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CHAPTER 1.1

Intracranial Neurosurgery

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Craniotomy for Intracranial Aneurysms

Surgical Considerations

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Description: Intracranial aneurysms are focal protrusions arising from vessel wall weaknesses at major bifurcations of the arteries at the base of the brain (some frequent sites of aneurysms are shown in Fig. 1.1-1), and are most commonly treated by microsurgical clip ligation. The high rate of mortality and morbidity from aneurysmal rupture necessitates treatment for symptomatic lesions. Treatment for asymptomatic lesions generally is recommended when the lifetime risk of rupture exceeds the risk of treatment. The most important surgical considerations include: clinical presentation, aneurysm size and location, patient age and neurologic status, and medical comorbidities. Aneurysm rupture into the subarachnoid spaces is the most common clinical presentation; however, symptoms from the mass effect of enlarging aneurysms or ischemic symptoms from emboli also may occur. Aneurysm morphology, size, and location are important in determining the surgical approach, and these aneurysm characteristics, as well as patient age, condition, and comorbidities, affect the overall outcome. The **Hess and Hunt clinical grading system** (Table 1.1-1) has been proven useful in describing patients with ruptured intracranial aneurysms because it has been shown to have prognostic value in terms of ultimate clinical outcome. Grading is based on the neurologic examination and ranges from grade I (minimal headache, no neurologic deficit) to grade V (moribund).

Through a **craniotomy** or **craniectomy,** using microscopic techniques, the parent vessel giving rise to the aneurysm is identified. The aneurysm neck is isolated, and a small, nonferromagnetic alloy spring clip is placed across the aneurysm neck, excluding it from the circulation. A **frontotemporal (pterional) craniotomy** normally is used to approach anterior circulation aneurysms. This requires extensive drilling of the medial sphenoid wing (pterion) and *(Print pagebreak 5)* allows access to most aneurysms on the anterior and lateral Circle of Willis vessels: internal carotid-paraclinoid/superior hypophyseal artery; internal carotid-ophthalmic artery; posterior communicating artery; anterior choroidal artery; internal carotid artery bifurcation; middle cerebral artery; and anterior communicating artery. Posterior circulation aneurysms are approached via a pterional or subtemporal exposure (upper basilar artery, posterior cerebral artery, superior cerebellar artery), a suboccipital exposure (vertebral artery, posterior inferior cerebellar artery), or a combined subtemporal and suboccipital exposure (basilar trunk, vertebrobasilar junction). Circulatory arrest under CPB with deep hypothermia (16–20°C) is used for repairing some giant (> 2.5 cm) aneurysms.

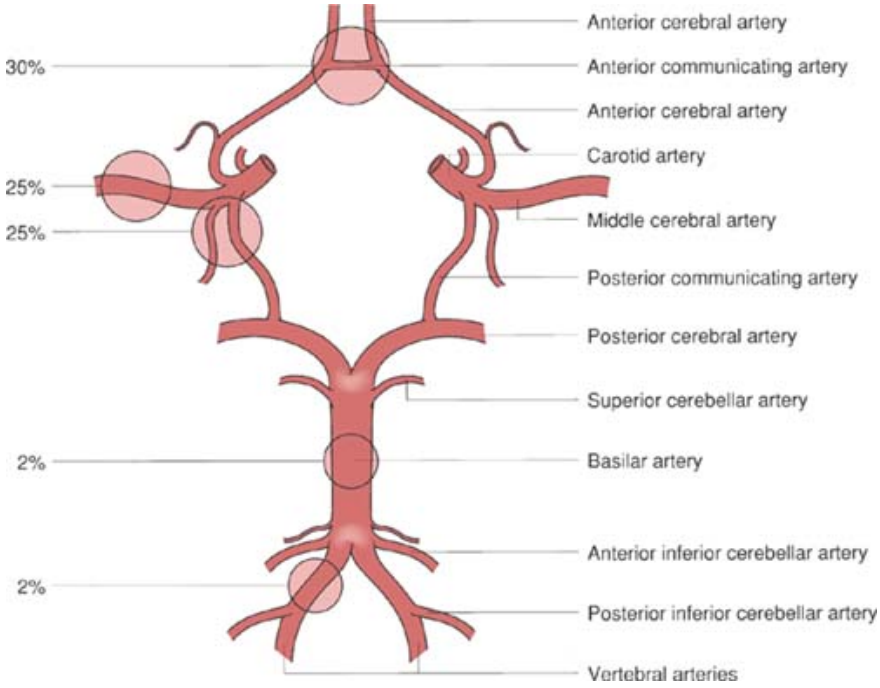

Figure 1.1-1. 1. Locations of aneurysms of the circle of Willis and their relative occurrence. (Reproduced with permission from Greenfield LJ, Mulholland MW, Lillemore KD, et al: *Surgery: Scientific Principles and Practice*, 3rd edition. Lippincott Williams & Wilkins, Philadelphia: 2001.)

Table 1. 1-1 Hunt-Hess Grading System for Aneurysmal SAH*

Grade	Description
I	Asymptomatic or minimal headache and slight neck stiffness (mortality 2%)
II	Moderate-to-severe headache, neck stiffness, no neurological deficit (except cranial nerve palsy)—(mortality 5%)
III	Drowsiness, confusion, or mild focal deficit (mortality 15–20%)
IV	Stupor, moderate-to-severe hemiparesis, possible early decerebrate rigidity, and vegetative disturbances (mortality 30–40%)
V	Deep coma, decerebrate rigidity, moribund (mortality 50–80%)

*The presence of serious systemic disease—such as hypertension, diabetes, severe arteriosclerosis, chronic pulmonary disease, and angiographic vasospasm—results in placement in the next less-favorable category.

Usual preop diagnosis: Cerebral aneurysm; subarachnoid hemorrhage (SAH); intracerebral hemorrhage; progressive neurological deficits (mass effect on cranial nerves or CNS structures); TIAs; cerebral infarct

Summary of Procedures

	Anterior Circulation Aneurysms	Posterior Circulation Aneurysms	Circulatory Arrest (CPB) W/Deep Hypothermia
Position	Supine, head in Mayfield headrest, turned 30–45° to side away from aneurysm, vertex dropped (Fig. 1.1-5)	Or lateral decubitus, head lateral in Mayfield headrest	+ Both groins must be accessible for arterial + venous cannulation; access to chest for defibrillation
Incision	Frontotemporal	Or temporal, temporosuboccipital, or suboccipital	

Special instrumentation	Operating microscope; aneurysm clips; radiolucent table, and headrest for intraop angiography	+ Aperture clips to accommodate cranial nerves and critical vessels	+ CPB pump; femoral cannulae; defibrillator; CUSA for partially thrombosed aneurysms
Unique considerations	Temporary arterial clipping; mild hypothermia (33°C); intraop angiography with access to femoral artery; electrophysiological monitoring (SEPs, BAERs); brain relaxation; ± lumbar subarachnoid CSF drainage; dexamethasone 8–12 mg iv		+ No manipulation of brain retractors after systemic heparinization; meticulous attention to hemostasis
Antibiotics	Ceftriaxone 1–2 g iv q d (avoid Ca^{++} containing solutions)		
Surgical time	3–5 h	3–6 h	6–8 h
Closing considerations	Rewarming		
EBL	250–1000 mL		2000–4000 mL
Postop care	ICU 1–7 d; postop CBF monitoring. Transcranial Doppler (TDC) monitor		+ ICP monitoring
Mortality	Unruptured: 0.5% Ruptured: Hunt and Hess grades I–III:< 10% (See Table 1.1-1.)Hunt and Hess grades IV–V:20–40% (See Table 1.1-1.)	1.5% 15% 20–50%	5–10% (giant aneurysms) 5–15% 15–50%
Morbidity	Neurological: 5–20% Cranial nerve injury Stroke Hydrocephalus: 15–25% Hyponatremia Respiratory failure: Rare Seizures: 2–20% Thromboembolism: Rare CSF leak: Rare Infection: Rare Massive blood loss: Rare	10–30%	10–50%
Pain score	3–4	3–4	4–5

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Patient Population Characteristics

Age range	30–70 yr (Peak incidence in 5th and 6th decades)
Male:Female	44:56
Incidence	8–16/100,000/yr for ruptured aneurysms with SAH (highest in Japan and Finland) Overall incidence of rebleeding is 11% (4% in the first 24 h).
Etiology	Idiopathic (probably acquired and related to hemodynamic stress at arterial branch points, although may have congenital predisposition to loss of internal elastic lamina); traumatic; infectious; familial

Associated conditions	Polycystic kidney disease; coarctation of the aorta; Marfan syndrome; Ehlers-Danlos syndrome; intracranial AVMs; aortic aneurysm; fibromuscular dysplasia; pseudoxanthoma elastica; Rendu-Osler-Weber syndrome; cocaine/amphetamine use

Anesthetic Considerations

(Procedures covered: craniotomy for intracranial aneurysms; craniotomy for cerebral embolectomy)

Preoperative

Aneurysms may occur in any age group, although they generally become symptomatic and are diagnosed in young or middle-aged adults who are usually in otherwise good health. Most patients have warning Sx before the first major bleed, but these tend to be mild and nonspecific (e.g., headache, dizziness, orbital pain, slight motor or sensory disturbances). The symptoms are generally disregarded by both patients and physicians. Most patients with SAH will be receiving nimodipine; SBP should be maintained between 100 and 160 mmHg using pressors if necessary. Patients with symptomatic vasospasm may benefit from triple-H therapy (see below).

Respiratory	Respiratory complications (e.g., neurogenic pulmonary edema (up to 23%), pneumonia, ARDS, PE) are the most common non-neurologic causes of death following SAH. Pulmonary aspiration may have occurred as the result of a neurological deficit from an intracranial hemorrhage. **Tests:** As indicated from H&P. Elevated cTnI may predict pulmonary complications.
Cardiovascular	Generally, these patients do not have other cardiovascular diseases, although intracerebral aneurysms occur more commonly in patients with certain congenital disorders, such as polycystic disease of the kidneys, coarctation of the aorta, fibromuscular hyperplasia, and Marfan and Ehlers-Danlos syndromes. Patients who have had a recent intracranial hemorrhage from leaking or rupture of a cerebral aneurysm are prone to develop systemic HTN, hypovolemia, and ECG abnormalities. The HTN is thought to be due to autonomic hyperactivity, and is generally treated with antihypertensive medication, which should be continued up to the time of anesthesia and surgery. Why hypovolemia occurs following SAH is not clear, but may be due in part to cerebral vasospasm and to sustained bed rest. ECG abnormalities occur in most patients following an intracranial hemorrhage and may represent subendocardial injury 2° catecholamine release. Dysrhythmias (most commonly PVCs) occur in 30–80% of patients, and ischemic changes (typically T-wave inversion and S–T-segment depression) are seen in > 50%. Appropriate preop preparation includes ECG characterization of the abnormality. If patient has Hx of ischemic heart disease, ECHO and cardiac enzyme studies may be helpful in determining whether the myocardial injury and consequent ECG changes are likely to be of clinical significance. **Tests:** ECG; others (e.g., cardiac enzymes) as indicated from H&P.
	Seldom do aneurysms produce neurological Sx by enlarging to the point that they compress adjacent neural tissue or cause ↑ ICP. If an intracranial hemorrhage occurs, the neurological dysfunction will vary, depending on the site and extent of the hemorrhage. Typically, SAH → ↑↑ ICP → Sudden severe HA ± confusion and disorientation, ± motor deficit, ± coma. Admission GCS < 12

Neurological	predicts ↑ mortality. Subsequent cerebral vasospasm (70% incidence) may cause worsening of the neurological deficits. Clinically detectable vasospasm commonly begins on day 1 reaching a maximum at 6–8 d, and usually is resolved within 2–3 wk. The precipitating agent is believed to be free Hb → release of vasospastic substances from brain tissue. Treatment of vasospasm usually involves support of BP (occasionally, induced HTN), hydration to normovolemia, and systemic Ca^{+} antagonists (e.g., nimodipine (60 mg PO q 4 h) or iv (3–15 mg/h iv)). Triple H therapy (↑ volume, ↑ BP, ↓ Hct) does not appear to confer any additional advantage in most patients. Control of BP is important: any substantial ↑ BP may → serious rebleed, permanent neurological deficits or death; any substantial ↓ BP may → cerebral ischemia and infarction. Arterial catheterization and continuous beat-to-beat monitoring of BP prior to induction of anesthesia is essential in these patients. **Tests:** CT; MRI; CTA, which the anesthesiologist should examine preop to identify the nature and site of the aneurysm.
Hydrocephalus	Acute hydrocephalus occurs in 15–20% of patients within 24 h of SAH. External ventricular drainage may be necessary.
Hematologic	Thrombocytopenia may develop following SAH. **Tests:** Hct; PT; PTT
Radiographic findings	CT: Blood in basal cisterns (95% of patients) or ventricles; CTA (replacing conventional angiography): anatomical characteristics of the aneurysm.
Laboratory	Cardiac enzymes if ECG changes observed (↑ cTnI predicts ↓ neurologic outcome and ↑ mortality); others as indicated from H&P.
Hepatic	Hepatic dysfunction following SAH is not uncommon (24% in one series). **Tests:** LFTs, as indicated from H&P.
Premedication	Small doses of midazolam 1–3 mg iv are preferable to opiates. Detailed discussion with the patient about the anesthetic plan, with appropriate reassurance, is essential. Should an intracranial aneurysm leak or rupture in the immediate preop period, its signs may be difficult to distinguish from those associated with excessive response to premedication.

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Intraoperative

Anesthetic technique: GETA. The goals of anesthesia for this operation are to: (a) maintain optimum CPP (cerebral MAP minus cerebral venous pressure or ICP, whichever is greater), although it may be necessary to ↓ CPP rapidly if intracranial hemorrhage occurs during surgery; (b) decrease intracranial volume (blood and tissue) to optimize working space for surgeons within the cranial compartment, thereby minimizing the need for surgical retraction of brain tissue; and (c) decrease metabolic rate and $CMRO_2$ with the expectation that the brain will tolerate hypotension and ischemia if sudden decreases in MAP and, hence, CPP become necessary.

Induction	Smooth induction is essential. STP 2–5 mg/kg (or propofol 1–2 mg/kg iv) to provide amnesia and ↓ cerebral blood volume by inducing cerebral vasoconstriction. Fentanyl 7–10 mcg/kg iv to blunt response to intubation and provide analgesia for the first hours of surgery. Pancuronium 0.1 mg/kg, vecuronium 0.15 mg/kg, or rocuronium 0.7–1 mg/kg to provide muscle relaxation for tracheal intubation and positioning patient. Patients on nimodipine may require pressors (e.g., phenylephrine) during and after induction. Poor H-H grade patients (IV–V) may not tolerate ↓ MAP during induction. These patients may benefit from moderate hyperventilation during induction. Isoflurane or sevoflurane (1/2 MAC if EP monitoring is used), inspired with O_2 Avoid

Maintenance	N_2O > 50% and entirely in patients with ↑↑ ICP. Propofol (75–100 mcg/kg/min) may be used to further ↓ cerebral blood volume, ↓ cerebral metabolism, and ↓ $CMRO_2$ Generally, if pancuronium is used, no additional NMBs are needed; however, if movement is of concern, rocuronium 10 mcg/kg/min will provide adequate neuromuscular blockade. A remifentanil infusion (0.05–0.1 mcg/kg/min) can be used to supplement the anesthetic without interfering with EP monitoring. TIVA is not necessary for monitoring SSEPs or MEPs.
Emergence	H-H grade IV–V patients are not extubated and should be kept sedated on ventilator support postop. For grade I–III patients, with the start of dural closure, consider using low-dose sevoflurane (e.g., 0.5%) in 50% N_2O, supplemented with a low-dose remifentanil infusion (e.g., 0.05 mcg/kg/min). As recovery from anesthesia occurs, the patient's BP generally will increase in response to the emergence stimuli. Titration of β-adrenergic blocking drugs (e.g., labetalol or esmolol) with vasodilators (e.g., SNP) may be needed; if so, the dose should be stabilized before transport to ICU. (See Control of BP, below.) The inhalation agent can be D/C'd at the time of dressing application. Most patients will breathe spontaneously and can be extubated uneventfully while on the remifentanil infusion. If the brain has not been injured by the surgical procedure, the patient should awaken within 10 min after cessation of remifentanil administration. As the patient is awakening, it is important to assure full reversal from neuromuscular blockade and close regulation of BP. If the patient begins to cough on ETT, either it should be removed or cough reflex suppressed with iv lidocaine (0.5–1 mg/kg). Patient is placed in bed in a 30° head-up position and transported to ICU for monitoring overnight. Supplemental O_2should be administered and close regulation of BP maintained. Prophylactic antiemetics (e.g., metoclopramide 10–20 mg and ondansetron 4 mg or dolasetron 12.5 mg) should be given 30 min before extubation.
Blood and fluid requirements	IV: 14–18 ga × 2 NS @ <10 mL/kg + UO Expand blood volume with albumin 5% if Hct >30%. Albumin + PRBC if Hct <30% Maintain colloid oncotic pressure Hetastarch may → coagulopathy.
	Maintain euvolemia. If blood volume is normal, crystalloid fluid should not exceed 10 mL/kg beyond that required to replace UO. Rapid, massive blood loss is possible. If blood volume is low because of vasospasm or prolonged bed rest, albumin 5% is given if Hct > 30%; combinations of albumin and blood, if Hct is < 30%. Hetastarch 6% may be used in place of albumin, but limit use because of potential for coagulopathy.
Brain relaxation	Hyperventilate to $PaCO_2$= 30 mmHg ($PetCO_2$= 25 mmHg). PaO_2> 100 mmHg ↓ fluids < 10 mL/kg + UO Consider propofol infusion to replace N_2O ↓ isoflurane/sevoflurane to < 1/2 MAC Mannitol 0.5–1 g/kg ± Furosemide 0.3 mg/kg ± Steroids (e.g., 8 mg dexamethasone) ± Lumbar CSF drain Head up to provide venous drainage Minimize neck flexion/rotation Standard monitors (see p. B-1). Arterial line
	↓ $PaCO_2$→ ↓cerebral vascular volume (better surgical access) + ↑ CBF to ischemic areas (“Robin Hood” effect) + ↓ anesthetic requirements + ↑ lactic acid buffering. Remifentanil infusion (0.05–0.2 mcg/kg/min) may be required to provide adequate analgesia Mannitol/furosemide →↓K^+; monitor level and replace as necessary. If mannitol is administered too rapidly, ↓ BP may occur, probably from peripheral vasodilation. CSF drain may be placed after induction of anesthesia, avoid rapid drainage → rupture Direct monitoring of arterial BP is essential because marked fluctuations may occur, necessitating drug therapy, as well as need

