arteries that bridge major arterial territories, e.g., the boundaries between anterior, middle, and posterior cerebral artery territories. These connections are called by various names. *Pial-to-pial anastomoses* or *collaterals* seem to be the most logical, but they also are called *leptomeningeal pathways.*[20] These pathways may protect the so-called *borderzones* or *watershed* areas between vascular territories. There is a considerable amount of confusion in terminology in this domain.[11] Physiologically, a more precise term might be "equal-pressure boundary",[127] *i.e.*, where, under normal circumstances, pial flow does not cross collateral pathways into an adjacent territory because the pressure on either side of this distal territorial boundary is equal. There is considerable variation in the anatomic location of these boundaries, and they can change during the course of treatment if the vascular architecture is altered, *e.g.*, after multiple AVM embolizations.

Collateral pathways are most efficacious during chronic ischemia, when they can enlarge gradually over time. Acutely, it is frequently necessary to augment blood pressure to drive flow across them effectively. Absence of adequate collateral pathways, especially in the Circle of Willis, is a normal anatomic variant, so deliberate hypertension is not guaranteed to succeed. An example of deliberate hypertension to treat acute cerebral ischemia by improving pial-to-pial collateral flow across a watershed is shown in figure 8.

Our first-line agent is a phenylephrine (≈1 μg/kg) bolus followed by a titrated infusion to increase the pressure to levels that reverse the neurologic deficit, empirically, 30–40% above baseline. The electrocardiogram and ST-segment monitor should be inspected carefully for signs of myocardial ischemia. Blood pressure goals must be tempered by the patient's preexisting medical status. Based on the best available evidence, deliberate hypertension in the face of symptomatic cerebral ischemia from vascular occlusion during AVM embolization should not be avoided because of fear of rupturing the malformation.[120]

If the heart rate is very low to start, *e.g.*, due to preoperative beta blockade or sinus-node disease, an alternative choice would be dopamine, with or without phenylephrine. In our experience, the use of dopamine alone to induce hypertension frequently results in unacceptable tachycardia.

Deliberate Hypercapnia. Venous malformations of the face or dural fistulas have the potential to drain into intracerebral veins or sinuses. During general anesthesia, hypercapnia is desirable in circumstances in which agents are injected into the venous circulation. By increasing the Pa_{CO_2} to 50–60 mmHg, one can cause the cerebral venous outflow to greatly exceed extracranial venous outflow and the pressure gradient to favor movement of either a sclerosing agent, chemotherapeutic agent, or glue away from vital intracranial