	Bladder temperature Blood glucose (100–180 mg/dl)	for ABGs. Transducers should always be at the level of the head rather than the heart.
Monitoring	CVP line	Monitoring CVP is desirable in virtually all patients to assess adequacy of fluid therapy, for infusion of vasoactive drugs both intraop and postop, and for aspiration of VAE. Localization of the catheter can be determined by CXR, ECG tracing (noting P-wave changes), or pressure-wave contour and value as the catheter is withdrawn from the right atrium.
	UO (Foley catheter) ± Evoked potentials	
Hypothermia	Water-circulating pads Cold OR Bladder irrigation	Mild hypothermia (33–34°C) is used in some centers to ↓$CMRO_2$and to ↓ susceptibility to ischemic injury during temporary clip application. $CMRO_2$ decreases 30%@ 33°C. This level of hypothermia has minimal effect on coagulation or the incidence of cardiac dysrhythmias. Rewarming using surface means can be quite slow. The use of bladder irrigation (40°C saline) or an InnerCool-type device is useful.
	InnerCool or equivalent	A heat-exchange catheter may be placed in the vena cava (via femoral vein) to facilitate patient cooling and rewarming.
	IHAST Study	Although it implied that mild hypothermia was not useful in aneurysm surgery, the IHAST study was not designed to assess the effectiveness of mild hypothermia in the patient group most likely to benefit: patients requiring temporary clip times > 3 min. Generalizing the IHAST findings to all aneurysm patients is an unfortunate disservice to many of them.
	↓ superficial blood flow	Administer drugs through CVP line in hypothermic patients to ensure prompt effect.
Triple H therapy (if requested)	Hypervolemia	Goal: CVP = 10–12; PCWP = 12–16
	Hypertension	Goal: SBP 120–150 mmHg (preclipping); 160–200 mmHg (postclipping)
	Hemodilution	Goal: HCT = 30–35%
Control of BP	During application of head fixation device (Mayfield): remifentanil: 100–200 mcg iv bolus 1–2 min in advance. During exposure: ↓ MAP to 80% of baseline. Temporary clipping: ↑ MAP to 120% of baseline.	Control of BP is critical to the successful outcome of the case. ↑↑ BP →↑↑ transmural pressure across the aneurysmal wall → rupture of the aneurysm. Many neurosurgeons apply a temporary clip on the major feeding vessel(s) in advance of clipping the aneurysm. This technique collapses the aneurysm and makes the clipping easier and less likely to cause inadvertent rupture. If this technique is used, it is essential for the anesthesiologist to↑ BP 20% above baseline pressure to maximize collateral flow while the feeding vessel(s) is occluded. Phenylephrine is preferred because it has minimal dysrhythmogenic potential. If it becomes necessary to ↓ BP, use

	Postclipping: MAP typically 70–90 mmHg	esmolol 50–200 mcg/kg/min to ↓ HR to 60 supplemented as necessary with SNP 0.1–4 mcg/kg/min to desired effect. Responses to vasoactive drugs are much easier to regulate if a normal blood volume has been established and maintained throughout the anesthetic period. Labetalol (5–100 mg total dose) is a useful adjunct for postop pressure control.
Aneurysmal rupture	↓ MAP to 40–50 mmHg, if necessary. Consider carotid compression	Bolus SNP: 20 mmHg ↓ MAP/15 mcg SNP. Ipsilateral or bilateral carotid occlusion is often effective in controlling hemorrhage while a temporary clip is applied.
	Adenosine 12 mg iv (CVP line)	Adenosine will produce asystole, allowing time for the application for a temporary clip. An external pacemaker should be available.
Positioning	For most aneurysms: Supine, head turned Three-point fixation (beware of marked ↑ BP with use of pins). Use shoulder roll. and pad pressure points. eyes.	Anesthetic gas hoses and all monitoring and vascular catheter lines are directed to patient's side or feet, where the anesthesiologist is positioned during surgery. SCDs used to minimize DVT. Shoulder roll to ↓ brachial plexus stretch. Remifentanil (100–200 mcg bolus) to minimize ↑ BP during skull pinning.
Complications	Aneurysm rupture (intraop)	6–18% incidence; up to 2% rupture during induction.
	Hypothermia (mild)	Many patients can be extubated safely at core T ≤ 35°C with active rewarming in progress.

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Postoperative

Complications	Intracranial hemorrhage Stroke Cerebral vasospasm	New deficits or delayed emergence may necessitate urgent transport to CT scanner and/or return to the OR Prophylactic triple H therapy (↑ volume, ↑ BP, ↓Hct) may have no advantage over simple euvolemia.
Pain management	Meperidine (10–20 mg iv prn) Codeine (30–60 mg im q 4 h prn)	Meperidine will ↓ postop shivering. Avoid oversedation → interferes with neuro exam.
Tests	CT scan, if any change in neurological status	Be prepared to re-secure airway.

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Suggested Readings

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Anesthetic Considerations for Craniotomy for Giant Intracranial Aneurysms

(Requiring deep hypothermic circulatory arrest)

Preoperative

Aneurysms are classified as “giant” when they are > 2.5 cm in diameter. These represent 5% of all aneurysms. They occur twice as often in women, usually become symptomatic in the 4th or 5th decade of life, and present particularly difficult surgical challenges: (a) their large size makes direct visualization of the vascular anatomy difficult; (b) vascular branches essential to maintaining flow to normal brain may be an integral part of the giant aneurysm and cannot be included in the clipping without causing permanent neurological injury; (c) standard aneurysm clips may not occlude a large, turgid aneurysm, or may slip or move, once applied; and (d) giant aneurysms may rupture during dissection or clip application, resulting in severe neurological morbidity or mortality. Many

of these aneurysms are amenable to coiling or other interventional radiologic techniques. For those requiring craniotomy, a special anesthetic and surgical management, using deep hypothermia to 18°C, achieved with fem-fem CPB and temporary circulatory arrest, is employed. These techniques decompress the aneurysm, making it easier to clip, and protect the brain during circulatory arrest. The duration of cardiac arrest may be as long as 45 minutes.

Respiratory	None unless patient has Hx of smoking or has sustained pulmonary aspiration as a result of a neurological deficit from an intracranial hemorrhage. **Tests:** As indicated from H&P; elevated cTnI may predict pulmonary complications.
Cardiovascular	Generally, these patients do not have other cardiovascular diseases. (See Anesthetic Considerations for Intracranial Aneurysms, p. 6.) **Tests:** ECG; others as indicated from H&P.
Neurological	These patients usually present with complaints of intermittent or persistent headaches or visual disturbances that are probably due to aneurysmal compression of adjacent neural tissue or ↑ ICP. If an intracranial hemorrhage occurs, neurological dysfunction varies, depending on site and extent of the hemorrhage. Cerebral vasospasm is a major complication of intracranial hemorrhage (see discussion in Anesthetic Considerations for Intracranial Aneurysms, p. 6). **Tests:** CT; MRI; angiogram. The anesthesiologist should examine the cerebral angiogram preop to visualize the size and site of aneurysm.
Hematologic	T&C for 6 U PRBCs. **Tests:** Hct; PT; PTT; hemogram; others as indicated from H&P.
Laboratory	Other tests as indicated from H&P.
Premedication	If premedication is desirable, small doses of midazolam (e.g., 1–2 mg) are preferable to opiates. Detailed discussion with patient about the anesthetic plan, with appropriate reassurance, is effective in reducing premedication requirements. Should an intracranial aneurysm leak or rupture in the immediate preop period, it may be difficult to distinguish this event from changes associated with excessive responses to premedication.

Intraoperative

Anesthetic technique: GETA. The goals of anesthesia for this procedure are to: (a) provide adequate surgical anesthesia; (b) ↓ intracranial volume (blood and tissue) and optimize working space within the cranial compartment, thereby minimizing the need for surgical retraction of brain tissue; and (c) ↑ tolerance of the brain to ischemia by decreasing $CMRO_2$, which occurs with the use of deep hypothermia, barbiturate therapy, and isovolemic hemodilution.

Induction	STP 2–5 mg/kg or propofol 2–3 mg/kg iv to provide amnesia and ↓ CBV by inducing cerebral vasoconstriction. Fentanyl 7–10 mcg/kg iv to provide analgesia for the first hours. Vecuronium 0.15 mg/kg or rocuronium 0.7–1 mg/kg to provide relaxation for intubation and positioning.
Maintenance	Propofol 100–200 mcg/kg/min administered by constant-infusion pump. These doses provide additional amnesia and ↓ CBV and $CMRO_2$ Isoflurane ≤ 1%. N_2O not used. An additional dose of NMB is administered just prior to the start of CPB.
Emergence	Because of the length and nature of the operation, and the potential for temporary neurological injury, it is advisable to leave the ETT in place immediately postop, and send patient to the ICU on controlled ventilation. If patient begins to cough, the reflex should be suppressed with opiates, NMBs, and/or LTA sprayed down the ETT. The patient is placed in bed in a 30° head-up position and transported to ICU for overnight monitoring. Supplemental O_2should be administered and close regulation of BP maintained. Prophylactic antiemetic (e.g., droperidol 0.625 mg or metoclopramide 10–20 mg, and ondansetron 4 mg) should be given 30 min before extubation.

Blood and fluid requirements	IV: 14–16 ga × 2 NS @ 1–2 mL/kg/h PRBC 4–6 U	Maintain euvolemia Cold NS up to 10 mL/kg, + a volume equal to UO, is administered during surgery.
	5% albumin 8 × 250 mL NS 4 × 1000 mL	Albumin and NS are placed in a refrigerator at 4°C the night before surgery to be used as necessary for cooling during isovolemic hemodilution.
Isovolemic hemodilution	CPD bags	After induction of anesthesia, a 2nd arterial or large-vein cannula is placed for removal of blood into CPD bags. Generally, about 1000 mL of blood are removed and replaced with 1 L of cold albumin 5%. This usually →↓Hct to 22–26%. Frequent intraop Hct checks are appropriate. The withdrawn blood is held at room temperature for reinfusion at the conclusion of operation. In addition, the perfusate from the CPB unit is spun down and packed cells are returned to patient.
Control of ICP (brain relaxation)	Hyperventilate to $PaCO_2$= 25–30 mmHg Limit crystalloid < 10 mL/kg + UO Limit isoflurane ≤ 1%. Mannitol 0.5–1 g/kg ± Furosemide 0.3 mg/kg Dexamethasone 8–12 mg	Ventilation is controlled and TV and RR adjusted such that $PaCO_2$ranges from 25–30 mmHg. There are several advantages to hypocarbia, including: ↓ CBV to provide more surgical working space, thereby lessening the need for vigorous retraction of brain tissue; improving regional distribution of CBF by preferentially diverting blood to potentially ischemic areas of the brain; better buffering of brain lactic acid that may form as a result of focal ischemia; and decreasing anesthetic requirement.
	± Lumbar CSF drainage Position head to promote venous drainage	Elevate head of bed and minimize neck flexion/rotation if possible.
Monitoring	Standard monitors (see p. B-1). Temperature = esophageal, bladder and brain surface Arterial line	Frequent checks are made of Hct, electrolytes, ACT values, before, during, and after CPB.
	CVP (triple-lumen) line	CVP line is used for infusions of esmolol, SNP, and phenylephrine.
	Blood glucose UO	Keep glucose between 80 and 180 mg/dl. Keep UO > 0.5 mL/kg/h.
Control of BP	Maintain BP normal-to-20% below normal.	BP control is critical to successful surgery. ↑ BP during induction or prior to CPB will ↑ transmural pressure across the aneurysmal wall and ↑ likelihood of rupture. Prior to CPB, BP is generally kept to normal-to-20% below normal for patient, using anesthetic agents alone or with an esmolol infusion to ↓ HR to a range of 50–60 bpm. If desired level of BP is not achieved with this combination, SNP or propofol infusion may be added. SNP also facilitates both cooling and rewarming because of its vasodilatory effect.
	Phenylephrine	If a vasoconstrictor is needed, particularly during CPB while patient is still cold, phenylephrine is preferred because of its

		minimal dysrhythmogenic potential.
Positioning	Shoulder roll 180° table rotation and pad pressure points. eyes. Circuit extension tubes	Anesthetic gas hoses and all monitoring and vascular catheter lines are directed to patient's feet. Make sure that all will reach the foot of operating table.
	SCD	SCDs used to minimize DVT.
Deep hypothermia and CPB	Initial cooling: Circulating water Thermal blankets Ice packs SNP infusion	Surface cooling, and/or central cooling is begun as soon as induction is complete, using either a central venous heat exchange device (e.g., INNERCOOL) or thermal blankets above and below patient, ice packs and infusion of cold fluids during establishment of isovolemic hemodilution.
	Innercool-type device	A SNP infusion (if tolerated) will promote surface cooling.
	ACT monitoring	Once the aneurysm is exposed and the decision is made that CPB is required, systemic **heparinization** (load: 300 U/kg; maintenance: 100 U/kg/h) is established, and patient is put on CPB using fem-fem bypass and cooled to 18°C. During CPB cooling, the heart will usually fibrillate between 22–26°C. After 18°C is reached, the CPB unit is shut off to deflate the aneurysm; it may be activated and shut off several times during clipping to evaluate adequacy of the surgical occlusion of the aneurysm and to apply additional clips**. Total circulatory arrest time should not exceed 45 min**.
	Heparinization	
	Rewarming	When clipping is complete, CPB is resumed and warming instituted. Partial CPB is continued until normal cardiac rhythm is established, and body temperature reaches 36°C. Once partial CPB is D/C, patient will tend to cool unless vigorous efforts at warming are continued. Warming the OR and iv fluids, and use of warming lights, Bair-Hugger, and an Innercool-type device will facilitate the warming process. ACT analysis is performed to establish that heparin reversal is complete 5–10 min after protamine (1 mg/100 U heparin activity). Blood is sent for clotting studies, and Plts, FFP, and calcium gluconate are administered as needed. Hetastarch 6% is not used in these patients because of its potential for inducing a coagulopathy.
Complications	↓↓ BP 2° failure to maintain circulating volume Dysrhythmias 2° ↓ K^+ from diuresis and cold	

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Postoperative

Complications	HTN Vasospasm Intracranial hemorrhage, stroke Hypothermia Hypervolemia Coagulopathy DVT Seizures PE	HTN Rx: esmolol + SNP titrated to effect. Vasospasm Rx: fluid-loading Patient should be rewarmed to 36–37°C before terminating CPB. coag status. Sz Rx: Phenytoin (1 g iv slowly to avoid ↓ BP). WB: Phenytoin is incompatible with dextrose containing solutions.
Pain management	Meperidine (10–20 mg iv prn) Codeine (30–60 mg im q 4 h prn)	Meperidine minimizes postop shivering.
Tests	CT scan Coag panel	If any question about neurological status arises, a CT scan is performed postop. Coagulation studies are needed early postop to assure normal coagulation.

Suggested Readings

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Craniotomy for Cerebral Embolectomy

Surgical Considerations

Gary K. Steinberg
Robert L. Dodd

Description: While intravenous or endovascular intra-arterial thrombolysis is the current standard therapy for intracranial intravascular clots, embolic occlusion of a major intracranial vessel occasionally requires microsurgical embolectomy. In particular, when the embolus is a large atherosclerotic plaque or foreign body (such as a balloon or microcoil from endovascular treatment), surgery may be the treatment of choice. Because cerebral ischemia often proceeds to irreversible infarction before the surgeon can restore blood flow, early diagnosis is of the utmost importance, and several studies have demonstrated that the best results from embolectomy occur when the procedure is performed within 6 h following the onset of a neurologic deficit.

A standard craniotomy is fashioned as previously described for other lesions involving the vasculature at the skull base (see p. 4), and the occluded intracranial artery is exposed using microsurgical techniques. The involved arterial segment is isolated, temporarily occluded with miniature clips, and an arteriotomy is performed to remove the thrombus or embolus (Fig. 1.1-2). The arteriotomy is then closed and blood flow reestablished.

Usual preop diagnosis: Stroke; TIA; intracranial arterial occlusion; catheter embolization to intracranial artery

Summary of Procedures

Position	Supine or lateral decubitus
Incision	Frontal, temporal, or occipital
Special instrumentation	Microscopic instruments (fine forceps, miniature vascular clips, operating microscope)
Unique considerations	Neuroprotective agents during arterial segment occlusion (barbiturates, mannitol), mild hypothermia (33°C)
Antibiotics	Ceftriaxone 1–2 gm iv (avoid Ca^{++}-containing solutions)
Surgical time	3–4 h
Closing considerations	Avoid ↓BP (MAP 80–100 mmHg). Induced mild HTN (MAP 90–110) during temporary arterial occlusion.
EBL	100–250 mL
Postop care	Control BP (MAP 80–100 mmHg); start aspirin postop d 1; ICU: 1–2 d.
Mortality	5–10%
Morbidity	Intracerebral hemorrhageStroke MI: Rare Thromboembolism: Rare Respiratory failure: Rare Infection: Rare
Pain score	3–4

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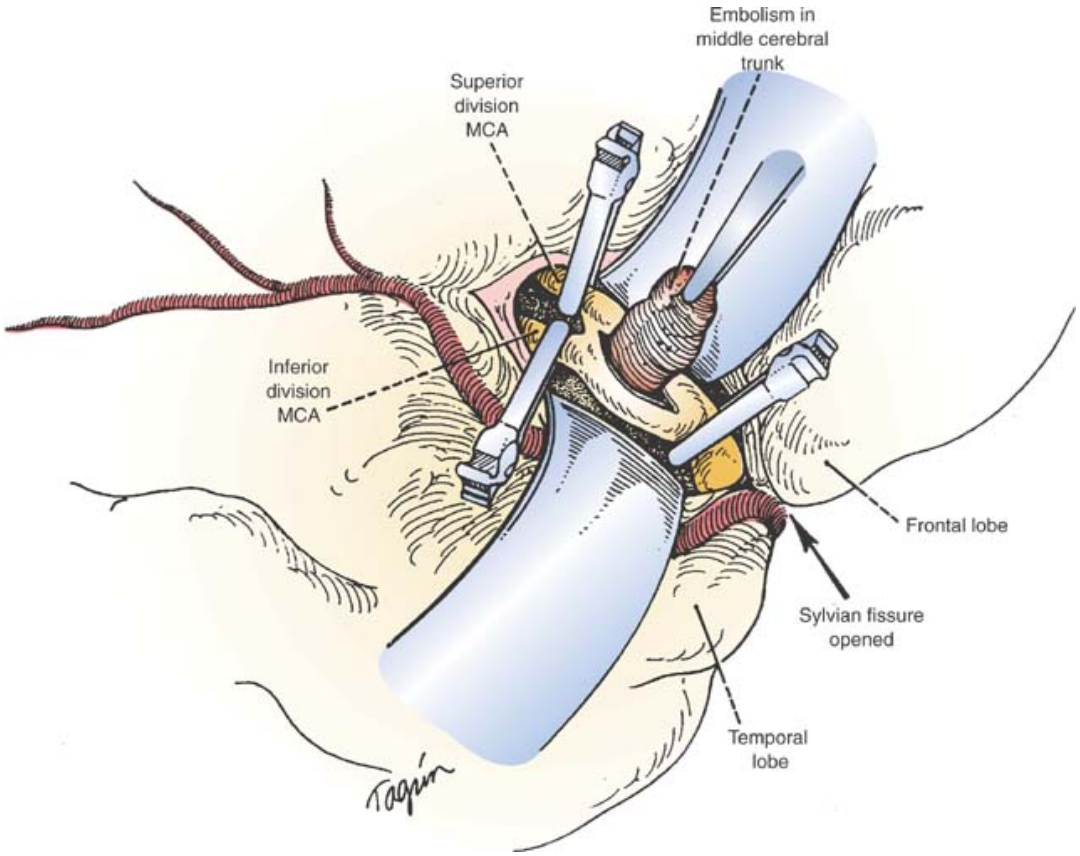

Figure 1.1-2. 2. Middle cerebral artery (MCA) embolectomy. Exposure of right MCA in the sylvian fissure and removal of an embolus from the MI segment. (Reproduced with permission from Ojemann RG, Ogilvy CS, Crowell RM, et al: *Surgical Management of Neurovascular Disease,* 3rd edition. Williams & Wilkins, Philadelphia: 1995.)

Patient Population Characteristics

Age range	50–80 yr
Male:Female	1:1
Incidence	Rare
Etiology	Atherosclerosis; carotid artery disease; atrial fibrillation; iatrogenic endovascular catheter complication
Associated conditions	HTN; CAD; PVD; carotid artery disease; hyperlipidemia; smoking; alcohol abuse; obesity; atrial fibrillation

Anesthetic Considerations

See Anesthetic Considerations for Craniotomy for Intracranial Aneurysms, p. 6.

Suggested Readings

1. Gomez CR, Orr SC, Soto RD: Neuroendovascular rescue: interventional treatment of acute ischemic stroke. *Curr Treat Options Cardiovasc Med* 2002; 4:405–19.

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Craniotomy for Intracranial Vascular Malformations

Surgical Considerations

Gary K. Steinberg

Description: Intracranial vascular malformations are congenital abnormalities that cause intracranial hemorrhage, seizures, headaches, progressive neurological deficits, or audible bruits. Intracranial vascular malformations comprise high-flow, arteriovenous malformations (AVMs); low-flow, angiographically occult vascular malformations (AOVMs), including cavernous malformations (a collection of enlarged capillaries with thin inelastic walls prone to leaking), “cryptic” AVMs, capillary telangiectasias and transitional malformations; and low-flow, venous angiomas (developmental venous anomalies). **Microsurgical resection** is the optimal treatment for these lesions, although preop endovascular embolization and preop or postop focused **stereotactic radiosurgery** (heavy particle or photon) may be useful adjuncts.

Most moderate-sized and large **AVMs** (> 3 cm diameter) are resected using a standard **scalp flap** and with **craniotomy** centered over the area of the AVM using image-guidance techniques. The patient is positioned appropriately to place the craniotomy site uppermost in the field and parallel to the floor. For example, a patient with a left frontal AVM would be positioned supine, head turned to the right, a left frontal or bicranial scalp flap raised and a left frontal craniotomy bone flap removed. A patient with a right medial occipital AVM would be positioned in the left lateral decubitus position with head turned semiprone and a right occipital scalp flap and craniotomy performed. Smaller AVMs (< 3 cm diameter), many low-flow AOVMs, and many deep-seated vascular malformations (AVMs and AOVMs) require a small, **stereotactic craniotomy.** This is performed by attaching fiducial markers or (less frequently) a stereotactic base frame to the patient's skull (using local anesthetic and sedation). Next, a CT or MRI scan is obtained. The location of the AVM in relation to the markers (or frame) is calculated, using a computer and stereotactic geometric principles. The patient is taken to the OR, intubated (fiber optically if a frame is used), and positioned for surgery. The surgical navigation system reference is attached to the headrest and microscope and calibrated. A small scalp flap and a small craniotomy (a few cm in diameter) can be fashioned precisely for microscopic exposure of the malformation. Microsurgical resection of brain stem and thalamic vascular malformations often necessitate special positioning. In frame-based surgery, a three-dimensional arc frame is fixed to the base frame, and coordinates are set to localize the vascular malformation within the brain.

Usual preop diagnosis: Cerebral AVM; dural AVM; cavernous malformation; angiographically occult vascular malformation; intracerebral hemorrhage; subarachnoid hemorrhage; seizures; epilepsy; progressive neurological deficit; migraine or vascular headaches

Summary of Procedures

	Standard Craniotomy (High-Flow AVM)	Stereotactic Craniotomy (Low-Flow AOVM)	Brain Stem/ Thalamic Vascular Malformations
Position	Supine, lateral, Concorde (modified prone) (Fig. 1.1-3)		Lateral, Concorde, or semisitting (Fig. 1.1-4)
Incision	Frontal, temporal, parietal, occipital, suboccipital, or combination		Suboccipital (midline), paramedian or occipital
	Operating microscope;		

	irrigating bipolar coagulation; radiolucent table and headrest.		
Special instrumentation	Sundt mini aneurysm and micro AVM clips. Access to femoral artery for intraop angiography.	+ Surgical navigation system	
Unique considerations	Induced hypotension (MAP 60–65 mmHg) during resection, use of neuroprotective agents (see Craniotomy for Aneurysms, pp. 6–7). Relaxed brain. Mild hypothermia (33°C); ± lumbar CSF drain.	Use of neuroprotective agents and relaxed brain (see Craniotomy for Aneurysms, pp. 8–9).	
Antibiotics	Ceftriaxone 1 gm iv (avoid Ca^{++}-containing solutions)		
Surgical time	4–10 h	2–5 h	3–6 h
Closing considerations	Maintain MAP 65–75 mmHg. Avoid ↑ venous pressure. For supratentorial vascular malformations, administer additional anticonvulsants; give loading dose of phenytoin (1 g iv for adults) if not previously on anticonvulsants.	Keep MAP 70–90 mmHg.	No anticonvulsants necessary
EBL	500–3000 mL	< 250 mL	
Postop care	ICU × 1–2 d. Maintain MAP 65–75 mmHg for 1 d. ICP monitoring, ventricular drain; normovolemic in ICU.	ICU × 1 d; normovolemic in ICU	
Mortality	1–10%, depending on AVM size, location, and venous drainage pattern	< 0.5%	< 2%
Morbidity	Overall: 5–30% Breakthrough bleeding: < 5% Intracranial hemorrhage Cerebral edema Stroke < 15% Hydrocephalus Massive blood loss: Occasional Thromboembolism: Rare Infection: Rare	< 5%	10–50% (transient) < 5%
Pain score	3–4	3–4	3–4

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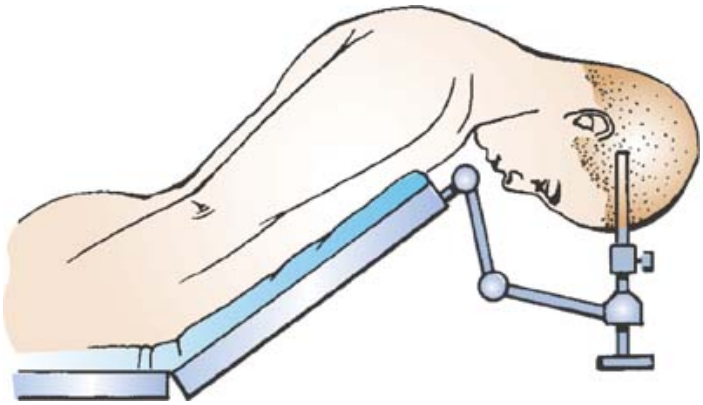

Figure 1.1-3. 3. Concorde (modified prone) position for resection of posterior fossa vascular malformations.

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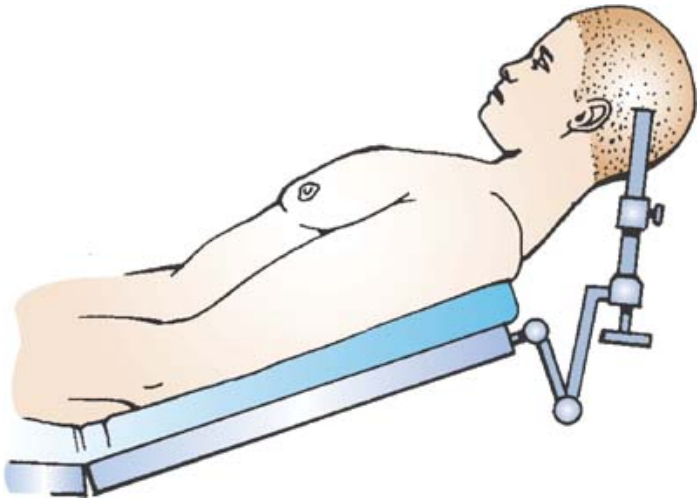

Figure 1.1-4. 4. Semisitting position for resection of deep posterior corpus callosum or thalamic vascular malformations.

Patient Population Characteristics

Age range	15–40 yr (most common), 41–60 yr (less frequent)
Male:Female	1:1
Incidence	0.5–1% of the U.S. population
Etiology	Congenital; traumatic for dural AVM
Associated conditions	Intracranial aneurysm (10% of AVM patients [up to 58% in some studies]); Von Hippel-Lindau disease; Rendu-Osler-Weber syndrome; familial cavernous malformation syndrome

Anesthetic Considerations

Preoperative

AVMs are direct arterial-to-venous communications without intervening normal capillary circulation. On histological exam, the vessel walls are thin and lack a muscular layer; consequently, the vessels exhibit loss of normal vasomotor control or responsiveness to changes in $PaCO_2$. **Stereotactic localization** is essential for safe excision of deep-seated AVMs (e.g., those located in the corona radiata, basal ganglia, visual center, cerebellar white matter, or corpus callosum). Untreated, the overall risk of rupture is 2–4% per year. AVMs > 6 cm in maximum diameter and located near eloquent areas of cortex or with deep venous drainage (grade IV and V)

have a high complication rate and may not be suitable for surgical resection.

Respiratory	Not usually significant unless patient has Hx of smoking, or has pulmonary aspiration as a result of a neurological deficit from an intracranial hemorrhage. **Tests:** As indicated from H&P.
Cardiovascular	Generally, these patients do not have other cardiovascular diseases. Occasionally, ECG changes are noted following intracranial hemorrhage and may represent subendocardial injury 2° catecholamine release. **Tests:** ECG; others as indicated from H&P.
Neurological	Presenting Sx depend on location and size of AVM, and whether it is a low- or high-flow lesion. Hemorrhage with resultant headache and/or neurological deficit is the most common Sx, although patients also may present with intractable seizure disorder, recurrent HAs, or Sx of cerebral ischemia 2° high-flow AV shunts, causing an intracerebral steal. Surgical treatment is essential to prevent future hemorrhage (incidence of 2–4%/yr) with substantial mortality (6–30%) or severe morbidity (15–80%). Unlike hemorrhages from an intracerebral aneurysm (generally subarachnoid), hemorrhages from an AVM are usually ventricular or intraparenchymal; hence, they are seldom associated with cerebral vasospasm. **Tests:** CTA; MRI; cerebral angiogram. Preop cerebral angiogram indicates size and location of the AVM, and whether it is likely to be a low- or high-flow lesion.
Hematologic	After surgery for AVM, it is fairly common for surrounding brain tissue to swell and resection sites to bleed. The cause of this is unknown, but it may be related to diversion of former AVM blood flow into the surrounding vasculature, producing a fragile hyperemic state. Thus, it is advisable to obtain coag studies preop and stage excision over more than one sitting when the AVM is large. **Tests:** Hct; PT; PTT; Plt count
Laboratory	CBC; other tests as indicated from H&P.
Premedication	If medication is desirable, small doses of midazolam (e.g., 1–3 mg iv) are useful. Detailed discussion with patient about the anesthetic plan, with appropriate reassurance, is essential.

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Intraoperative

Anesthetic technique: GETA. The goals of anesthesia for this operation are to: (a) ↓ $CMRO_2$ to lessen the dependence of normal brain on vessels feeding the AVM; (b) decrease intracranial volume (blood and tissue) to optimize surgical working space within the cranial compartment and minimize the need for surgical retraction of brain tissue; and (c) maintain a somewhat decreased (10–20% below normal) CPP to lessen blood loss during excision of the AVM (CPP = cerebral arterial pressure minus cerebral venous pressure or ICP, whichever is greater). For stereotactic surgery, scalp localizing markers are attached. The patient is then sent to MR or CT, where the exact coordinates defining the AVM and critical adjacent structures are established. The patient is brought to OR and anesthesia is induced.