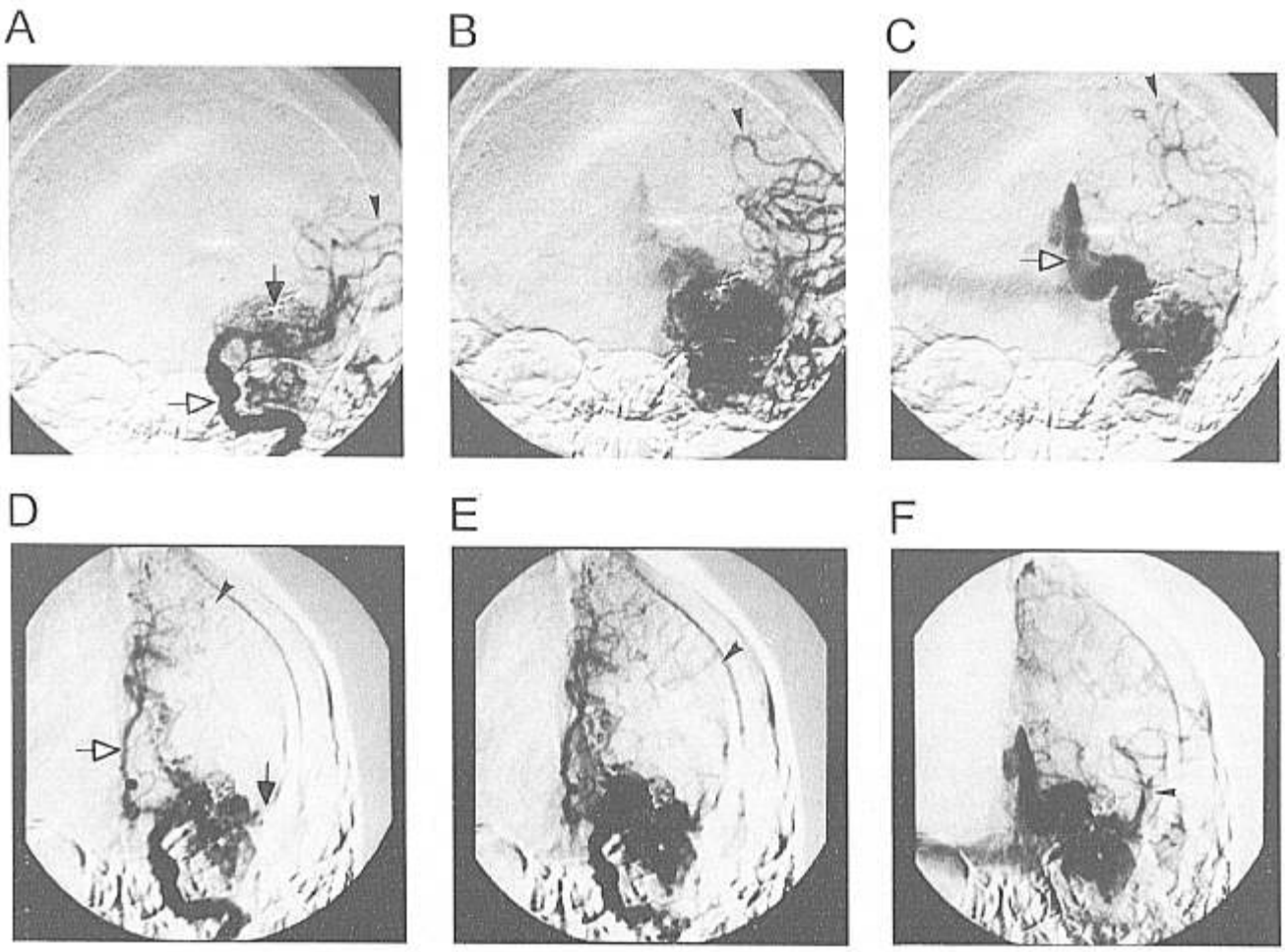

Fig. 8. An example of how deliberate hypertension can be used as a temporizing measure to treat acute proximal occlusive ischemia. This 54-yr-old woman underwent glue embolization of a temporal arteriovenous malformation (AVM). *(A–C)* Anterior–posterior views of contrast injection demonstrate the passage of dye through the ipsilateral normal middle cerebral artery (MCA) and the AVM. *(A)* The open arrow indicates the internal carotid artery, just proximal to the origin of the MCA. The closed arrow indicates the nidus with n-butyl cyanoacrylate (NBCA) from a previous embolization. The arrowhead indicates normal filling of the distal MCA branches. *(B)* Several seconds later. The arrowhead indicates contrast moving distally out of the MCA territory. *(C)* The open arrow shows the draining vein, and the arrowhead indicates the borderzone of a normal MCA territory. After NBCA injection, reflux of glue around the catheter resulted in the tip becoming cemented to the vessel wall. Removal was attempted with gentle, intermittent traction. The patient complained of pain at one point, and there was a question of vascular rupture. The blood pressure was kept in the low to normal range, and the heparin was reversed with protamine. The patient was neurologically unchanged at this point. Shortly thereafter, profound hemiparesis and aphasia developed, and fluoroscopy demonstrated occlusion of the first (M1) portion of the MCA. Systemic blood pressure was increased 40–50% with phenylephrine, and the deficit improved dramatically. *(D–F)* Contrast injection during deliberate hypertension demonstrates how contrast now passes through the (previously unvisualized) ACA and, *via* pial-pial collaterals, retrograde into the ischemic MCA territory. *(D)* The open arrow shows contrast now filling the ipsilateral ACA, the closed arrow indicates the occluded MCA stem, and the arrowhead indicates contrast reaching the normally expected edge of the ACA borderzone. *(E)* Contrast is seen advancing into the MCA distribution retrogradely, finally filling the proximal MCA in *F*. Within an hour, the clot in the MCA spontaneously lysed and migrated distally, and antegrade filling of both the ACA and the MCA was observed (not shown). The patient's neurologic examination stabilized with very mild aphasia and hemiparesis. A small temporal infarct was visualized on post-procedure computed tomography at 24 h.

drainage pathways. Although actual pressure gradients have never been studied, increased intracranial outflow is readily demonstrable in clinical practice with angiography. Addition of carbon dioxide gas to the inspired gas mixture is the easiest and safest way to achieve hypercapnia. Airway collapse and atelectasis

can be prevented by the maintenance of adequate tidal volume. However, hypoventilation can be employed if carbon dioxide gas is not available; in this case, addition of positive end-expiratory pressure may be useful to maintain oxygenation.

Transport and Postprocedure Considerations. After intracranial or intraspinal procedures have been performed, patients spend the first postprocedure night in the intensive care unit. Complicated cases may first undergo computerized tomography or SPECT scan; only rarely is an emergent craniotomy indicated. Patients should receive oxygen during transport. Arterial blood pressure and, if the situation dictates, oxygen saturation should be monitored en route. Blood pressure control, either modest hypotension in the case of AVM embolization or deliberate hypertension in the patient with occlusive or vasospastic cerebrovascular disease, should be continued during transport.

Postprocedure nausea and vomiting can be caused by anesthetic agents or the large volumes of contrast agent. This general topic is reviewed elsewhere.[134] After procedures performed in the posterior fossa, small degrees of ischemia and swelling from contrast frequently result in symptomatic local brain swelling in the postprocedure period. In the more capacious supratentorial compartment, such minor swelling is rarely symptomatic. In the posterior fossa, this may be evident as delayed deficits or decreased sensorium during the course of the first evening after the procedure, particularly if CSF pathways have become obstructed. This eventuality should be factored into decisions regarding airway management.

Complications and Special Considerations

Management of Neurologic Catastrophes. Complications during instrumentation of the cerebral vasculature can be rapid and dramatic and require a multidisciplinary collaboration.[95] Having a well thought out plan for dealing with intracranial catastrophe can make the difference between an uneventful outcome and death. The catastrophe plan outline shown in table 6 is based on currently recommended approaches to the treatment of acute cerebral injury.[27,140]

If a neurologic catastrophe occurs, rapid and effective communication between the anesthesia and radiology teams is vital. The appropriate neurology and neurosurgical consultants should be contacted as soon as possible. The anesthetist should know enough about the nature and extent of the problem to treat it effectively. The primary responsibility of the anesthesia team is to secure the airway and preserve gas exchange. If tracheal intubation is necessary, a thiopental and relaxant induction should not be avoided because of the possibility of a transient decrease in perfusion pressure.

Simultaneous with airway maintenance, the first branch in the decision-making algorithm is for the anesthesiologist to communicate with the INR team and determine whether the problem is hemorrhagic or occlusive.

In the situation of vascular occlusion, a method to increase distal perfusion either by blood pressure augmentation with or without direct thrombolysis (see also thrombolysis of acute thromboembolic stroke) is the primary strategy. Note that thiopental probably will provide some degree of protection even after an occlusion.[104] An example of the use of deliberate hypertension in the setting of acute hemispheric ischemia from middle cerebral artery occlusion is shown in figure 8.

If the problem is hemorrhagic, *immediate* reversal of heparin is indicated. Protamine is given as rapidly as possible to reverse heparin without undue regard for the systemic blood pressure. The cardiac output need be only as high as is necessary to achieve reversal of heparin.** The dispatch with which heparin is reversed may very well be the critical step between a good and a poor outcome from the bleed.

As an emergency reversal dose, 1 mg protamine can be given for each 100 U heparin total dosage during the case. The ACT then can be used to fine-tune the final protamine dose. Blood pressure control requires second-to-second communication with the radiologist. While bleeding is occurring and during the reversal of heparin, the blood pressure should be kept as low as possible.†† In concert with securing the airway, thiopental should be considered as a first-line method of lowering blood pressure; it also will prevent seizure activity from acute subarachnoid hemorrhage. Once the bleeding has been controlled, especially if by temporary vascular occlusion, blood pressure should be kept as high as clinically appropriate after consulting with the INR team.

Bleeding catastrophes usually are heralded by headache, nausea, vomiting, and vascular pain related to

** Because of experience with cardiopulmonary bypass and postbypass left-ventricular dysfunction, there is a reflexive reluctance for most anesthesiologists to administer protamine rapidly enough.

†† These are recommendations based on clinical experience and intuition. Ideally, some index of cerebral perfusion will be used to optimize both cerebral blood flow and cerebral perfusion pressure.[33]

the area of perforation. The radiologist often can see the contrast extravasating seconds before the patient becomes symptomatic. In cases of vessel puncture, heparin reversal *before* the withdrawing of the offending wire or catheter back into the lumen of the vessel will keep the perforation partially blocked until hemostatic function is restored. Rupture or perforation of vessels is often treatable with glue, coils, or balloons. An example of vessel perforation and resultant symptomatic ICH is shown in figure 9.

If an episode of suspected contrast extravasation or vessel puncture turns out not to be a bleed, the patient can be reheparinized. If significant mass effect is present, the decision to intervene operatively can be undertaken after consultation with the other specialists involved.

Contrast Reactions. This subject is dealt with extensively in the literature, especially for the older ionic contrast agents.[4,5] Most important for modern INR is the use of low osmolality nonionic contrast agents such as iohexol, which in its usual application for INR is an iodine–salt concentration of 300 mg/ml. This corresponds to an osmolality of 672 mOsm/kg, as opposed to the older ionic agents, which have osmolalities of approximately 2,000 mOsm/kg.[5,6] Although fatal reactions probably occur at the same frequency with these agents as with ionic agents (on the order of 1: 100,000 exposures), nonionic agents have a lower incidence of mild and moderate reactions.[16,17,56,117]

Despite a controversy over the general use of nonionic agents and cost-effectiveness for radiologic imaging, for INR purposes, the lower osmotic activity allows for relatively generous use in single cerebral vessels. A single vascular pedicle may receive in the vicinity of 100–200 ml of contrast during the course of a procedure. This could not be accomplished with older ionic agents.

For patients with a history of reactions, pretreatment with steroids and antihistamines is recommended.[4,5] Prednisone (50 mg) given the evening before and the morning of the procedure, and diphenhydramine (50 mg) given intravenously before the beginning of the procedure is our current regimen.