Induction	STP 2–5 mg/kg or propofol 2–3 mg/kg iv provides amnesia and ↓ CBV by inducing cerebral vasoconstriction. Fentanyl 7–10 mcg/kg iv blunts the response to laryngoscopy and provides analgesia for the first hours. High-dose opiates used as a primary anesthetic technique do not alter CBF or $CMRO_2$ enough to provide any special benefits. Vecuronium (0.15 mg/kg), rocuronium (0.6–1 mg/kg), or pancuronium (0.1 mg/kg), provide muscle relaxation for intubation and patient positioning. Occasionally the patient may be in a stereotactic frame and ET intubation must be

		accomplished before anesthesia is induced, because the frame partially occludes the mouth, making conventional laryngoscopy impossible. Awake oral fiber optic intubation of the trachea is the easiest method for accomplishing this (see p. B-5).
Maintenance		Isoflurane ≤ 1% or sevoflurane ≤ 2% (limit to 1/2 MAC maximum if EP monitoring is used) with 1:1 O_2/N_2O. With EP monitoring, a remifentanil infusion (0.05–0.15 mcg/kg/min) may be necessary to supplement the anesthetic. Propofol (75–150 mcg/kg/min) by continuous infusion may be administered to provide ↓ CBV and ↓ $CMRO_2$ and allow for reduction in inhalation agent concentration or elimination of N_2O. Mild hypothermia (33°C) provides additional cerebral protection (see below). Additional neuromuscular blocking drugs are usually not necessary, but can be administered if patient movement is of concern. Induced hypotension is often useful during AVM resection. Following resection, induced hypertension (e.g., 90 mmHg) may be requested to inspect hemostasis.
		With the start of dural closure, consider changing the anesthetic to low-dose sevoflurane (e.g., 0.5%) in 50% N_2O, supplemented with a low-dose remifentanil infusion (e.g., 0.05 mcg/kg/min). A propofol infusion (if used) should be discontinued at the start of the scalp closure. The patient's BP generally will increase and titration of β-adrenergic blocking drugs (e.g., labetalol or esmolol) and/or vasodilators (e.g., SNP) may be needed. (See Control of BP, below.) The inhalation agents can be D/C'd at the time of dressing application.
Emergence		Most patients will breathe spontaneously and can be extubated uneventfully while on the remifentanil infusion. If the brain has not been injured by the surgical procedure, the patient should awaken within 10 min after cessation of remifentanil administration. Close regulation of BP is essential. If the patient begins to cough on ETT, either it should be removed or cough reflex suppressed with iv lidocaine (0.5–1 mg/kg). Patient is placed in bed in a 30° head-up position and transported to ICU for monitoring overnight. Supplemental O_2 should be administered and close regulation of BP maintained (typically at 10% below baseline values). Prophylactic antiemetics (e.g., metoclopramide 10–20 mg and ondansetron 4 mg) should be given 30 min before extubation.
Blood and fluid requirements	IV: 14–16 ga × 2 NS @ < 10 mL/kg + UO Massive blood loss possible Maintain euvolemia Maintain colloid oncotic pressure	If blood volume is normal, NS—not to exceed 10 mL/kg beyond that required to replace UO—is given. If hypovolemic, albumin 5% is given if Hct > 30%; combinations of albumin and blood, if Hct < 30%.
Hypothermia	Thermal blankets Cool air blower Cold OR InnerCool-type device Bladder irrigation	Mild hypothermia (33°–34°C) is used in some centers for cerebral protection and to ↓ brain size. This level of hypothermia does not interfere appreciably with coagulation, nor is it generally associated with cardiac dysrhythmias. Warming is begun as soon as possible after lesion resection and is greatly facilitated by the use of a central warming device (e.g., InnerCool).
Brain Relaxation (control of ICP)	Hyperventilate to $PaCO_2$ = 30 mmHg or $PetCO_2$ = 25 mmHg. Limit isoflurane ≤ 1%. Maintain euvolemia Consider propofol infusion to replace N_2O Mannitol 0.5–1 g/kg Furosemide 0.3 mg/kg Lumbar CSF drainage	↓ $PaCO_2$ → ↓ cerebral vascular volume → ↑ working space and lessens need for vigorous retraction of brain tissue. ↓ $PaCO_2$ also improves the regional distribution of CBF by preferentially diverting blood to potentially ischemic areas of the brain. If AVM is superficial, decreasing brain volume is less important, and the first four techniques listed (at left) are usually sufficient. If AVM is deep, the additional listed therapies may be needed. Direct monitoring is essential for rapid control of BP. Transducer should always be placed at the level of the head rather than

		the heart, since CPP is arterial pressure at the brain level minus CVP or ICP, whichever is higher. Monitoring CVP via a near right atrial catheter is desirable in virtually all patients to assess adequacy of fluid therapy, for infusion of vasoactive drugs and aspiration of VAE. Localization of the catheter can be determined by CXR, ECG tracing, noting P-wave changes, or pressure-wave contour and value as the catheter is withdrawn from the right atrium. **NB**: If patient has a high-flow AVM causing a large AV shunt, venous blood may appear arterialized (bright red) during central venous catheterization.
Monitoring	Standard monitors (see p. B-1). Core temp: deep esophageal best Arterial line CVP line, triple lumen UO Blood glucose (100–180 mg%)	
Control of BP	Isoflurane/sevoflurane Esmolol infusion SNP infusion Maintain normovolemia. Labetolol (emergence)	Close regulation of BP during induction and prior to excision of AVMs is important, both to prevent bleeding (↑ BP) and to avoid ischemia 2° steal (↓ BP). After surgical excision is under way, however, modest decreases in MAP (≤ 20% below normal) using isoflurane, alone or in combination with esmolol and/or SNP, should be used to prevent excessive bleeding. Responses to vasoactive drugs are much easier to regulate if the patient is euvolemic.
Positioning	Shoulder roll Three-point fixation and pad pressure points. eyes. 180° rotation SCDs	For most AVMs, patient is supine, head turned laterally in three-point fixation, a roll under shoulder on the side of operation (Fig. 1.1-5). Anesthetic hoses and all monitoring/vascular catheter lines are directed toward patient's feet or side. Make sure that all will reach the foot of operating table. SCDs are used to minimize DVT. Remifentanil (2–4 mcg/kg iv bolus) should be used to minimize ↑ BP during skull pinning.

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Postoperative

Complications	Neurological deficits	If these complications occur, the patient likely will have to be reintubated and transported to the CT scanner for further neurological evaluation or possible reoperation.
	Cerebral edema and ↑ ICP Intracerebral hemorrhage	Careful regulation of BP is essential to avoid postop hemorrhage.
	Seizures	*Sz Rx: Phenytoin (1 g loading dose). **NB**: Incompatible with dextrose-containing solutions.
Pain management	Meperidine 10–20 mg iv/70 kg Codeine (30–60 mg im q 4 h prn)	At this dose, meperidine minimizes postop shivering without producing excessive sedation.
Tests	CT scan	If neurological status changes.

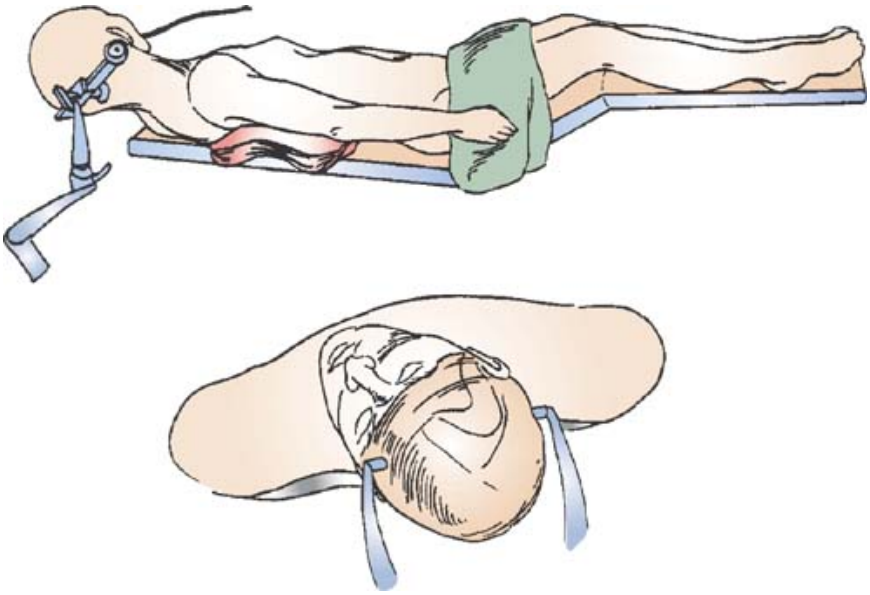

Figure 1.1-5. 5. Supine position, head elevated above heart, turned 30–45° to side, vertex dropped for approach to anterior circulation aneurysms and frontal vascular malformations. (Reproduced with permission from Long DM: *Atlas of Operative Neurosurgical Technique*, Vol 1. Williams & Wilkins, Philadelphia: 1989.)

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Craniotomy for Extracranial-Intracranial Revascularization (EC-IC BYPASS)

Surgical Considerations

Gary K. Steinberg
Robert L. Dodd

Description: Extracranial-intracranial (EC-IC) revascularization procedures are performed when: (a) deliberate occlusion of a major cervical artery (carotid or vertebral) is necessary and inadequate collateral CBF is available or (b) stenosis or occlusion of major cervical or intracranial arteries causes TIA or stroke, despite the use of maximum medical therapy (e.g., antiplatelet drugs, heparin, or Coumadin). The chief causes of stenosis or occlusion are atherosclerotic disease, radiation injury, and moyamoya disease. The subset of patients who benefit from revascularization are those whose radiographic and metabolism studies demonstrate that they have ↓ CBF and poor or absent vascular reserve. The most important surgical considerations include site of stenosis, adequacy of donor graft, and patient age.

A standard craniotomy is fashioned as previously described for other lesions involving the vasculature around the skull base, and the intracranial site of anastomosis is exposed using microscopic techniques. Typically, a donor extracranial scalp artery (e.g., superficial temporal artery) is anastomosed to an intracranial artery (e.g., middle cerebral artery) distal to the site of stenosis. When scalp vessels are inadequate, an interposition vein segment can be sutured to a cervical artery and then anastomosed to the designated intracranial artery. The most common EC-IC procedure is a **superficial temporal artery (STA)-to-middle cerebral artery (MCA) branch anastomosis** (See Figs 1.1-6, 1.1-7, 1.1-8, 1.1-9 and 1.1-10). Other grafts include STA-to-posterior cerebral artery, STA-to-superior cerebellar artery, occipital artery-to-posterior inferior cerebral artery or interposition saphenous vein segment graft from the cervical external carotid artery to the middle cerebral artery, posterior cerebral artery, or superior cerebellar artery.

Variant procedure or approaches: Indirect revascularization is commonly employed in patients in whom the graft vessels are too small for direct anastomosis. Vascular source tissues include a temporalis muscle flap and a flap of dura folded under so that its outer vascular surface is apposed to the cortical surface. **Encephalo-duro-arterio-synangiosis (EDAS)** is an indirect variant procedure wherein the STA is dissected circumferentially with its *(Print pagebreak 24) (Print pagebreak 25)* adventitia in the scalp, left in continuity and laid on the surface of the brain after opening the dura. **Omentum-to-brain transposition** is a rarely used variant, wherein the omentum, with its luxuriant blood supply, is lengthened, left attached to the right gastroepiploic artery, tunneled subcutaneously in the chest and neck, and laid over a large area of poorly vascularized cerebral cortex after opening the dura. Sometimes a free omental graft is transposed to the brain by anastomosing the omental gastroepiploic artery and vein to the superficial temporal artery and vein. Revascularization is induced by angiogenesis factors and growth substances secreted by the brain and omentum.

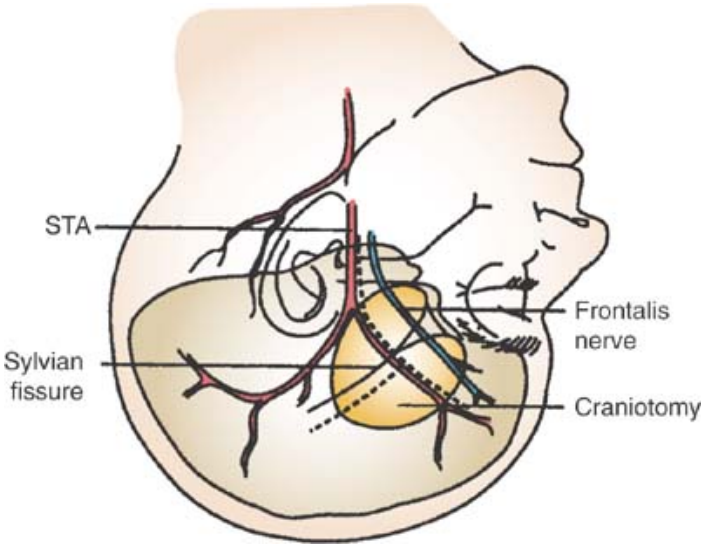

Figure 1.1-6. 6. Typical skin incision for EC-IC bypass. The main incision is planned over the superficial temporal artery (STA) with a T extension to allow exposure of the bone. (Reproduced from Chang SD, Steinberg GK: Superficial temporal artery to middle cerebral artery anastomosis. *Tech Neurosurg* 2000; 6(2):86–100.)

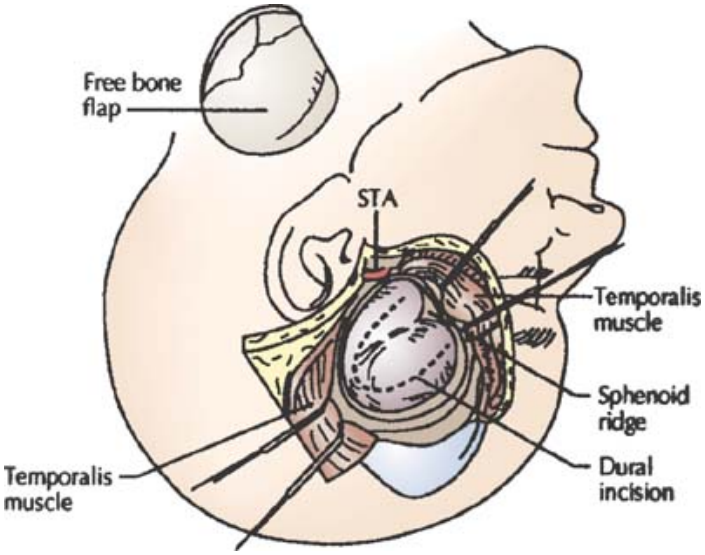

Figure 1.1-7. 7. After the STA is dissected out, the temporalis muscle is divided and the bone flap is made to allow exposure of the brain over the anterior sylvian fissure. (Reproduced from Chang SD, Steinberg GK: Superficial temporal artery to middle cerebral artery anastomosis. *Tech Neurosurg* 2000; 6(2):86–100.)

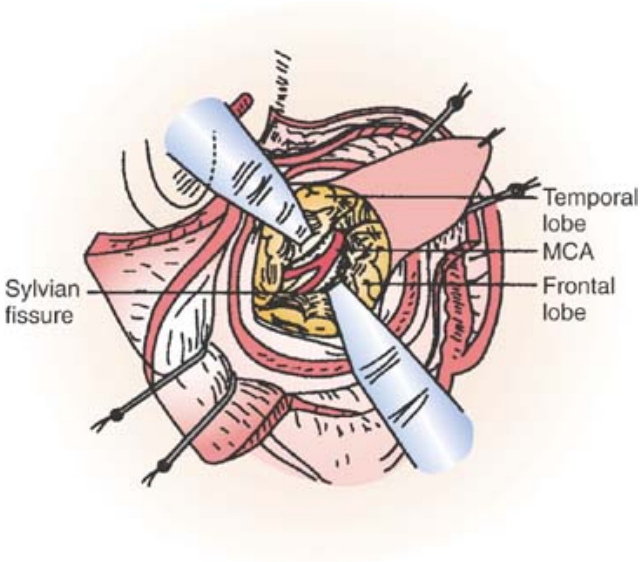

Figure 1.1-8. 8. A middle cerebral artery (MCA) recipient vessel is identified. The sylvian fissure can be split to allow identification of a larger, more proximal branch of the MCA, preferably an M3 branch. (Reproduced from Chang SD, Steinberg GK: Superficial temporal artery to middle cerebral artery anastomosis. *Tech Neurosurg* 2000; 6(2):86–100.)

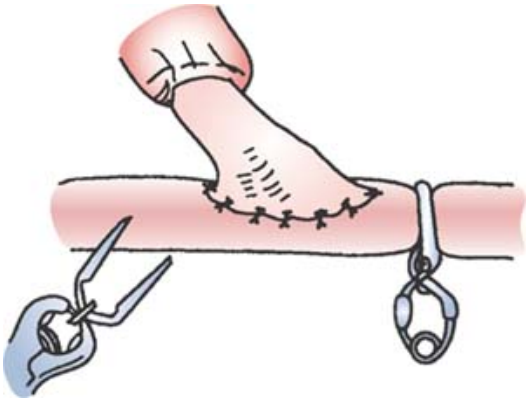

Figure 1.1-9. 9. When the anastomosis is complete, the vascular clips are removed. (Reproduced from Chang SD, Steinberg GK: Superficial temporal artery to middle cerebral artery anastomosis. *Tech Neurosurg* 2000; 6(2):86–100.)

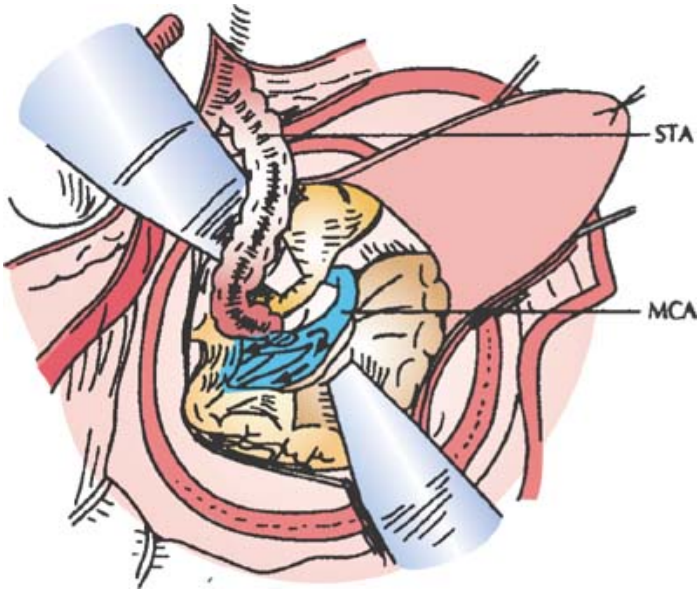

Figure 1.1-10. 10. The completed anastomosis shows the STA positioned such that flow is directed toward the proximal portions of the MCA. (Reproduced from Chang SD, Steinberg GK: Superficial temporal artery to middle cerebral artery anastomosis. *Tech Neurosurg* 2000; 6(2):86–100.)

Usual preop diagnosis: Moyamoya disease (cerebral ischemia due to occlusion of vessels at base of the brain); stroke; TIA; carotid artery stenosis (inaccessible to carotid endarterectomy); carotid artery occlusion; middle cerebral artery stenosis or occlusion; vertebral artery stenosis or occlusion; basilar artery stenosis or occlusion.

Summary of Procedures

	EC-IC Bypass	EC-IC Bypass with Vein Graft	Indirect Procedures
Position	Supine or lateral decubitus		
Incision	Frontal, parietal, temporal, occipital, or a combination of these, depending on area to be vascularized.	+ Medial aspect of leg and thigh for harvesting greater saphenous vein	+ possible abdominal incision for harvesting omentum and chest/ neck incision for tunneling

Special instrumentation	Microscopic instruments; microvascular Doppler to identify scalp donor artery course and confirm graft patency.	+ Tunneling instruments	
Unique considerations	Neuroprotective agents (barbiturates, mannitol) and induced mild HTN (MAP 90–110 mmHg) during cross-clamp of recipi-ent intracranial artery. Avoid excessive brain relaxation. Mild hypothermia (33°C). Dexamethasone 8–12 mg iv.	+ Attention to proper alignment of vein when tunneled, to avoid kinking; heparinization if major cervical artery (carotid, vertebral) is temporarily occluded.	+ Avoid devascularizing source tissue during dissection or compromise to blood supply during closure.
Antibiotics	Ceftriaxone 1–2 gm iv q d (avoid Ca^{++} containing solution)		
Surgical time	3–5 h		
Closing considerations	Careful attention to hemostasis. Avoid compromise of graft with dural closure, bone replacement or scalp closure.		
EBL	< 100 mL		100–500 mL
Postop care	Start aspirin on POD 1; monitor for subdural hygroma (CSF fluid collection in subdural space); ICU × 1 d.		
Mortality	< 0.5%		
Morbidity	Transient neurologic deficit: 20–40% Subdural hygroma: Rare Wound infection: Rare Stroke: Rare		Abdominal hernia: Rare
Pain score	3	3	3

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Patient Population Characteristics

Age range	40–80 yr; 2–20 yr for moyamoya disease
Male:Female	1:1 for atherosclerotic disease; 1:1.4 for moyamoya disease
Incidence	Thromboembolic stroke common, but indications for EC-IC bypass rare; 1/million/yr for moyamoya disease
Etiology	Atherosclerosis; embolism from heart or carotid artery
Associated conditions	HTN; CAD; PVD; hyperlipidemia; smoking; alcohol abuse; obesity; moyamoya disease

Anesthetic Considerations

Preoperative

Patients range in age from pediatric (classic moyamoya disease) to adult. These patients may have significant pre-existing neurologic deficits. HTN in these patients is often an adaptation to cerebral vascular insufficiency → hypoperfusion. Inappropriate

treatment of this compensatory HTN → cerebral ischemia and stroke. Patients with symptomatic moyamoya disease or bilateral carotid stenosis or occlusion may be good candidates for EC-IC bypass, especially since there are no alternative forms of therapy that have proven to be effective. Most patients present with h/o TIAs or stroke and most are receiving antiplatelet medication.

Respiratory	None unless patient has Hx of smoking or has sustained pulmonary aspiration 2° neurological deficit. **Tests:** As indicated from H&P.
Cardiovascular	HTN is a common adaptive mechanism to maintain cerebral perfusion; therefore "normalization" of BP preop may be undesirable. Adult patients may have generalized vascular disease, including CAD, so a careful cardiac Hx, physical exam, and ECG analysis should be done. If findings are positive, consider a more complete evaluation, including ECHO and coronary angiography. Be aware that cardiac insufficiency is the cause of about half of the deaths in patients with cerebrovascular disease. **Tests:** Consider ECG, others as indicated from H&P.
Neurological	Patients present with Sx of focal ischemic lesions. Cerebral angiography is necessary to r/o other causes of TIAs and characterize collateral circulation. Regional CBF studies are generally not helpful, because measurement does not distinguish between low flow due to cerebrovascular obstruction from that due to low metabolic demands 2° prolonged cerebral ischemia. Pre-existing deficits should be well-characterized. **Tests:** CTA; MRI; angiogram
Hematologic	For most patients: a platelet-suppressive dose of aspirin should be continued through the day of surgery; drugs affecting PT/PTT should be D/C'd at least 1 wk before surgery to avoid excessive bleeding; Clopidogrel (Plavix) should be D/C'd 5 d before surgery. **Tests:** Hct; PT; PTT; hemogram
Laboratory	Tests as indicated from H&P.
Premedication	If medication is desirable, small doses of midazolam (e.g., 1–3 mg iv) are useful. Detailed discussion with the patient about the anesthetic plan with appropriate reassurance is essential.

Intraoperative

Anesthetic technique: GETA. The goals of anesthesia for this procedure are to: (a) maximize flow to the ischemic area through collateral channels by maintaining BP at normal or somewhat elevated values; (b) ↑ tolerance of the brain to ischemia by decreasing $CMRO_2$ with the use of mild hypothermia (33–34°C) and barbiturate therapy; (c) ↓ intracranial volume (blood and tissue) to optimize working space within the cranial compartment, thereby minimizing the need for surgical retraction of brain tissue; and (d) provide adequate surgical anesthesia.

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Induction	STP 2–5 mg/kg (or propofol 1–2 mg/kg iv) to provide amnesia and ↓ cerebral blood volume by inducing cerebral vasoconstriction. Fentanyl 7–10 mcg/kg iv to blunt response to intubation and provide analgesia for the first hours of surgery. Pancuronium (0.1 mg/kg; helps offset ↓ HR/BP 2° induction drugs), vecuronium 0.15 mg/kg, or rocuronium 0.7–1.0 mg/kg to provide muscle relaxation for tracheal intubation. Bolus ephedrine and/or phenylephrine are often necessary to maintain CPP during induction.
Maintenance	Isoflurane ≤ 1% or sevoflurane ≤ 2% (1/2 MAC maximum if EP monitoring is used) with 1:1 O_2/N_2O. A phenylephrine infusion is usually necessary to maintain CPP during the procedure. STP (2–5 mg/kg) is given just before surgical occlusion of the cerebral vessel in preparation for anastomosis. A bolus dose of phenylephrine and/or ephedrine is often given concomitantly to offset the hypotensive effects of STP. If mild hypothermia was employed, rewarming should start as soon as revascularization is

	complete.	
Emergence	With the start of dural closure, consider changing the anesthetic to low-dose sevoflurane (e.g., 0.5%) in 50% N_2O, supplemented with a low-dose remifentanil infusion (e.g., 0.05 mcg/kg/min). A propofol infusion (if used) should be discontinued at the start of the scalp closure. The patient's BP generally will increase and titration of β-adrenergic blocking drugs (e.g., labetalol or esmolol) and/or vasodilators (e.g., SNP) may be needed. (See Control of BP, below.) The inhalation agents can be D/C'd at the time of dressing application. Most patients will breathe spontaneously and can be extubated uneventfully while on the remifentanil infusion. If the brain has not been injured by the surgical procedure, the patient should awaken within 10 min after cessation of remifentanil administration. Close regulation of BP is essential. If the patient begins to cough on ETT, either it should be removed or cough reflex suppressed with iv lidocaine (0.5–1.0 mg/kg). Patient is placed in bed in a 30° head-up position and transported to ICU for monitoring overnight. Supplemental O_2should be administered and close regulation of BP maintained (typically at 10% below baseline values). Prophylactic antiemetics (e.g., metoclopramide 10–20 mg, ondansetron 4 mg) should be given 30 min before extubation.	
Blood and fluid requirements	IV: 18 ga × 2 NS @ < 10 mL/kg + UO	In hypothermic patients, IV drugs should be administered through the central line to ensure timely entry into the central circulation.
	CVP (triple-lumen)	CVP use low volume (1 mL) extension for drug infusions. Reserve distal lumen for pressure monitoring and air aspiration.
Control of brain volume (ICP)	Avoid hypovolemia ↓ isoflurane ≤ 0.5%, or sevoflurane < 1% N_2O ≤ 50% ± Furosemide 10–20 mg iv ± Mannitol 0.5–1.0 g/kg	Maintain normovolemia. Generally, vigorous control of brain volume is not necessary since surgeon is working with cerebral vessels on the surface of the brain. Keep $PaCO_2$= 30 mmHg. Excessive hypocarbia may cause unwanted cerebral vasoconstriction in these patients.
Monitoring	Standard monitors (see p. B-1). Core temperature Arterial line CVP (triple-lumen catheter) UO	Core temperature is best approximated by a deep esophageal temperature probe. Bladder temp tends to lag behind core temp. The distal CVP lumen is used for pressure monitoring.
Control of BP	Maintain normal BP (MAP). Phenylephrine infusion	Maintenance of normal MAP is important because cerebral autoregulation is often impaired in these patients, particularly during temporary occlusion of the surgical vessel being anastomosed. If a vasoconstrictor is needed, a pure α-adrenergic agonist, such as phenylephrine, is preferred because it has minimal dysrhythmogenic potential.
	Normovolemia	Responses to vasoactive drugs are much easier to regulate if a normal blood volume has been maintained throughout the anesthetic period.
Positioning	and pad pressure points. eyes. Shoulder roll SCDs	Anesthetic hoses and monitoring and vascular lines directed to patient's feet where anesthesiologist is positioned during surgery. Used to minimize DVT. Surface cooling is quite effective in patients with high surface-to-volume ratios, and should be started as soon as induction of

Hypothermia	Cold-water circulating blankets. Core temp goal: 33–34°C. InnerCool-type device Bladder irrigation	anesthesia is complete, using a cold-water circulating blanket underneath and above the patient. When anastomosis is nearly complete, vigorous efforts at warming, including bladder irrigation, are initiated. Cooling, and especially rewarming are greatly facilitated by the use of a central heat-exchange device (e.g., InnerCool) placed in the inferior vena cava through a femoral vein introducer.
Complications	Seizures Stroke Hemorrhage at anastomosis Brain swelling	*Sz Rx: Phenytoin (1 g loading dose). **NB**: Incompatible with dextrose-containing solutions. May be 2° hyperemia in revascularized territory.

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Postoperative

Complications	Localized scalp necrosis	Major complications uncommon; localized scalp necrosis unique to this procedure.
	Brain swelling	May be 2° to delayed hyperemia. Careful control of BP is important.
Pain management	Meperidine (10–20 mg iv prn) Codeine (30–60 mg im q 4 h prn)	Useful to suppress shivering Postop pain is usually not severe.
Tests	Cerebral angiogram	Cerebral angiography documents patency of graft and collateral flow.
	Regional blood flow studies	Some centers have the capability of performing regional blood flow studies.
	CT scan	If any question about neurological status, a CT scan is performed.

Suggested Readings

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Craniotomy for Tumor

Surgical Considerations

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Description: Many classification systems exist for brain tumors. Generally, brain tumors can be classified by their location and a differential diagnosis can be generated based on tumor location and patient age. Brain tumors are either supratentorial or infratentorial (Table 1.1-2), or intraaxial or extraaxial (Table 1.1-3). The surgical approach depends on the location of the lesion, the need for brain relaxation, and whether exposure will require brain resection. Patient positioning generally depends on the location and surgical approach to the tumor (Table 1.1-4). Once positioned, the patient's head typically is placed in a Mayfield pin fixation system to prevent head movement during surgery and to allow for intraoperative image-guided stereotactic navigation.

Several types of incisions are used for these procedures. **Linear incisions** can be used to resect small tumors over the convexity or when using a midline approach to the posterior fossa, and often have the advantage of a more rapid wound closure. **Curvilinear** or **horseshoe-shaped incisions** are commonly used for larger tumors. After the skull is exposed, burr holes are made with a drill and the bone flap is cut with the craniotome. Some surgeons routinely use a **free-bone flap**, in which the bone is completely removed and stored for the duration of the case. Other surgeons turn an **osteoplastic flap**, where the bone is left attached to muscle and/or pericranium to keep it partially vascularized. When performing certain posterior fossa resections (e.g., a retromastoid craniotomy or low suboccipital craniotomy), the surgeon may choose to remove the bone without replacement, performing a **craniectomy** instead of a craniotomy. After the bone is removed, a few small holes are drilled near the edge of the craniotomy. These holes are used to suspend the dura using sutures. This helps prevent blood from accumulating in the epidural space during the rest of the case. The dura is then opened either in a stellate or curvilinear fashion. The method of dural opening generally is based on the size of the bone opening and its proximity to venous sinuses. The surgeon then proceeds with tumor removal if it is on the surface or with brain retraction/resection if the tumor is deep to the surface. At this point, the surgeon may request anesthetic interventions for brain relaxation (osmotic diuresis, hyperventilation) and specific BP control. After the tumor is removed, hemostasis is achieved and the dura is closed. The bone flap is replaced and fixated, the galea is closed with sutures, and the skin incision is closed with staples. During closure, the surgeon may ask for specific BP parameters to ensure that there is hemostasis in the resection bed and surgical field. Patients typically are extubated after cranial surgery, because it is paramount to obtain a neurologic exam as soon after surgery as possible. Patients undergoing a craniotomy almost always require a postoperative ICU course.

Table 1. 1-2. Common Tumor Location (Supratentorial vs Infratentorial)

Supratentorial	Infratentorial
Metastatic tumors	Metastatic tumors
Astrocytoma/glioblastoma	Acoustic neuromas
Oligodendroglioma	Meningiomas
Ependymoma	Hemangioblastoma

Meningioma	Medulloblastoma
Choroid plexus papilloma	
Craniopharyngioma	
Primitive neuroectodermal tumor (PNET)	

Table 1. 1-3. Intraaxial (Within-the-Brain Parenchyma) vs Extraaxial (Outside-the-Brain Parenchyma)

Intraaxial Tumors	Extra-axial Tumors
Astrocytoma/glioblastoma	Meningioma
Metastatic tumors	Acoustic neuroma
Oligodendroglioma	Choroid plexus papilloma
Ependymoma	Craniopharyngioma
Hemangioblastoma	Hemangiopericytoma
Medulloblastoma	
PNET	

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Table 1. 1-4. Common Patient Positions Based on Tumor Location

Supine: Most supratentorial tumors in the frontal, temporal, or anterior parietal lobe. Tumors of the lateral ventricles and 3rd ventricle. Tumors of the anterior 2/3 of the interhemispheric fissure.
Lateral: Tumors of the posterior or lateral parietal lobe, posterior temporal lobe, cerebellopontine angle, or lateral cerebellum. Tumors of the posterior ventricular horn.
Prone: Tumors of the occipital lobe, most midline cerebellar tumors, and tumors of the 4th ventricle. Tumors of the posterior 1/3 interhemispheric fissure and the tentorium.
Sitting: Tumors of the pineal region. Tumors of the 4th ventricle or midline cerebellum.

Electrophysiologic monitoring detects changes in SSEP, brain stem auditory evoked potentials (BAEP), and motor cranial nerve EMGs. Such monitoring is commonly used for tumors of the brain stem and skull base, or when resecting tumors in critical locations. Early changes in electrophysiologic monitoring potentials may alert the anesthesiologist to manipulate BP and may help the surgeon to further define the extent of safe retraction and tumor resection to minimize the likelihood of a postoperative deficit.