To prevent renal complications, intraoperative fluid management should be aimed at maintaining euvolemia to offset the diuretic effect of the injected contrast. Maintaining an isotonic or slightly hypertonic state for neurosurgical patients[124] is generally not a problem, because contrast-induced diuresis usually encourages a hypertonic state. However, patients who have undergone diagnostic procedures in the week prior to an INR procedure are frequently quite volume-depleted and can be hemodynamically unstable.

Radiation Safety. As a potential risk to anesthesia personnel,[2] there are three sources of radiation exposure typically encountered from the imaging equipment: *direct* (from the x-ray tube), *leakage* (through the collimators' protective shielding), and *scattered* (reflected from the patient and the area surrounding the body part being imaged). A fundamental knowledge of radiation safety is essential for staff working in an environment such as the angiography suite. All personnel should wear lead aprons and thyroid shields and have exposure badges. Movable lead glass shields are positioned for the anesthesia team to stand or sit behind. Note that distal subtraction angiography (DSA) delivers considerably more radiation than does routine fluoroscopy, and personnel either should leave the room or stand behind lead barriers during DSA. A common error made by new staff is to turn their backs (which are usually unshielded) towards the x-ray source during anesthetic care of the patient.

Because the amount of exposure drops off proportionally to the square of the distance from the source of radiation (inverse square law), activity near the head of the patient should be kept at a minimum during fluoroscopy. For example, a person standing near the head of the table (and the radiation source) may receive a 100-fold greater radiation exposure than will one at the foot of the table. There must be effective communication between the anesthesia and radiology teams to take optimal care of the anesthetized or sedated patient and minimize staff exposure to ionizing radiation.

The annual recommended limit for occupational whole-body exposure is 5,000 mrems.[111] With proper precautions, the anesthesia team should be exposed to less than 0.1 mrem/h.

Synthesis and Comment. A major consideration for anesthetic management during INR is that while the patients receive intravenous sedative drugs, the routine and potential emergency management of these cases is much more interactive than a typical "monitored anesthesia care" case because of frequent changes in level of consciousness and manipulation of systemic arterial blood pressure. In addition, the nature of many of the potential complications require immediate intervention of the anesthesiologist. Although monitored anesthesia care is appropriate for a diagnostic procedure or a minor surgical procedure that is performed primarily with local anesthesia supplemented by minimal sedation, active manipulation of hemodynamics and

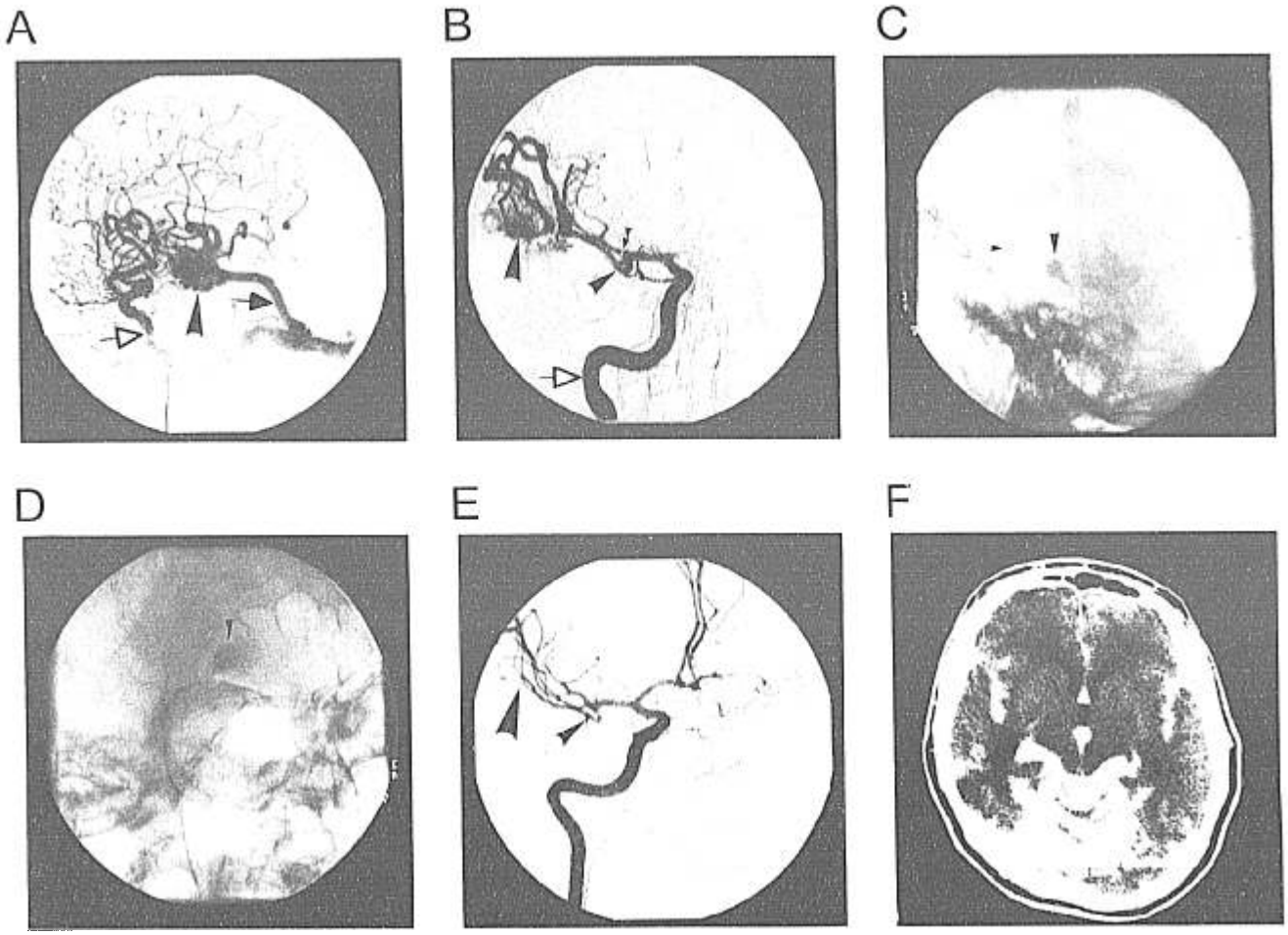

Fig. 9. The patient was a 50-yr-old man who originally presented with spontaneous intracranial hemorrhage (ICH). *(A)* A lateral carotid angiogram of a right posterior temporal arteriovenous malformation (AVM; arrowhead) fed by two middle cerebral artery (MCA) branches *via* the internal carotid artery (open arrow), with superficial drainage into the vein of Labbe (closed arrow). There is venous stenosis at junction with the sinus. *(B)* An anterioposterior internal carotid (open arrow) angiogram showing the AVM (large arrowhead at left). The microcatheter in superior division MCA (double arrowhead) and an MCA bifurcation aneurysm (smaller arrowhead) are indicated. During distal passage of the microcatheter, it perforated the MCA bifurcation aneurysm. The patient become acutely bradycardic (65 to 35 beats/min), hypertensive (MAP 90 to 140 mmHg), and comatose. *(C)* The microcatheter in the MCA (small arrowhead) and a mixture of contrast and blood in periambient cistern (larger arrowhead). *(D)* A more oblique view, again showing contrast and blood in periambient cistern (arrowhead). Anesthesia was induced immediately with thiopental and succinylcholine, the trachea was intubated, and modest hyperventilation was started. Protamine was given simultaneously with the induction sequence. The blood pressure target was 20–30% below the patient's baseline mean arterial pressure (MAP). When the thiopental effect waned after 10 min, the patient began to spontaneously move all four extremities. *(E)* The MCA bifurcation aneurysm no longer filling (small arrowhead) and slight spasm in the MCA branches (larger arrowhead). At this time, it was elected to place the patient in burst-suppression with thiopental and obtain a computerized tomography (CT) scan. *(F)* The postprocedure CT scan with contrast and blood in periambient cistern. The patient was brought to the intensive care unit, allowed to emerge from the thiopental, and extubated neurologically intact. He made an uneventful recovery and eventually underwent surgical resection without incident.