Image-guided navigation involves the use of a computerized workstation that can track the position of specific instruments before and during the operation. This allows the surgeon to: (a) plan appropriate skin and bone openings, (b) choose an optimal trajectory to the tumor, and (c) achieve a volumetric resection of the tumor. Image-guided navigation involves obtaining a CT or MRI scan prior to the start of the surgical procedure; the time required to obtain this study is usually offset by a shorter operative time. Data from the operative microscope can also be incorporated into the navigation system to aid in tumor dissection.

Mild hypothermia is often utilized when resecting tumors in eloquent areas of the brain, because hypothermia has been shown to provide neuroprotection.

CSF drainage usually is accomplished by use of a lumbar drain. Lumbar drainage typically is used for tumors in the cerebellopontine angle, in the pineal region, or tumors associated with significant edema. CSF drainage can also be accomplished by placing a ventriculostomy or by draining various cisterns during the operation.

Awake craniotomies typically are reserved for tumors adjacent to or within the speech areas of the dominant cortex. Patients are sedated during cranial opening, and are awakened once the dura is open. Direct cortical stimulation is performed to determine the relationship between the tumor and the speech centers. The surgeon then maps out eloquent and non-eloquent areas of the brain prior to beginning the tumor resection. The patient continues to converse during the tumor resection to ensure that language function is not altered.

Interstitial chemotherapy or brachytherapy can be performed after the tumor resection by implanting chemotherapy impregnated wafers or radioactive seeds in the resection cavity. Additionally, a balloon tip catheter can be placed in the resection bed and injected with radioactive material at a later date.

Variant procedure or approaches: Patient position varies depending on tumor location and surgeon preference (Table 1.1-4). Tumors on the convexity, or surface, of the brain may require minimal brain relaxation or exposure. Deep tumors, or tumors around

the brain stem or skull base, may require substantial brain relaxation and retraction for optimal exposure. For deep tumors or tumors within or adjacent to critical structures, the operating microscope is commonly used. Large vascular tumors may undergo preoperative embolization of the tumor blood supply in order to minimize intraoperative blood loss.

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Usual preoperative diagnosis: Glioma; glioblastoma multiforme; astrocytoma; oligodendroglioma; ependymoma; PNET; meningioma; craniopharyngioma; choroid plexus papilloma; hemangioblastomas; medulloblastoma; acoustic neuroma; brain metastasis; hemangiopericytoma

Summary of Procedures

Position	Supine, lateral, prone, or sitting, based on location of tumor
Incision	Linear or curvilinear, based on location of tumor
Special instrumentation	Operating microscope, laser, CUSA, electrophysiologic monitoring, image-guided navigation, lumbar drain, ventriculostomy
Unique considerations	ETT must be taped securely in a location satisfactory to the surgeon; anode or RAE tube is helpful in certain situations. Decadron is usually given preoperatively and dosed every 6 h to minimize brain edema. Brain relaxation techniques may be required. The patient with ↑ ICP may require special consideration for induction of anesthesia. Awake craniotomy (see p. 34) may be required for tumors involving eloquent brain areas.
Antibiotics	Ceftriaxone 1–2 g iv (avoid Ca^{++}-containing solutions)
Surgical time	3–5 h typically
Closing considerations	Possible requirement for dural graft. Drain often left in the epidural or subgaleal space. Good BP control during closing and extubation to prevent hemorrhage into tumor resection bed.
EBL	50–500 mL (Meningiomas and renal cell mets are highly vascular tumors and blood loss may be substantial.)
Postoperative care	ICU or close observation unit X 1–3 d. Fluid and electrolytes require frequent monitoring. BP may need to be controlled with antihypertensives.
Mortality	0–5% (mortality higher for tumors in critical locations)
Morbidity	Infection: 1% Neurological: neurologic disability, nerve injury: 0–10% CSF leak: 1–3% Venous sinus injury, air embolus Endocrine disorder Massive blood loss
Pain score	2–7

Patient Population Characteristics

Age range	Infant to 85 yr (usually 20–60 yr)
Male:Female	1:1
Incidence	Common neurosurgical procedure (40,000 dx/yr in the United States)
Etiology	Neoplastic

Anesthetic Considerations

(Procedures covered: craniotomy for tumor; craniotomy for skull tumor)

Preoperative

Typically this is a healthy patient population, apart from Sx attributable to intracranial pathology (↑ ICP, Sz, HA, N/V, visual disturbances). Discuss surgical approach, positioning, tumor type and potential for blood loss with surgeon. These patients are often receiving steroids to ↓ ICP.

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Respiratory	Occasionally neurogenic pulmonary edema may occur. H/o chemotherapy or radiation therapy may affect pulmonary function and airway management. Otherwise no special considerations, unless indicated from H&P.
Cardiovascular	Benign or malignant brain tumors cause edema formation in adjacent normal brain tissue, which may →↑ ICP. If ICP increases sufficiently to cause herniation of the brain stem, patients develop the "Cushing triad" of HTN, bradycardia, and respiratory irregularity. These changes will resolve when ICP is reduced, so vigorous attempts to regulate BP and HR prior to craniotomy are not warranted. After the diagnosis of brain tumor is made, most patients are placed on high-dose steroid therapy to lessen edema in surrounding normal brain. Steroids are extremely effective in this setting, and Sx of ↑ ICP will often abate. **Tests:** Consider ECG; others as indicated from H&P.
Neurological	Patients may present with complaints related to ↑ ICP (e.g., HA, N/V, visual changes, recent onset of Sz), neurological deficits from compression of motor area, or as a result of hemorrhage from the tumor or edema in surrounding normal brain. Document preop physical findings. **Tests:** A CT scan or MRI will delineate the site and size of the tumor, especially if iv contrast material, such as gadolinium, is administered to enhance the margins of the tumor. for intracranial mass effects: midline shift, ↓ ventricular volume, ↓ peri-brainstem (basal cisterns) CSF space, obstructive hydrocephalus.
Laboratory	Coags should be normal. Hct, electrolytes, and other tests as indicated from H&P.
Premedication	Standard premedication (except for patients with the possibility of ↑ ICP: no sedation).

Intraoperative

Anesthetic technique: Small tumors, particularly those located in deeper brain structures, may be localized and resected using stereotactic or image-guidance techniques. Generally, scalp markers (fiducials) or a stereotactic frame are placed on the patient's head; then the patient is taken to CT/MR for determination of the exact tumor site. GETA is almost invariably used for tumor removal, although MAC (see Awake Craniotomy, p. 34) is used on rare occasions when the surgeon needs to assess motor or sensory function during the resection of the tumor adjacent to critical motor or speech areas.

If the patient is in a stereotactic frame, or a difficult intubation is anticipated, orotracheal intubation will need to be accomplished before induction of GA. Awake fiber optic intubation (see p. B-5) is the best choice, because fitting a mask on the face with the stereotactic frame in place is impossible. After the airway is secured, anesthesia usually is induced with STP (2–5 mg/kg) or propofol (1–3 mg/kg) and fentanyl (3–5 mcg/kg) in combination with a NMR (e.g., vecuronium 0.1 mg/kg or rocuronium 0.6

Induction	mg/kg). To minimize ↑↑ BP and ↑ ICP with ET intubation, it is important that the patient be well anesthetized (and paralyzed) before undertaking laryngoscopy. Induction doses of STP, propofol, or midazolam may not be sufficient to abolish increases in MAP, CPP, and, hence, ↑ ICP is associated with laryngoscopy and tracheal intubation. Consider hyperventilating the patient and using remifentanil (2–3 mcg/kg) as part of the induction technique to further blunt the response to laryngoscopy in patients with ↑ ICP.
Maintenance	Isoflurane or sevoflurane ≤1 MAC (≤1/2 MAC if EP monitoring is used), inspired with 50:50 O_2/N_2O. Propofol (50–100 mcg/kg/min) can replace N_2O and may be used to further ↓cerebral blood volume, ↓ cerebral metabolism, and ↓ $CMRO_2$ Generally, if pancuronium is used, no additional NMBs are needed; however, if movement is of concern, rocuronium 10 mcg/kg/min will provide adequate neuromuscular blockade. A remifentanil infusion (0.05–0.2 mcg/kg/min) can be used to supplement the anesthetic without interfering with EP monitoring.
Emergence	Surgeons should request transient ↑ MAP to 90–100 mmHg to test hemostasis after the tumor has been resected. With the start of dural closure, consider changing the anesthetic to low-dose sevoflurane (e.g., 0.5%) with 50% N_2O (or propofol replacement), supplemented with a low-dose remifentanil infusion (e.g., 0.05 mcg/kg/min). The patient's BP generally will increase and titration of β-adrenergic blocking drugs (e.g., labetalol or esmolol) and/or vasodilators (e.g., SNP) may be needed. (See Control of BP, below.) Reverse NMB as necessary. The anesthetic agent can be D/C'd at the time of dressing application while continuing the remifentanil infusion. Most patients will breathe spontaneously and can be extubated uneventfully while on 0.05 mcg/kg/min remifentanil. If the brain has not been injured by the surgical procedure, the patient should awaken within 10 min after cessation of remifentanil administration. Keep close regulation of BP. Patient is placed in bed in a 20–30° head-up position and transported to ICU for monitoring overnight. Supplemental O_2should be administered and BP control maintained. Prophylactic antiemetics (e.g., metoclopramide 10–20 mg and ondansetron 4 mg) should be given iv 30 min before extubation.
Blood and fluid requirements	IV: 16–18 ga × 2 NS @ 2–3 mL/kg/h Brain tumors (e.g., meningioma) can be highly vascular. To minimize postop cerebral edema, limit NS to ≤ 10 mL/kg + replacement of UO. If volume is needed, administer albumin 5% as required.
Monitoring	Standard monitors (see p. B-1). Arterial line CVP line ± Precordial Doppler ± BAER, SSEP, MEP UO If the head of the OR table is significantly elevated (≤ 30°) or if the patient is in the seated position, a precordial Doppler monitor is necessary to monitor for VAE. Evoked potentials can be monitored in the presence of inhaled anesthetics (e.g. ≤ 0.6% isoflurane with 50% N_2O).
Positioning	Supratentorial: supine/lateral Posterior fossa: prone/sitting and pad pressure points. eyes. For brain tumors in the frontal, parietal, or temporal lobes, patient will be supine with head in Mayfield-Kees skeletal fixation, turned to the side and a roll under the shoulder on the operative side (Fig. 1.1-5). For occipital or posterior fossa tumors, patient may be prone or, occasionally, sitting (see Anesthetic Considerations for Cervical Neurosurgical Procedures, Positioning, p. 109). Acoustic neuromas are generally most easily removed with patient in the lateral ("park-bench") position with a roll under the axilla. The patient generally lies on a "bean bag" which holds her/him firmly in the lateral position.

		Patients with intracranial tumors may be on the steep portion of the intracranial compliance curve such that any increase in intracranial volume may →↑↑ ICP. Transient increases in ICP—even up to 50–60 mmHg—are tolerated, provided they are promptly terminated. Sustained increases in ICP > 25–30 mmHg are associated with severe neurologic injury and poor outcome.
Control of ICP	Maintain normovolemia Control BP in normal range for patient PaO_2 > 100 mmHg Limit isoflurane or sevoflurane to 1/2 MAC Steroids: 8–12 mg decadron iv	
	$PaCO_2$ = 25–30 mmHg	↓ $PaCO_2$→↓ CBV (providing better surgical access) + ↑ CBF to ischemic areas ("Robin Hood" effect) + ↓ anesthetic requirements.
	Replace N_2O with propofol infusion	If further reduction in ICP is needed consider replacing N_2O with a propofol infusion.
	Mannitol 0.5–1.0 g/kg ± Furosemide 10–20 mg	Mannitol/furosemide →↓ K^+; monitor level and replace as necessary. If mannitol is administered too rapidly, profound ↓ BP will occur, probably from peripheral vasodilation.
	± Lumbar CSF drain Head up 20–30°	CSF drain often placed after induction of anesthesia, and may be opened as required to ↓ CSF volume and pressure.

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Postoperative

		* Sz Rx: Phenytoin (1 g loading dose). **NB**: Incompatible with dextrose-containing solutions.
Complications	Seizures Neurologic deficits Tension pneumocephalus Hemorrhage requiring reexploration Edema and ↑ ICP	In seated position, additional rare, but possible complications include quadriplegia from excessive flexion of head or tension pneumocephalus from air in cerebral cavities. Severe tension pneumocephalus may delay emergence from anesthesia or cause postop neurologic deficits. CT scan may be necessary for Dx.
Pain management	Meperidine (10–20 mg iv prn) Codeine (30–60 mg im q 4 h)	Meperidine minimizes postop shivering. Avoid excessive sedation.
Tests	CT scan	If patient exhibits any delay in emergence from anesthesia and surgery, or any new neurologic deficits emerge postop, a CT scan is invariably obtained.

Suggested Readings

1. Apuzzo MLJ: *Brain Surgery: Complication, Avoidance and Management.* Churchill Livingstone, New York: 1993, 175–688.

2. Buckner JC, Brown PD, O'Neill BP, et al: Central nervous system tumors. *Mayo Clin Proc* 2007, 82(10): 1271–86.

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5. Eng C, Lam AM, Mayberg TS, et al: The influence of propofol with and without nitrous oxide on cerebral blood flow velocity and CO_2 reactivity in humans. *Anesthesiology* 1992; 77(5):872–9.

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8. Minton MD, Grosslight KR, Stirt JA, et al: Increases in intracranial pressure from succinylcholine: prevention by prior nondepolarizing blockade. *Anesthesiology* 1986; 65(2):165–9.

Anesthetic Considerations for Awake Craniotomy

Preoperative

These patients typically are otherwise healthy except for a tumor (or AVM) involving eloquent (speech) areas of the brain. In some centers, awake craniotomies are still used for epilepsy surgery. Awake craniotomies for functional procedures (e.g., deep brain electrode placement) usually involve 1 or 2 burr holes with small dural punctures, and are discussed on page 71.

In addition to the preoperative anesthetic considerations for brain tumors (see p. 31) or vascular malformations (see p. 19), it is essential that these patients (and family) meet with their anesthesiologist and monitoring neurologist to discuss in detail both the anesthetic and surgical aspects of the procedures, as well as the specific speech mapping techniques that will be used. The patient should be warned that a Foley catheter will be placed while they are sedated; *(Print pagebreak 35)* however after the sedation is stopped, the catheter will become annoying, and the patient will be convinced that he/she has a full bladder. Intraoperative cooperation is critical, and there is no pharmacologic substitute for a well-informed patient.

Premed	Midazolam 1–3 mg iv is usually tolerated. Beware of respiratory depression →↑ ICP. Antiemetic: e.g., ondansetron 4 mg iv slowly and q 2–3 h during the procedure

Intraoperative

Anesthetic technique: Although some centers use an asleep-awake-asleep technique with airway control (ETT or LMA) during the sleep phases, we prefer the following technique that minimizes airway manipulation and its inherent risk of coughing and bucking.

Premapping

Most patients with space-occupying lesions will benefit from the early administration of mannitol 0.5 gm/kg by slow iv infusion (check with surgeon for possible contraindications). With the patient in a beach-chair position and following the application of standard monitors and nasal prong O_2 initial sedation is provided by incremental midazolam titrated by effect (1–10 mg iv). A loading dose of dexmedetomidine (1 mcg/kg iv over 10–15 min as tolerated) is given while the sites for arterial line and central line (subclavian or antecubital long-line) placement are infiltrated with local anesthetic. Following the loading dose, a maintenance infusion of dexmedetomidine (0.2–0.7 mcg/kg/h) typically 0.4 mcg/kg/h is started. The dexmedetomidine produces cooperative sedation and analgesia without respiratory depression. A ↓ BP and ↓ HR are common and occasionally will require treatment with vagolytics and/or pressors. If necessary the sedation can be deepened with a propofol infusion (25–75 mcg/kg/min) to cover the more painful aspects of the procedure. A nasal airway may be useful in some patients. Scalp blocks (supraorbital, temporal, and occipital nerves) are placed using 0.5% bupivacaine with epinephrine.

Arterial line, cerebral line, and Foley catheter are placed. Pin sites for the Mayfield skull clamp are infiltrated with local anesthetic and the clamp is applied. The incision is infiltrated with local anesthetic and, after patient comfort has been assured, the craniotomy is allowed to proceed. Drapes should be arranged so that the anesthesiologist and neuromonitoring team have an unobstructed view of, and access to, the patient's face and airway.

Mapping

The dexmedetomidine (and propofol) infusions are stopped at the end of the craniotomy. Initial mapping (e.g., phase-reversal localization) can be accomplished while the patient is recovering from the sedation. Some direct cortical stimulation can produce seizures; it is important that sterile, iced saline be immediately available for cortical irrigation to suppress the seizure activity. If seizure activity spreads, it will be necessary to administer a bolus dose of STP (25–75 mg iv) through the central line. Airway support (e.g., LMA) may be necessary in the post-ictal phase. The patient should be awake and cooperative 20 min after the dexmedetomidine and propofol infusions have stopped. Occasionally, flumazenil may be necessary to remove residual midazolam effects. Detailed speech-mapping can then be readily accomplished. We use a miniature wireless microphone taped below the patient's mouth to amplify the patient's voice during testing and tumor resection.

Resection

If the lesion is located safely away from the mapped speech areas, the resection can be accomplished after reinstituting the dexmedetomidine and propofol infusions. Otherwise, the patient must be kept awake and talking throughout the resection. Often the patient will benefit from a low-dose remifentanil infusion (e.g., < 0.05 mcg/kg/min) to improve ability to tolerate positional discomfort.

Closing

Closing is best accomplished by resuming the previously effective infusions of dexmedetomidine and propofol. The infusions should be stopped during the final phase of wound closure. Careful control of BP may require the use of labetalol, esmolol, and SNP, which should be titrated during emergence. Typically, the patient will be awake and alert for transport to the ICU.

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Craniotomy for Skull Tumor

Surgical Considerations

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Steven D. Chang

Description: Tumors of the skull fall into the classification of other bony tumors. Examples of types of skull tumors often requiring surgery include eosinophilic granuloma, histiocytosis, hemangioma, osteoma, epidermoid, dermoid, metastases, osteosarcoma, fibrous dysplasia, chordoma, chondrosarcoma, and meningioma. These tumors may occur anywhere on the skull, except chordoma and chondrosarcoma, which usually arise from the skull base. The exact positioning of the patient depends on the location of tumor. For example, the sitting or prone position often is used for tumors in the occipital or suboccipital regions and the supine position is used for frontal, temporal, or parietal tumors. Some surgeons prefer the lateral position for temporal or parietal bone lesions and the prone position for occipital and some suboccipital bone lesions. Complete immobilization of the head is often not necessary, so the patient's head can be placed in a horseshoe or a Shea headrest. If complete immobilization is required, Mayfield pin fixation is used. The tumor is removed by placing burr holes near or around the tumor. Then the affected skull is removed by cutting a bone flap around the tumor with the craniotome. In a suboccipital or posterior fossa craniectomy, the bone often is removed piecemeal with either a drill or a series of rongeurs. The dura usually is not opened unless it is involved with the tumor. The surgeon may elect to perform a **cranioplasty** to cover the defect, depending upon the size and location. The defect can be repaired using a variety of methods, which include the use of a polymer, such as methylmethacrylate, or a bone cement, such as hydroxyapatite. Additionally, autologous bone may be harvested from another location on the skull or another site (hip or rib) for reconstruction. Lastly, the surgeon may elect to repair the cranial defect at a later date, using a custom implant made from titanium, methylmethacrylate, or polyethylethylketone (PEEK). After the tumor is removed and the reconstruction is completed, the skin incision is closed. Occasionally, the skull tumor will be approached intracranially, if its location favors that approach. Examples include tumors of the petrous portion of the temporal bone, fibrous dysplasia involving the optic canal, or skull base tumors, such as chordomas and chondrosarcomas.

Usual preop diagnosis: Eosinophilic granuloma; histiocytosis; hemangioma; osteoma; epidermoid; dermoid tumor; metastatic tumors; osteosarcoma; fibrous dysplasia; chordoma; chondrosarcoma; meningioma

Summary of Procedures

Position	Supine, lateral, prone, or sitting, based on the location of the tumor
Incision	Dependent on location of tumor
Special instrumentation	Neuro drill, craniotome, image-guided navigation (if intracranial)
Unique considerations	ETT must be taped securely in a location satisfactory to the surgeon. Anode or RAE tube may be helpful in certain situations.
Antibiotics	Cefazolin 1 g iv (if dura not opened)
Surgical time	1–4 h
Closing considerations	Drain often left in epidural space. Surgeon often requests control of BP to avoid hemorrhage into the bed of tumor.
EBL	25–500 mL
Postop care	ICU or close observation unit
Mortality	0–2% (higher for tumors in critical locations)
Morbidity	Usually < 5% Infection Neurological disability CSF leak Massive blood loss 2° venous sinus injury
Pain score	2–5

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Patient Population Characteristics

Age range	Infant–85 yr (usually 20–60 yr)
Male:Female	1:1
Incidence	Unknown
Etiology	Neoplastic

Anesthetic Considerations

See Anesthetic Considerations following Craniotomy for Tumor, p. 31. Note, however, that patients with skull tumors rarely have problems with ICP.

Suggested Readings

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2. Scolozzi P, Martinez A, Jaques B: Complex orbito-fronto-temporal reconstruction using computer-designed PEEK implant. *J Craniofac Surg* 2007; 18(1):224–8.

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Craniotomy for Trauma

Surgical Considerations

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Description: Head injuries occasionally require emergent surgical procedures to evacuate mass lesions or débride contused or contaminated brain. The majority of these injuries are supratentorial. The surgical procedure depends on the exact type and location of the injury (e.g., epidural, subdural, intraparenchymal hematomas, or depressed skull fracture); however, most traumatic injuries can be addressed through a wide frontotempoparietal craniotomy. Often the entire head is shaved and placed in a headrest (pins, suction cups, or horseshoe). If the C-spine has not been cleared, the patient may be placed in a lateral position to minimize neck involvement. Timing is often important for these procedures, because the patient usually has a component of ↑ ICP, either from a hematoma or from cerebral edema due to the trauma. Hyperventilation, mannitol, hypertonic saline, and diuresis may be necessary to control the patient's ICP until the patient can be taken into the operating room and undergo a wide decompressive hemicraniectomy (frontotempoparietal craniectomy). For a standard trauma craniotomy, the scalp incision starts anterior to the tragus and continues superiorly in a question-mark type path, ending in the frontal area (Fig. 1.1-11). This incision can be modified according to the location and extent of the injury. The skin is reflected and the skull is perforated with a cranial drill. The temporal burr hole should be placed first, because if the patient's condition deteriorates rapidly, the temporal burr hole can be enlarged quickly to a craniectomy for decompression before continuing with the craniotomy. A chronic subdural hematoma may be drained through burr holes. However, if there is an acute subdural hematoma, epidural hematoma, intraparenchymal hemorrhage, depressed fracture, or penetrating wound, a formal bone flap is elevated. Wide exposure is used to visualize and control sources of bleeding. Gentle warm irrigation may be used to aid in removal of an acute subdural hematoma. Mass lesions and associated contused brain parenchyma are identified and removed (Fig. 1.1-12). For penetrating head injuries or depressed skull fractures, the wound is débrided, foreign bodies are removed, bleeding is controlled, and the dura is repaired if lacerated. It is important to débride the

wound as much as possible, but it is often not possible to safely remove all fragments from the wound. Depending on the injury and the presence of brain swelling, it may be necessary to close the dura with autologous pericranium, or fascia lata. If these are not available, a synthetic dural substitute or bovine pericardium can be used. Alternatively, the dura may be left open, or multiple relaxing incisions may be made in the dura to allow for brain swelling. The surgeon can often repair and reconstruct a depressed skull fracture after removing a bone flap that surrounds the fracture. Additionally, the bone flap may be left out to compensate for brain swelling and replaced later. A subgaleal drain may be inserted and brought out through a separate incision. An ICP monitor or a ventriculostomy may be placed at the end of the procedure.

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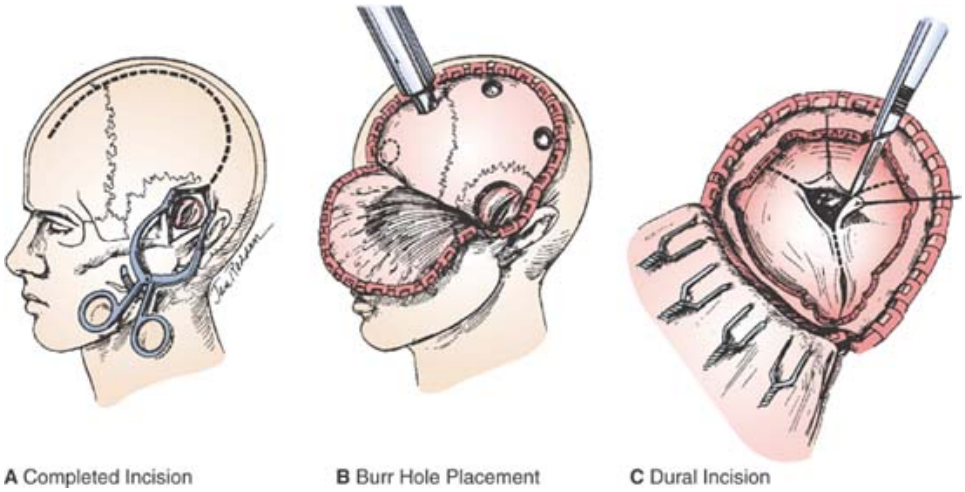

Figure 1.1-11. 11. In an acute subdural hematoma: **A:** burr hole is made, followed by craniectomy, and the incision is extended upward to form a large question mark, the medial extent of which follows the midline. **B:** Additional burr holes are made, with the medial ones 1.5 cm off the midline to avoid injury to the major venous structures and granulations. The anterior burr hole is placed above the frontal sinus (the size of which can be estimated from preop radiographs). **C:** The dura can be opened with a Y- or X-shaped incision, with a flap being based on the superior sagittal sinus. (Reproduced with permission from Grossman RG, Loftus CM: *Principles of Neurosurgery,* 2nd edition. Lippincott-Raven, Philadelphia: 1999.)

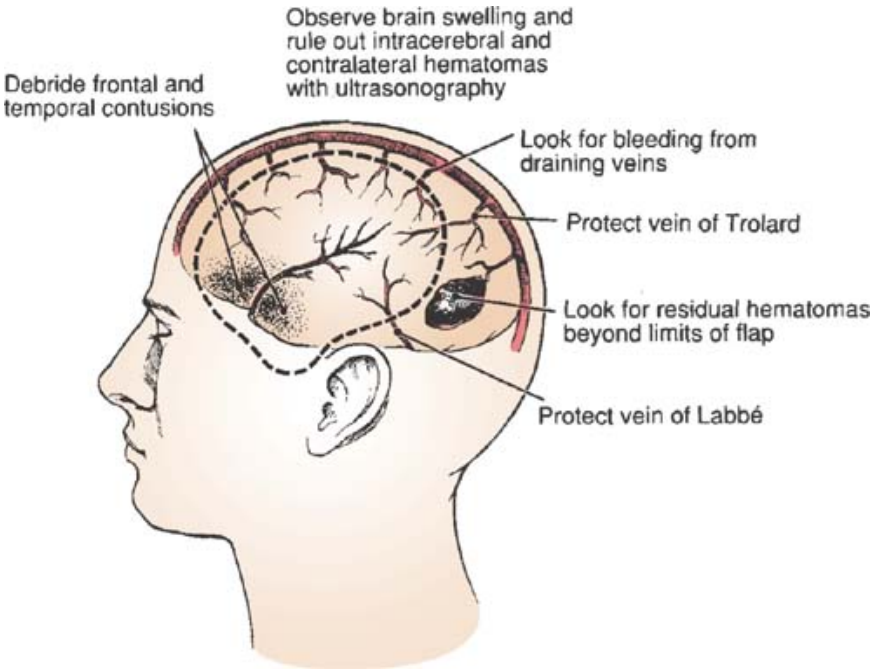

Figure 1.1-12. 12. Precautions to be taken during a craniotomy for subdural hematoma. (Reproduced with permission from Grossman RG, Loftus CM: *Principles of Neurosurgery,* 2nd edition. Lippincott-Raven, 1999.)

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Frontal sinus fracture: If a skull fracture involves the frontal sinus and/or there is a CSF leak, the sinus may need to be obliterated

or cranialized. Obliteration involves removal of the mucosa of the sinus and filling the sinus with antibiotic soaked Gelfoam sponges. Cranialization of the frontal sinus involves removing the posterior bony wall of the sinus and removing the mucosa. If there is a CSF leak, the dura will need to be repaired.

Variant indication: Some patients, after having a large ischemic stroke, will develop malignant cerebral edema, which may necessitate a decompressive hemicraniectomy (frontotempoparietal craniectomy) in order to control rising ICPs and prevent herniation.

Usual preop diagnosis: Epidural hematoma; acute subdural hematoma; intracerebral hematoma; depressed skull fracture; cerebral contusion; gunshot wound of the brain; malignant cerebral edema

Summary of Procedures

Position	Supine, lateral, prone, or sitting, depending on site of injury
Incision	Varies with location of injury (typical incision shown in Fig. 1.1-11)
Unique considerations	The patient may have ↑↑ ICP. Because incipient herniation and/or associated injuries may be a concern, timing is critical. C-spine precautions may be necessary. Patients may also be on anticoagulation medications, necessitating transfusions of blood products or recombinant activated factor VII.
Antibiotics	Ceftriaxone 1 g iv (avoid Ca^{++} containing solutions)
Surgical time	1.5–6 h
Closing considerations	Application of head dressing may jostle ETT at end of case →↑ BP. Patient may stay intubated postop. ICP monitor or external ventricular drain may be placed. Antiepileptic medications may be given for prophylaxis, if the patient had a seizure.
EBL	25–500 mL
Postop care	ICU or close observation unit until stable. Fluid and electrolytes require frequent monitoring, as the patient may develop SIADH. BP may need to be controlled with vasodilator and β-blocker infusions.
Mortality	10–50%, depending on lesion; higher for acute subdural hematomas, lower for epidural hematomas.
Morbidity	Morbidity depends on lesion; morbidity from craniotomy itself is low. Infection Neurologic disability Nerve injury CSF leak Endocrine disorders: SIADH Panhypopituitarism Diabetes insipidus (DI) Massive blood loss: venous sinus injury
Pain score	2–4

Patient Population Characteristics

Age range	Infant–85 yr (usually 15–40 yr)
Male:Female	2:1
Incidence	Relatively common
Etiology	Trauma

Associated conditions	Abdominal injuries; C-spine fractures

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Anesthetic Considerations

Preoperative

Traumatic brain injury (TBI) is the leading cause of death of persons < 24 years old. A penetrating injury of the skull usually will cause major damage to the brain as a result of both focal and diffuse neuronal injury with hemorrhage into brain tissue. Surgery is necessary to evacuate hematomas, control intracranial bleeding, to débride the wound, and to remove bone fragments, foreign material, and damaged brain so that the cranial vault can better accommodate the brain swelling that inevitably occurs. Epidural hematomas form between the skull and dura, and are usually due to bleeding from an artery (e.g., anterior cerebral or middle meningeal). Hence, time is of the essence and rapid evacuation and control of the bleeding is essential if permanent neurological injury is to be avoided. Subdural bleeding occurs between the dura and the leptomeninges lining the brain surface. This bleeding is usually venous in origin, and usually occurs more gradually. Focal intracranial hemorrhages may be either arterial or venous, and, as with subdural hematomas, must be evacuated if they are enlarging.