sensorium render a description more akin to "dynamic akinetic sedation-controlled hemodynamics". *Dynamic* refers to repeated lightening and deepening of sedation, *akinetic* stresses the importance of an immobile patient, and *controlled hemodynamics* refers to the physiologic trespass by the manipulation of blood pressure. What's in a name? The goals of the anesthetic technique are capsuled by such a denomination and may help establish the level of expectations and degree of interaction of the INR and anesthesia teams. Semantics aside, a challenging problem in orienting new staff to the INR environment is that the anesthetic care of

these patients may be potentially much more involved than in other, superficially similar, settings.

Specific Procedures

Therapeutic Embolization of Intracranial Arteriovenous Malformations

There has been considerable progress in the embolization of cerebral AVMs over the past several years.[21,38] There are two main types of AVMs to consider: parenchymal cerebral AVMs and dural fistulas. Most of the cases are the former, and dural fistulas will be discussed separately.

Typically, patients who present for embolization have large, complex AVMs composed of several discrete fistulas with multiple feeding arteries. The goal of the therapeutic embolization is to obliterate as many of the fistulas and their respective feeding arteries as possible. Although in rare cases INR treatment is aimed at total obliteration, embolization usually is used as an adjunct in preparation for surgery or radiotherapy.[129] Radiosurgery of AVMs remains a controversial issue.[108,116]

As a presurgical adjunct, embolization is thought to facilitate operative removal, with less bleeding. This has been suggested[58] but not convincingly demonstrated. Obliteration of deep feeders, in particular, can make surgery easier[75,115] and thereby should reduce the surgical risk. Staging‡‡ obliteration of arteriovenous shunts also theoretically allows the surrounding brain to accommodate to the alteration of hemodynamics and may prevent "normal perfusion pressure breakthrough".[112,113] Obliteration of high-flow feeders can be of benefit in patients with progressive neurologic deficits or intractable seizures, ostensibly by diminishing steal.[50] Approximately 10% of patients with AVMs harbor intracranial aneurysms; our general approach is to treat the symptomatic lesion first.[18] Because intranidal aneurysms appear to increase risk of spontaneous hemorrhage from AVMs,[74] obliteration of intranidal aneurysm during the initial embolization may decrease the rate of intercurrent hemorrhage during the course of treatment.

The main anesthetic goals are to render the patient comfortable and, if possible, unconscious during periods in which wakefulness is not necessary. During SAFE and neurologic examinations, the patient should be awake and responsive. N-butyl cyanoacrylate was the primary agent used in our series. Polyvinyl alcohol particles, coils, or silk thread are recanalized in days to weeks and used only as adjuncts for surgery planned within that period. Proximal occlusion with coils or sutures is not useful for long-term management[26,94] because it appears only temporarily to decrease flow through the fistula; it leaves the low-resistance arteriovenous connection intact and, therefore, capable of recruiting flow from other channels.[62] The most deleterious type of recruitment is from deep-perforating arteries, which make surgical resection especially difficult.[115]

Depending on the tortuosity of the vascular pathway and other technical considerations, it can be either very difficult or extremely easy for the INR team to place the catheter tip exactly where they want it. The ease of passing the superselective catheter will determine, in part, how many pedicles can be embolized on a given day. The patients, despite best efforts, usually will not tolerate more than 4–5 h of conscious sedation and remain still enough to allow satisfactory performance of the neuroimaging procedures.

As the superselective catheter is passed distally, pressure measurements can be made at the tip of the catheter. The pressure typically will decrease in a stepwise fashion as it is advanced distally.[28,37] When the catheter has been placed in position for potential glue injection, the level of sedation is decreased and a baseline neurologic examination is performed. SAFE then is performed (as described in superselective anesthesia functional examination). If this test is positive, *i.e.,* if a focal neurologic deficit is encountered, then the catheter is repositioned or embolization of that pedicle can be aborted. If negative, the glue or embolic material can be injected.

Once the superselective catheter is in optimal position, profound but tolerable systemic hypotension is induced while the radiologists prepare the glue for injection. Hypotension slows the flow through the fistula and provides for a more controlled deposition of embolic material, the glues in particular. Deliberate hypotension is used to achieve *flow arrest.* Ideally, there will be zero flow through the AVM, so the distribution of glue will be controlled totally by the radiologist injecting the glue. (Complications of glue injection are described at the end of this section.)

Adequate flow arrest appears to occur at a different systemic pressure for each patient. In fact, it seems that flow through the fistula remains relatively constant until a certain pressure is reached, when it drops off sharply.

‡‡ Staged embolization sometimes is referred to as fractionated embolization.

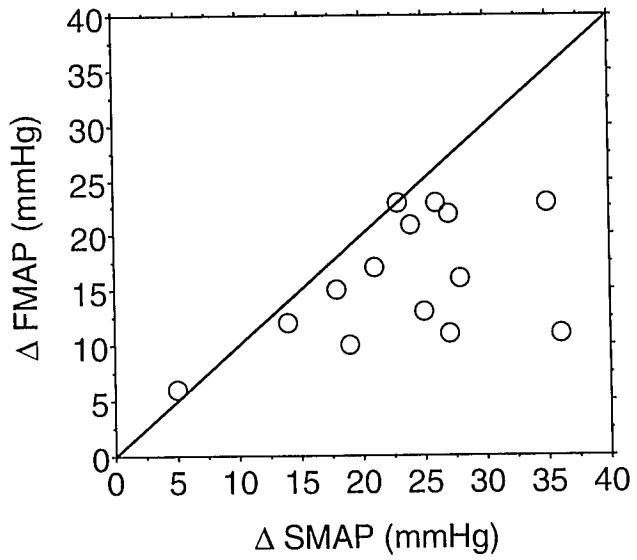

Fig. 10. There is a disparity between changes in the mean arterial pressure (MAP) measured in an arteriovenous malformation (AVM) feeding artery (FMAP) and changes in the systemic mean arterial pressure (SMAP). This data was taken from reference 141 using pressure recording methods described in this review and elsewhere.[37] This was a study of 14 patients during 15 embolization procedures. SMAP was increased with a phenylephrine infusion. The SMAP and FMAP were recorded simultaneously at the beginning and end of the phenylephrine challenge. The ΔFMAP is shown as a function of ΔSMAP with the line of identity indicated. Note that, for a given change in SMAP, the change in FMAP may be less. In some cases, there is no change in FMAP with a change in SMAP (see therapeutic embolization of intracranial arteriovenous malformations). Since the microcatheter placement in this series was rather proximal (to measure normal cortical regions using ^{133}Xe washout), the disparity between distal FMAP and SMAP may be even more pronounced.

As the pressure is lowered, the radiologist performs several contrast injections with fluoroscopy and visually determines the optimal systemic pressure to slow the flow through the fistula. Typically, we reduce systemic mean arterial pressure (MAP) to ≈50 mmHg, but the use of greater or lesser degrees of hypertension depends on the speed of the contrast transit through the fistula.

This subject has not been studied rigorously, and it is not clear whether systemic hypotension decreases shunt flow solely on the basis of a reduction in pressure, a limitation of total (and, in the case of an AVM, increased) flow to the brain, or some combination of both factors. In any event, in the presence of a cerebral fistula, reducing systemic pressure does not effect downstream feeding artery pressure to the same degree (fig. 10), and different patients may require different degrees of hypotension to adequately slow flow through the fistula for glue deposition.

Another technique used to achieve flow arrest is the placement of a balloon catheter *via* the other femoral artery. The balloon is positioned proximally to the superselective catheter to be used for gluing. Before glue injection, the balloon can be inflated to either slow or completely arrest distal flow. However, this necessitates the passage of another intracranial balloon catheter from the contralateral femoral artery and the attendant risks of vessel rupture from balloon overinflation.

Measurement of immediate postembolization pressures has been suggested as a means of following the course of hemodynamic changes[28,60] and predicting postprocedure complications,[1] as large increases in feeding-artery pressure appear to be associated with ICH. Additional studies are needed to further define the clinical use of such measurements. At the present time, unfortunately, immediate postembolization pressure measurements are practical only with thread, coils or polyvinyl alcohol particles. Available superselective catheters must be withdrawn immediately after glue injection (so they are not cemented into place).

Typically, the pressures in the proximal feeding artery are quite low, *i.e.*, 40–60% of MAP. The proximal por-

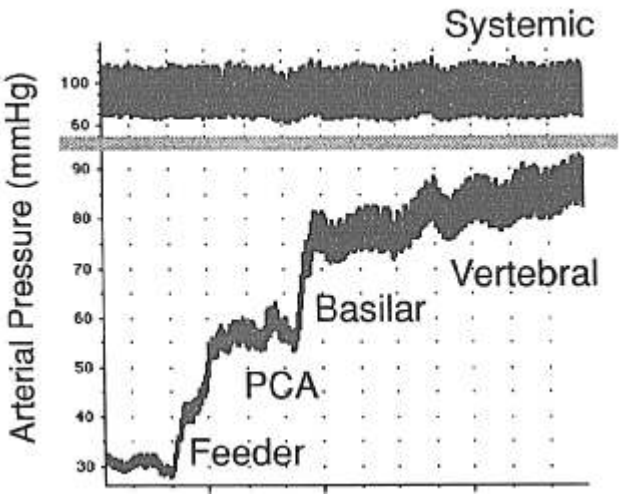

Fig. 11. Recording made of vascular pressures in a patient with an occipital arteriovenous malformation (AVM) fed by the posterior cerebral artery (PCA). Data taken from reference 37. As the catheter was withdrawn slowly from the distal feeding artery adjacent to the fistula, the pressure can be seen to increase, with step increases at branch points. The simultaneously recorded systemic arterial pressure is shown also. Note that the pressure in the proximal portion of the PCA, which irrigates a large area of normal, eloquent tissue, is modestly hypotensive in this asymptomatic patient. (We have proposed that the autoregulatory curve may be shifted to the left in such hypotensive vascular territories in some patients.[141]) (Adapted with permission from Young WL: Clinical Neuroscience Lectures. Munster, Cathenart, 1992.)

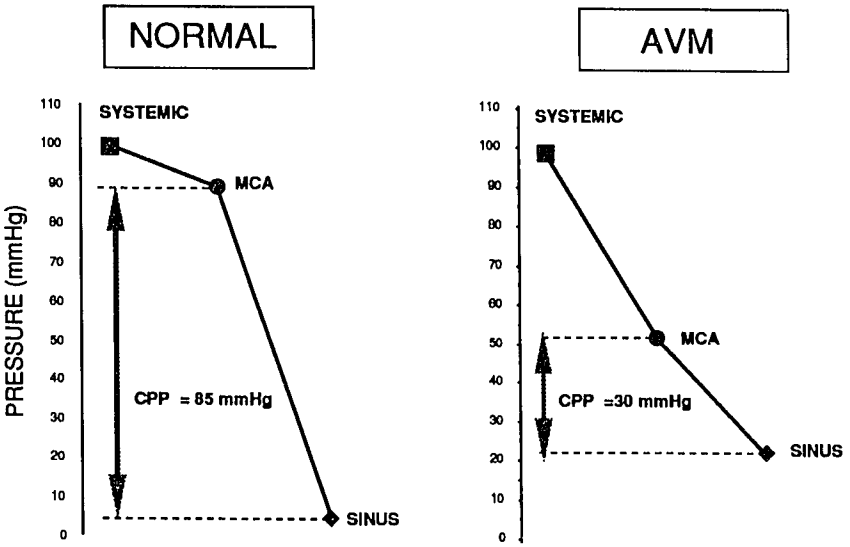

Fig. 12. Hypothetic depiction of pressure relationships between arterial and venous sides of the circulation in a normal and an arteriovenous malformation (AVM) hemisphere. The low arterial (*e.g.*, middle cerebral artery (MCA)) and high venous pressures (*e.g.*, sagittal sinus) routinely observed at the inflow and outflow of the AVM may be transmitted to functional brain regions, reducing the available cerebral perfusion pressure (CPP) to the adjacent normal vascular territories. The values represent an extreme case, as would be seen in very large AVMs, based on previously reported data.[6,37,52,83,112]

tion of the artery usually feeds large areas of functional eloquent brain, as shown in figure 11. The mean pressure in feeding arteries near the entry to the AVM nidus is usually 15–25% of MAP. Pressure is transmitted to the cerebral venous system and can pressurize normal venous drainage areas.[6,52,112] This is illustrated in figure 12, contrasted with the pressures expected in a normal hemisphere. There is not a direct relationship between AVM feeding artery and draining vein pressures: Outflow pressure from the nidus probably is determined partially by the architecture of the venous drainage.[61]

Because AVM feeding arteries supply variable degrees of normal vascular territories, abrupt restoration of normal systemic pressure to a chronically hypotensive vascular bed can overwhelm autoregulatory capacity and result in hemorrhage or swelling (normal perfusion pressure breakthrough). It is for this reason that the target range for posttreatment blood pressure is maintained strictly at 10–20% below the patient's normal ward blood pressure. An alternative approach to explain hemorrhage and swelling after AVM treatment has been termed "venous overload"[136] or "occlusive hyperemia",[3] emphasizing that venous outflow obstruction also can result in complications. The exact pathophysiology of hemodynamic complications after treatment of AVMs remains controversial.[139]

Although any injected embolic material can occlude normal vessels, injection of the glue is fraught with particular hazards. The time of glue injection is a critical moment (not unlike the moment when a surgeon closes the clip on the neck of an aneurysm). The catheter can become glued to the vessel. If the catheter cannot be removed by intermittent firm, gentle traction, it may be necessary to leave the catheter intravascularly, where it eventually will endothelialize.

Similarly, the catheter, as it is withdrawn, may drag a piece of glue into the proximal part of the artery and occlude it. In this event, territories fed by nutrient vessels distal to the occlusion can become ischemic. As mentioned, venous outflow obstruction can result in ICH.

Glue also can pass into the pulmonary circulation. Small amounts (< 0.5 ml) may not be clinically significant. Larger amounts, however, can result in a syndrome akin to acute idiopathic pulmonary embolism. Because the glue is extremely thrombogenic, it can pick up thrombus en route and form more clot once lodged in the pulmonary vasculature. This is of particular concern in small children with large AVMs. At the time of gluing, the anesthetist must be ready to intervene immediately in the event of catastrophe.

Treatment of Spinal Cord Lesions

Embolization can be used for intramedullary spinal AVM, dural fistulas, or tumors invading the spinal canal.[5,122] The experience of the anesthetist and INR team will determine the optimal choice of anesthetic technique for a given patient. For cases performed with general anesthesia with tracheal intubation, an intraoperative wake-up test may be requested. We find it useful to review the wake-up procedure carefully with the patient the night before the procedure and again the morning of the procedure. Several practice neurologic examinations are performed with the patient,

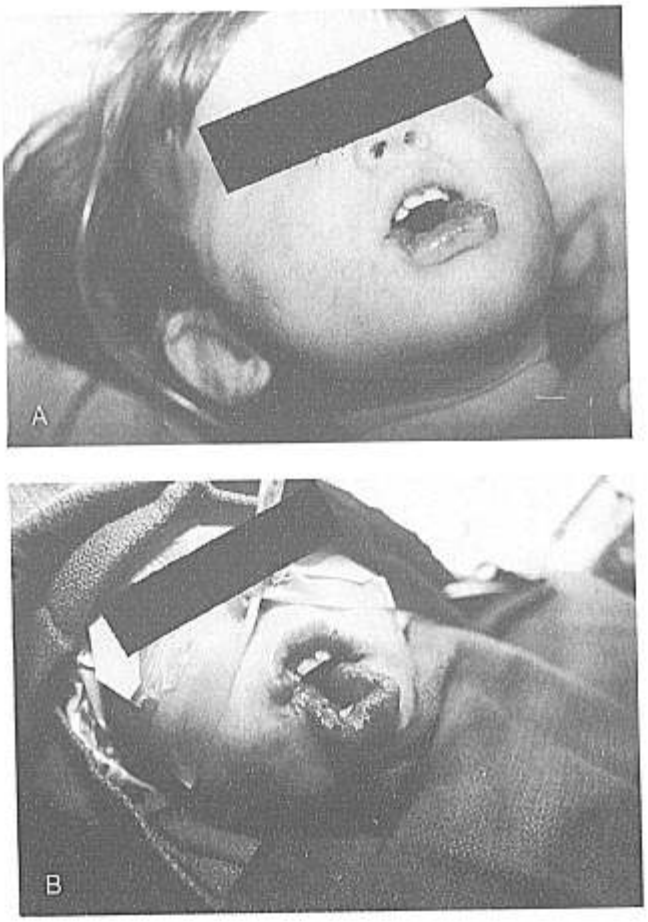

Fig. 13. Example of marked soft tissue swelling after 95% ethanol sclerotherapy. *(A)* A 5-yr-old girl with a venous malformation of the lower lip. *(B)* The patient has just undergone ethanol injection, and the dramatic degree of swelling can be appreciated.

both when awake and after initial sedation has been given. Our preferred regimen for patients requiring general anesthesia who are scheduled for intraoperative wake-up is nitrous oxide, narcotic, and propofol. Neuromuscular blockade, if used, should be readily reversible for the wake-up test.

For selected lesions, somatosensory and motor-evoked responses can be helpful in both anesthetized and sedated patients. When using motor-evoked–potential monitoring with transcranial-magnet stimulation during general anesthesia, we titrate neuromuscular blockade to a reduction of single twitch height (T1) to ≈ 40–50 of baseline.# Optimal anesthetic regimens for use of intraoperative motor-evoked potentials have yet to be formulated, and these are discussed elsewhere.[63,68] Pressure measurements from balloon catheters placed in the rectum may be of occasional use in assessing sacral root function.

Carotid Test Occlusion and Therapeutic Carotid Occlusion

Carotid occlusion, both permanent and temporary, can be used in several circumstances. Skull-base tumors frequently involve the intracranial or petrous portion of the carotid artery or its proximal Willisian branches. Large or otherwise unclippable aneurysms can be treated partly or completely by proximal vessel occlusion. To assess the consequences of carotid occlusion in anticipation of surgery, the patient can be scheduled for a test occlusion. Cerebrovascular reserve is evaluated by a multimodal combination of angiographic, clinical, and physiologic tests. Such testing is used to arrive at the safest course of action for a given patient's clinical circumstances.

First, after routine carotid and vertebral angiograms, the anatomic integrity of the circle of Willis (*i.e.*, the ability of blood to cross the two posterior communicating arteries and the anterior communicating artery) can be assessed. The radiologists will compress the ipsilateral carotid during contrast injection into the contralateral carotid to assess the cross-filling from the other hemisphere *via* the anterior communicating artery. Ipsilateral carotid compression followed by vertebral injection of contrast will demonstrate posterior communicating artery patency.

A catheter with both a lumen and a balloon is placed in the carotid artery. The balloon is long enough to block the distal branches of the internal carotid artery that can serve as a source of collateral circulation. A baseline neurologic examination is performed, transcranial Doppler ultrasound velocity of the middle cerebral artery is recorded, and CBF is measured by intracarotid 133-Xe injection. Baseline femoral and carotid pressures are noted.

The balloon then is inflated; the stump pressure in the carotid distal to the balloon is recorded.§§ When the balloon is inflated, focal headache may occur.[82] It is our clinical impression that the blood pressure often increases 10–15% with inflation of the balloon. Although it is probably wisest not to aggressively treat modest increases in blood pressure, the anesthetist must

§§ Although normal stump pressure does not guarantee normal cerebral blood flow (CBF), very low stump pressure certainly implies low CBF,[76] and there appears to be a good correlation between stump pressures and other indices of CBF.[59,64]

be prepared to treat hemodynamically significant bradycardia with atropine.

The neurologic examination is repeated. As in the case with SAFE, attention is directed to areas at risk, such as watershed regions, as well as "quiet areas" (see superselective anesthesia functional examination). After a few minutes of equilibration, 133-Xe CBF and transcranial Doppler ultrasound values are measured.

Immediately thereafter, a SPECT tracer can be given. At the present time, Ceretec (technetium-99m–labeled hexamethylpropylamineoxime) is used. Ceretec is a tracer that rapidly crosses the blood brain barrier and binds to cerebral tissues and takes a "snapshot" of the blood-flow distribution (not quantitative CBF levels) to the brain at that instant. The relatively long half-life of Ceretec ($\approx$ 6 h) allows for the patient to go to the nuclear medicine department after the procedure to have the "snapshot" developed by placement in the SPECT scanner.

To assess the extent of cerebrovascular reserve more completely, deliberate hypotension is begun after 15 min of observation of the patient at spontaneous postocclusion MAP. The *lack* of cerebral ischemic symptoms at relative normotension does not yield information on the status of cerebrovascular reserve. During subsequent open craniotomy, the blood pressure may be considerably lower, especially with rapid blood loss. In addition, the postoperative surgical patient is notoriously prone to sags in arterial blood pressure. Finally, superimposition of an embolic event, either perioperatively or at some later time, will require some degree of cerebrovascular reserve to prevent infarction.

For these reasons, the blood pressure is lowered gradually to determine at what point the patient begins to show evidence of cerebral ischemia.[4] We usually begin with esmolol (and consider adding nitroglycerin in a patient with coronary artery disease) and slowly bring the pressure down as the radiologist continually assesses neurologic function. One must proceed cautiously with blood pressure reduction to interpret the neurologic examination rationally. Frequently, the first sign of impending cerebral ischemia is yawning. If the radiologist feels that the patient is becoming symptomatic, the balloon is deflated and the hypotensive agent(s) are discontinued. Depending on the clinical circumstances, phenylephrine (or another clinically appropriate agent) can be used to bring the blood pressure back toward normal levels.

The patient's hematocrit, Pa_{CO_2}, and blood pressure at the time when the test occlusion is considered "passed" or "failed" should be noted carefully. The lowest systemic pressure obtained before symptoms, if any, and the results of the other imaging modalities are considered in formulating a treatment plan regarding INR occlusion or the advisability of vascular sacrifice during surgical resection. Although uniform guidelines have yet to be formulated, significant asymmetry on SPECT[79] (or stable Xe computerized tomography) or a > 25% decrease in 133-Xe CBF or transcranial Doppler ultrasound values[4] after test occlusion can be useful in selecting patients for extracranial-to-intracranial bypass procedures if the carotid artery must be sacrificed.[89] Limitations of SPECT scanning in this setting are discussed elsewhere.[114]

Intracranial Aneurysm Procedures

Aneurysm Ablation. Modern neurosurgical and neuroanesthetic techniques, coupled with improved postoperative management, have improved the care of the patient with intracerebral aneurysms.[109] Most lesions can be safely obliterated surgically with preservation of the parent vessel. However, difficulties in the management of certain types of aneurysms remain, such as giant or fusiform aneurysms.[110] In addition, patients with medical risks or of poor neurologic grade may not be surgical candidates. The two basic approaches for INR therapy are occlusion of proximal parent arteries and obliteration of the aneurysmal sac.

For some giant aneurysms, cervical carotid balloon ligation may be performed with or without subsequent extracranial-to-intracranial bypass.[89] The described test occlusion with physiologic monitoring can be useful in predicting which patients have borderline cerebrovascular reserve.[39]

Endovascular obliteration of the aneurysmal sac while sparing the parent vessel is still challenging.[53] Manipulation of the sac can cause distal thromboembolism and rupture. Incomplete obliteration can result in recurrence and hemorrhage. The aneurysmal sac can be obliterated by use of coils and balloons. The newest form of treatment for aneurysms is the Guglielmi detachable coil.[47,48] This is a curlicue type of platinum coil that is attached to a stainless steel guide wire. The coil is passed through a superselective catheter into the aneurysmal sac and then detached by the passage of an electric current through the guide wire, which causes the stainless steel portion to detach from the platinum coil by electrolysis, leaving the coil curled up in the sac. Small aneurysms may need only one coil; larger sacs may take several. Because these

procedures can be quite long (especially for large lesions requiring multiple Guglielmi detachable coils) and there is a lesser need for these procedures to follow the neurologic examination, these cases often are performed under general anesthesia with tracheal intubation for placement of coils.

The anesthetist should be prepared for aneurysmal rupture and acute subarachnoid hemorrhage at all times, either because of the spontaneous rupture of a leaky sac or direct violation of the aneurysm wall by the vascular manipulation. At the present time, there is not the same degree of certainty that the lesion has been completely removed from the circulation after coil ablation of the aneurysm as there is with application of a surgical clip. There may be areas of the dome that are still in contact with the arterial blood column. Therefore, attention to postprocedure blood pressure control is warranted.

Balloon Angioplasty of Cerebral Vasospasm from Aneurysmal Subarachnoid Hemorrhage. In cases of symptomatic vasospasm refractory to deliberate hypertension and intravascular volume expansion, angiography can be performed to assess the contribution of large proximal conductance vessels (usually internal carotid, middle, or anterior cerebral arteries).[81] Angioplasty usually is reserved for patients who already have had the symptomatic lesion surgically clipped (for fear of rerupture), and it is done early in the course of symptomatic ischemia to prevent transformation of a bland infarct into a hemorrhagic one. A balloon catheter is guided under fluoroscopy into the spastic segment and inflated to distend the constricted area mechanically.

The vasospastic segment functions in a fashion analogous to that of an atherosclerotic stenosis and induces a pressure drop across its length.[102] The microvasculature distal to the spastic segments is probably maximally vasodilated.[46] Dilating the spastic arterial segment alleviates the pressure drop and improves distal perfusion. As in the case of occlusive cerebrovascular disease reported by Schroeder,[102] angiographic evidence of a narrowed vessel does not correlate necessarily with an actual hemodynamic impairment. Therefore, a good angiographic result does not guarantee patient improvement.

These procedures commonly are performed in patients who are *in extremis* and, therefore, frequently are intubated, on vasopressor agents, and have either ventricular drainage or other intracranial-pressure (ICP) monitoring equipment in place. Blood pressure management after angioplasty must take into consideration the presence and age of any existing cerebral ischemia or cerebral infarction. If deliberate hypertension is being used to ameliorate a focal neurologic deficit before angioplasty, after angiographic demonstration of significantly widened spastic segment, blood pressure probably should be managed in the normal range.

Treatment of Other Central Nervous System Vascular Malformations

Dural Arteriovenous Malformations. Dural AVM, initially thought to be a congenital disorder, currently is considered an acquired lesion resulting from venous dural sinus stenosis or occlusion, opening of potential arteriovenous shunts, and subsequent recanalization. Dural AVMs may be fed by multiple intra- and extracranial arteries and multistaged embolization usually is performed.[51] SAFE is performed as it is in cases of intracranial AVMs. N-butyl cyanoacrylate usually is used as an embolic agent. Both transarterial and transvenous approaches can be used to access the dural sinuses.

Carotid Cavernous and Vertebral Fistulae. Carotid cavernous fistula are direct fistulae usually caused by trauma to the cavernous carotid artery leading to communication with the cavernous sinus.[50] They usually are associated with basal skull fracture but can result from penetrating injuries, collagen-deficiency diseases, ruptured aneurysms, arterial dissection, or fibromuscular dysplasia. The detachable balloon has nearly totally replaced the surgical treatment of carotid cavernous fistula.[22]

Vertebral artery fistulae are connections to surrounding paravertebral veins that usually are the result of penetrating trauma, but can be congenital, result from blunt trauma, or be associated with neurofibromatosis. In addition to cerebral involvement, spinal cord function also may be impaired.

Both carotid cavernous fistula and vertebral fistulae can induce arterial hypotension and venous hypertension in neighboring circulatory regions, analogous to true cerebral AVMs. Normal perfusion pressure breakthrough rarely has been described after fistula interruption,[49] so attention to postprocedure blood pressure control may be warranted.

Vein of Galen Malformations. These are relatively uncommon but complicated lesions that are evident in infants and require a multidisciplinary approach. The patients may have intractable congestive heart failure, intractable seizures, hydrocephalus, and mental retardation.[107] Several approaches have been attempted, including transarterial and transvenous methods.[72] An-

esthetic considerations for INR therapy are the same as those for surgical treatment.[77]

In infants with high output failure, preexisting right-to-left shunts, and pulmonary hypertension, a relatively small pulmonary glue embolism can be fatal (see therapeutic embolization of intracranial arteriovenous malformations).

Sclerotherapy of Venous Angiomas

Craniofacial venous malformations are congenital disorders and, in addition to causing significant cosmetic deformities, can impinge on the upper airway and interfere with swallowing. Many of these lesions are resistant to conventional surgery, cryosurgery, or laser surgery. In this procedure, USP-grade 95% ethanol‖‖ opacified with contrast is injected percutaneously into the lesion under fluoroscopic guidance, resulting in a chemical burn to the lesion, eventually shrinking it. Sclerotherapy alone may be adequate treatment, or it can be combined with surgery.[67]

Venous angiomas can occur anywhere in the body and usually are treated at multiple sessions. The procedures are short (30–60 min) but painful, and general anesthesia with tracheal intubation is used. Complex airway involvement may require tracheal intubation with fiberoptic techniques[99] or elective preprocedure tracheostomy. Because *marked* swelling occurs immediately after ethanol injection, the ability of the patient to maintain a patent airway must be considered carefully in discussion with the radiologist. An example of acute swelling is shown in figure 13. More graphic examples of venous malformations impinging on the airway are shown elsewhere.[67]

Ethanol has several noteworthy side-effects. First, on injection, it can cause changes in the pulmonary vasculature and create a short-lived shunt or a ventilation-perfusion mismatch. Desaturation on the pulse oximeter frequently is noted after injection; in our experience, at least a 2–3% drop in oxygen saturation is noted in approximately 25% of cases, but rarely more significant decreases. There have been unpublished anecdotal reports of cardiac arrest during ethanol sclerotherapy, as well as hypoglycemia. The systemic effects of ethanol in this setting need further study.

Placing the patient on 100% O_2 during ethanol injection is a possible consideration. The predictable intoxication and other side effects of ethanol may be evident after the patient's emergence from anesthesia, particularly as postemergence agitation in children.

Intraarterial Chemotherapy and Embolization of Tumors

Preoperative embolization can be performed for many hypervascular tumors in intracranial, craniofacial, and spinal territories.[19,73] Superselective intraarterial administration of chemotherapeutic agents can be used for neoplasms refractory to conventional treatments or as a primary adjunct. A combination of vascular obliteration and chemotherapy commonly is used. SAFE is used to assess the safety of vessel occlusion or, in some cases, eventual surgical sacrifice.

Hypervascular tumors can swell if there is venous occlusion, hemorrhage into the tumor bed, or significant tissue necrosis. These conditions are most likely to be seen when there is incomplete embolization of feeding arteries. Patients may present with an already compromised airway, and treatment can result in further compromise. Systemic effects of chemotherapeutic agents are minimal because, even though standard intravenous doses are used, the drugs appear to become trapped in tissue being embolized.

Paragangliomas present the possibility for catecholamine release from the tumor during the course of embolization,[66] and the means to treat a hypertensive crisis should be at hand. In addition, swelling after embolization of carotid body tumors can result in symptomatic bradycardia.

Management of Occlusive Cerebrovascular Disease

Angioplasty. Angioplasty for atherosclerosis has been tried in cervical and intracranial arteries with favorable results.[54,126] Risk of distal thromboembolism is the major issue to be resolved in this procedure, and methods such as a catheter system, which employs an occluding balloon distal to the angioplasty balloon, have been proposed.[123] Indications, and efficacy, in relation to carotid endarterectomy remain to be worked out. At present, patients offered carotid angioplasty are poor surgical candidates because of advanced age or concomitant medical disease. Encouraging initial results, coupled with clearer indications for revascularization from the North American Symptomatic Carotid Endarterectomy Trial study,[84–86] will speed the development and application of this form of angioplasty.

Anesthetic considerations for this procedure include those discussed for deliberate hypertension above (see blood pressure augmentation deliberate hypertension)

‖‖ One hundred percent ethanol may be contaminated with benzene.

and the general considerations pertinent to the care of the carotid endarterectomy patient.

Thrombolysis of Acute Thromboembolic Stroke. In acute occlusive stroke, it is possible to recanalize the occluded vessel by superselective intraarterial thrombolytic therapy.[13,23] Thrombolytic agents can be delivered in high concentrations by a microcatheter navigated closely to the clot. Neurologic deficits can be reversed without additional risk of secondary hemorrhage if treatment is completed within 6 h from the onset of carotid territory ischemia and within 24 h in cases of vertebrobasilar territory ischemia.

One of the impediments in development in this area has been the fear of increasing the risk of hemorrhagic transformation in the acute infarction patient. However, recent and accumulating evidence suggests that this paradigm (based on early autopsy series) may no longer be tenable.[71] The incidence of postthrombolysis hemorrhage is at least equal to, if not lower than, the incidence of spontaneous hemorrhagic transformation. Randomized studies of the efficacy of this treatment remain to be performed.

Anesthetic considerations for these patients include the usual concerns for elderly patients with symptomatic and probably widespread atherosclerotic disease. Blood pressure management is a particularly important consideration. Patients with acute thromboembolic stroke are commonly spontaneously hypertensive, and, in the face of a nonhemorrhagic focal neurologic deficit, should not have their blood pressure aggressively treated. After clot lysis, blood pressure probably should be maintained in the normal range and ideally titrated to some index of CBF to prevent hyperperfusion injury. The pathogenesis of hemorrhagic transformation may be related to collateral blood flow to ischemic regions, acting in concert with systemic hypertension,[71] but studies in this specific clinical setting, concerning both blood pressure management and the use of other cerebral protective techniques, are lacking.

As INR methods develop, this area could evolve into one of the most interactive with anesthesia care because of the high incidence of systemic disease present in these patients, coupled with the high morbidity associated with acute thromboembolic stroke.

Treatment of Epistaxis

Hypertension, arteriosclerosis, coagulopathies, trauma, or vascular dysgenesis can cause intractable epistaxis uncontrolled by the usual surgical maneuvers such as intranasal packing. Numerous tortuous vessels, fed by branches of external carotid artery, are usually visible in nasal mucosa by angiography. Polyvinyl alcohol particles and oxidized cotton pledgets are used in obliterating these arteries.[19] Packing can be removed immediately after the embolization.

Maintenance of an unobstructed airway and adequate gas exchange with minimal sedation is the primary goal of anesthetic management in these cases.

Conclusions and Future Directions

Interventional neuroradiology is a rapidly expanding field in the treatment of central nervous system disease. The selection of patients for operative neurosurgery will be influenced profoundly by the development of this specialty. Looking ahead to the future of neuroanesthesia,[137] the use of general anesthesia probably will decline as neurosurgical techniques, both in INR and in the areas of stereotactic and functional neurosurgery, progress. The traditional concepts of "conscious sedation", "monitored anesthesia care", and "general anesthesia" will have to be modified to allow rapid changes in sensorium in patients with unsecured airways and, at the same time, to provide manipulation of systemic arterial pressure and other physiologic functions. In the coming years, there will be a clear role for anesthesiologists to interact in the complex care of these patients and to contribute to therapeutic advances.

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