Respiratory	Ensure a secure airway and adequate ventilation. Localized injuries to the frontal or parietal lobes may not cause any respiratory changes. If ↑ ICP, respirations may become slow (< 10 min) and deep, and result in substantial hypocapnia. Many patients with head injuries demonstrate partial airway obstruction from the tongue falling back into the posterior pharyngeal space. If this occurs, or if the patient is comatose and unable to protect the airway and prevent aspiration of gastric contents, immediate tracheal intubation should be performed. Head injuries in the region of the occipital lobes may → hypoventilation or apnea. Neurogenic pulmonary edema may occur in up to 20% of TBI patients. **Tests:** As indicated from H&P and as time allows.
Cardiovascular	Most patients with head injuries evidence ↑ BP and ↑ HR. Relative hypotension (SBP < 90 mmHg) →↑ morbidity and mortality. If ICP increases sufficiently to cause herniation of brain stem, patients develop the “Cushing triad” of ↑ BP, ↓ HR and irregular respiration. These changes resolve when ↑ ICP is relieved, so vigorous attempts to regulate BP prior to craniotomy are not warranted. However, the patient should be taken to OR as quickly as possible. **Tests:** As indicated from H&P, and as time allows.
Neurological	Neurological evaluation of the head-injured patient is based on the Glasgow Coma Scale (Table 1.1-5). The scale involves evaluation of three functions: eye opening, verbal response, and motor response. Using this scoring system, the severity of brain injury may be classified as mild (13–15 points), moderate (9–12 points), or severe (8 points or less). By definition, any patient having 8 points or less is in coma. Additional useful neurological examinations include assessment of pupillary size and reactivity to the light, reflex responses, and evidence of asymmetry or flaccidity of the extremities or decerebrate (e.g., rigid arm extension) or decorticate (e.g., rigid arm flexion) posturing. Head-injured patients whose neurological function is deteriorating rapidly, and in whom an epidural or subdural hemorrhage is suspected, should be taken to OR immediately. Hyperventilation is appropriate if herniation is eminent. **Tests:** CT scan
	Severe head injury may be associated with a progressively

Hematologic	worsening coagulopathy, resulting in a clinical picture similar to that of DIC. The reason for this is not known, but the brain is rich in thromboplastin and other coagulation factors. **Tests:** Hct; PT; PTT; others as indicated from H&P.
Associated Injuries	Multiple organ system injury must be considered. Evidence of intrathoracic or intraperitoneal hemorrhage should be sought and repaired first if the patient is hemodynamically unstable. **Tests:** may include MRI, CT, ultrasound and others as indicated.
Laboratory	Other tests as indicated from H&P, and as time permits.
Premedication	Usually none

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Table 1. 1-5. Glasgow Coma Scale (GCS)

Category	Score
I. Eyes open:	
Never	1
To pain	2
To verbal stimuli	3
Spontaneously	4
II. Best verbal response:	
None	1
Incomprehensible sounds	2
Inappropriate words	3
Patient disoriented and converses	4
Patient oriented and converses	5
III. Best motor response:	
None	1
Extension (decerebrate rigidity)	2
Flexion abnormal (decorticate rigidity)	3
Flexion withdrawal	4
Patient localizes pain	5
Patient Obeys	6
	I+II+III Total = 3–15

Intraoperative

Anesthetic technique: GETA

Induction	↑ ICP is likely in most patients with head injury requiring operation, and induction of anesthesia is best accomplished with drugs that ↓ ICP. A mannitol infusion (0.25–1 mg/kg iv over 20 min) may be necessary to control ICP. If the patient is hemodynamically stable and not hypovolemic, titrated induction with STP (2–5 mg/kg) or propofol (1.5–3 mg/kg) + fentanyl (2–5 mcg/kg) is satisfactory. If hemodynamically unstable, etomidate (0.1–0.4 mg/kg) is suitable for induction. Ketamine is not used because of its ability to ↑ ICP. If the patient is comatose, anesthetic requirement is less, needing only $O_2$and muscle relaxant, or low-dose isoflurane (0.5%) or sevoflurane (< 1%). A nondepolarizing muscle relaxant (vecuronium [0.1 mg/kg] or rocuronium [1 mg/kg]) is administered for ET intubation. Succinylcholine may ↑ ICP transiently, but is still appropriate for emergency airway management. A defasciculating dose of a non-depolarizing NMB may attenuate the ICP ↑. Nasotracheal intubation is not recommended for patients with the possibility of maxillary and/or basilar skull fractures because of the potential for inserting the tube through the fracture site into the brain. To minimize ↑↑ BP and ↑↑ ICP with ET intubation, consider bolus remifentanil (2–5 mcg/kg iv) 1–2 min before laryngoscopy. In hypovolemic patients, hydration with a mixture of crystalloid (e.g., NS) and colloid (e.g., 5% albumin) should be initiated prior to induction.

Maintenance		The ideal drug for maintenance of anesthesia decreases ICP and $CMRO_2$ maintains cerebral autoregulation, redistributes flow to potentially ischemic areas, and provides protection for the brain from focal ischemia. Unfortunately, there is no ideal anesthetic. Both isoflurane and sevoflurane are good choices for patients undergoing neurosurgical procedures. Although these agents cause dose-dependent increases in CBF and volume, and hence ↑ ICP, these effects tend to be mitigated by the prior administration of STP (or propofol), hyperventilation, and by limiting the inspired concentrations to < 1 MAC. At these concentrations, CBF responses to changes in $PaCO_2$ are maintained, and cerebral autoregulation remains intact. N_2O is best avoided in patients with significantly elevated ICP. A propofol infusion is a reasonable alternative although its use has been associated with a ↓ CBF >↓ $CMRO_2$ →↑ potential for ischemia. Jugular bulb O_2 saturation may be reduced during propofol anesthesia, consistent with an unfavorable supply-demand balance. BP Goals: Keep SBP 90 mmHg and CPP 60–70 mmHg. Avoid prophylactic hyperventilation. Mild hypothermia may be beneficial if maintained > 48 h. DVT prophylaxis is recommended. High-dose steroid administration is associated with worsened outcomes. Muscle relaxation can be maintained as necessary.
Emergence		Because recovery from head injury is so unpredictable, it is generally advisable to leave the ETT in place and maintain controlled ventilation until there is sufficient clinical evidence that normal neurological recovery is occurring. Postop sedation can be accomplished using propofol (20–75 mcg/kg/min) not to exceed 5 mg/kg/h to minimize the occurrence of propofol-related infusion syndrome (including: metabolic acidosis, ↑ K ⁺, rhabdomyolysis).
Blood and fluid requirements	Possible marked blood loss IV: 14–16 ga × 1 NS @ 2–4 mL/kg/h ± Albumin 5%	Blood transfusion to keep Hct > 30. Minimize postop cerebral edema by limiting crystalloid volume to < 10 mL/kg + replacement of UO. Glucose-containing solutions should be avoided; blood glucose levels should be maintained between 80–180 mg%. If volume is needed, albumin 5% should be administered.
Control of blood loss	Keep CPP 60–70 mmHg Keep SBP > 90 mmHg Maintain normovolemia	Controlled hypotension generally is not used unless bleeding becomes profuse and difficult to control. ↓ BP is better treated initially with volume replacement than vasopressors.
Monitoring	Standard monitors (see p. B-1). ± Arterial line ± CVP line ± UO	If the injury is extensive or unknown, or if the patient is unstable, invasive monitoring is mandatory. Monitor for VAE if head is elevated above heart. Level the arterial line at the head. EEG, EP, and SjO_2 monitoring may be desirable.
Positioning	and pad pressure points. eyes.	For occipital or posterior fossa lesions, patient may be prone or sitting (see Anesthetic Considerations for Cervical Neurosurgical Procedures, Positioning, p. 107). Otherwise, patient will be supine with head in Mayfield-Kees skeletal fixation and turned to the side, and a roll placed under the shoulder on the operative side.
	Adequate anesthesia Head up 20–30° Keep SBP ≤ 90 mmHg STP or propofol infusion	Transient increases in ICP—even up to 50–60 mmHg—are generally tolerated, provided they are promptly terminated. Sustained increases in ICP > 25–30 mmHg are associated with severe neurologic injury and poor outcome. Patients with skull fractures or intracranial bleeding may be on the steep portion of intracranial compliance

		curve such that any increase in intracranial volume may cause ↑↑ ICP.
	Hyperventilation to $PaCO_2$= 25–30 mmHg PaO_2> 100 mmHg	↓ CO_2→ cerebral vasoconstriction →↓ CBV, ↓ CBF, and ↓ ICP. Patients with diffuse brain injury may have lost cerebrovascular sensitivity to $PaCO_2$ Despite maintaining adequate ventilation, oxygenation and BP, patients with diffuse head injury often exhibit arterial and CSF lactic acidosis, a further indication of the metabolic derangement that exists in the brain from the injury.
Control of ICP		
	Keep CPP 60–70 mmHg	Control MAP and cerebral venous pressure so that CPP = 60–70 mmHg. In severe, diffuse head injury with loss of autoregulation, some parts of the brain may exhibit 'luxury perfusion', while other areas exhibit severe ischemia.
	Mannitol 0.25–1.0 g/kg	Vigorous diuresis will commence in about 30 min (if blood volume is adequate) and brain shrinkage will follow. It is often necessary to provide supplemental potassium (20–30 mEq iv slowly).
	Furosemide 10–20 mg	Simultaneous administration of furosemide (10–20 mg) is recommended to avoid the transient ↑ CBV and ↑ ICP following mannitol. If mannitol is administered too rapidly, profound hypotension will occur, probably from peripheral vasodilatation.

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Postoperative

Complications	Seizures	*Sz Rx: phenytoin (1 g loading dose). **NB:** Incompatible with dextrose-containing solutions.
	Neurologic deficits Hemorrhage SIADH/DI	Some patients with severe head injury remain unconscious for weeks or months, without evidencing any substantial neurological recovery. A late complication of head injury is hydrocephalus requiring a shunt procedure.
	NPE ↑ICP DIC	Neurogenic pulmonary edema may result from massive sympathetic discharge 2° ↑↑ ICP. Rx: ↓ ICP, alpha-blockers to control BP, respiratory support. TBI is a risk factor for DIC.
Pain management	Fentanyl/propofol infusion	Most patients will remain intubated and sedated postop.
Tests	CT scan ICP monitor	Unless neurological recovery is rapid, periodic CT scans are obtained postop to follow the intracranial changes. In addition, in many institutions, a device for monitoring ICP postop is placed at the time of operation.

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(Print pagebreak 44)

Microvascular Decompression of Cranial Nerve

Surgical Considerations

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Steven D. Chang

Description: Microvascular decompression is used to treat various disorders of the cranial nerves, including trigeminal neuralgia, hemifacial spasm and, more rarely, glossopharyngeal neuralgia. Trigeminal neuralgia is characterized by brief episodes of intense, stabbing facial pain along the distribution of the trigeminal nerve. This pain usually can be elicited by gentle stimulation of the affected area. Hemifacial spasm is characterized by paroxysmal repetitive twitching of the facial muscles. The twitching usually starts with the muscles around the eye and can progress to involve the rest of the facial muscles. Glossopharyngeal neuralgia is characterized by paroxysmal pain that involves the ear and throat. Typically, the pain is described as 'stabbing' and radiates from one site to the other. Swallowing, drinking cold beverages, talking, or coughing can elicit the pain.

All of these conditions are usually unilateral and are often caused by compression of a cranial nerve by a vascular structure. In trigeminal neuralgia, the superior cerebellar artery is the usual culprit. In hemifacial spasm, usually the anterior inferior cerebellar or vertebral artery is compressing the facial nerve. In glossopharyngeal neuralgia, the posterior inferior cerebellar or vertebral artery is usually the offending artery. Large veins can also compress the cranial nerves. The goal of a microvascular decompression is to remove the pressure on the cranial nerve. To perform a microvascular decompression, a linear incision is made behind the ear on the affected side (Fig. 1.1-13A). Dissection is taken down to the skull, and several burr holes are made. The burr holes can be enlarged into a **craniectomy** (Fig. 1.1-13B) or used for elevation of a bone flap. The craniectomy is placed below the transverse sinus and medial to the sigmoid sinus to allow access to the cerebellopontine angle. Any venous sinus bleeding is controlled and the dura is opened (Fig. 1.1-13C). With brain relaxation, the cerebellum is retracted. The operating microscope allows the surgeon to explore the involved cranial nerve. If an offending vessel is identified, it is carefully dissected away from the nerve and shredded Teflon felt or a small, plastic sponge is placed to keep the vessel away from the cranial nerve. In the case of trigeminal or glossopharyngeal neuralgia, if no offending vessel is identified, a **partial section of the nerve** may be performed. Partial section of the 9th or 10th cranial nerve may cause some vasomotor instability. BAERs, facial nerve monitoring, and EMGs can be measured intraop to protect the cranial nerves. Once the cranial nerve has been decompressed, the dura is closed, and the wound is closed in layers. The main variations in this procedure are in patient positioning and surgeon's preference of monitoring modalities. Patient positioning may be lateral, prone, supine, or sitting. Intraop, mannitol (0.5–1 g/kg) and a lumbar drain (for CSF removal) may be needed for brain relaxation. Success rate is typically 75–95%, with a recurrence rate as high as 3.5% per year.

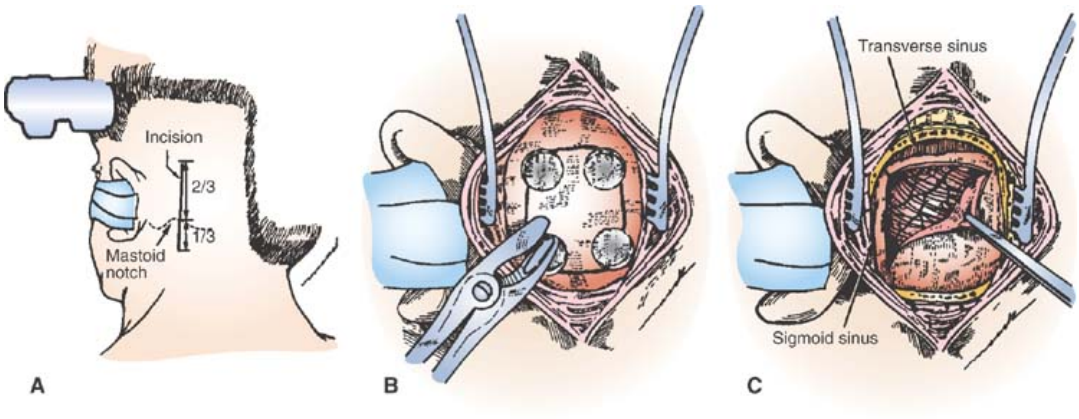

Figure 1.1-13. 13. Microvascular decompression: **A.** Location of incision. **B.** Enlargement of burr holes into craniectomy with a rongeur. **C.** Dural opening on the left side. Note position of venous sinuses. (Reproduced with permission from Wilson CB: *Neurosurgical Procedures: Personal Approaches to Classic Operations.* Williams & Wilkins, Philadelphia: 1992.)

(Print pagebreak 45)

Alternative Procedure: Gamma Knife surgery is less effective than microvascular decompression, but may be appropriate for patients unwilling or unable to undergo surgery. Other options include radiofrequency rhizotomy (see p. 86), glycerol injection, and balloon compression.

Usual preop diagnosis: Trigeminal neuralgia; tic douloureux; hemifacial spasm; tinnitus; glossopharyngeal neuralgia

Summary of Procedures

Position	Normally, lateral ("park-bench"), head elevated 30°; upper shoulder retracted caudally
Incision	Retroauricular (mastoid) (See Fig. 1.1-13A.)

Special instrumentation	Operating microscope; cranial perforator; ± facial nerve monitoring; ± EMG; ± BAER
Unique considerations	Risk of air embolus in head-elevated positions
Antibiotics	Ceftriaxone 1–2 gm iv q d (avoid Ca^{++}-containing solutions)
Surgical time	2–3 h
EBL	25–250 mL
Postop care	ICU or close observation unit. Observe for change in neurologic status (e.g., level of alertness, response to commands), usually for 12–24 h.
Mortality	0.2–2%
Morbidity	Facial sensory deficit: 25% (often transient) Aseptic meningitis: ≤ 20%, usually occurring 3–7 d postop Hearing loss: 3% Diplopia (usually transient) Facial weakness: 2% Deafness: 1% Infection CSF leak Massive blood loss due to vertebral artery injury or sinus laceration
Pain score	4–6

Patient Population Characteristics

Age range	40–85 yr (usually 60–70 yr)
Male:Female	2:3
Incidence	4/1,000,000 (trigeminal neuralgia); 0.06/100,000 (glossopharyngeal neuralgia)
Etiology	Vascular compression of cranial nerve; multiple sclerosis plaque
Associated conditions	HTN; multiple sclerosis

Anesthetic Considerations

Preoperative

Microvascular decompression involves a full craniotomy for decompression of a cranial nerve that is causing facial pain and/or spasm of facial muscles. Generally, these patients have trigeminal neuralgia or tic douloureux that has not been responsive to medical management (e.g., carbamazepine [Tegretol] therapy) and percutaneous rhizotomy or glycerol injection has failed.

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Respiratory	None unless the patient has a long-standing Hx of smoking and has COPD.
Cardiovascular	Many patients will have Hx of idiopathic HTN requiring antihypertensive medications. Good preop control of BP is important because it will make intraop and postop management of BP easier. Anticipate exaggerated hemodynamic response to anesthetic medications.
Neurological	The presenting symptom is pain ± muscle spasm in the maxillary and/or mandibular division of the trigeminal nerve, unaccompanied by any motor or sensory deficits. Identify trigger

	points so that they may be avoided during mask placement and induction.
Laboratory	None, except for routine preop studies.
Premedication	Generally, patients for these procedures are elderly and do not require any special premedication. Midazolam 1–3 mg iv will provide amnesia for the preop events, if that is desired by patient or surgeons.

Intraoperative

Anesthetic technique: GA is necessary because a full craniotomy is performed. ICP is not increased in these patients. Brain volume reduction, however, is important to provide the surgeon with sufficient space to identify and relieve the pressure on the offending nerve without requiring excessive brain retraction in the process.

Induction	Induction is best accomplished with drugs that cause brain volume reduction, including STP (2–5 mg/kg) or propofol (1–2 mg/kg), followed by neuromuscular blockade and ET intubation.	
Maintenance	Standard maintenance (p. B-2) is usually satisfactory. Desflurane has been associated with ↑ PONV. BP is maintained in the normal range during the operation. Hyperventilation to achieve a $PaCO_2$ of 25–30 mmHg is helpful to ↓ brain volume and ↑ workspace. Once the nerve has been isolated, stop hyperventilation. A spinal drain may be used to remove CSF during the operation and improve exposure. The drain usually is opened at the time of dural opening and closed as soon as surgery on the nerve is complete.	
Emergence	The ETT is removed at the conclusion of operation. Postop HTN may need to be controlled with esmolol and/or SNP by continuous pump infusion. Prophylactic antiemetic (e.g., metoclopramide 10 mg and ondansetron 4 mg) should be given 30 min before extubation. The incidence of PONV is high following this procedure.	
Blood and fluid requirements	Large blood loss is rare IV: 18 ga × 1 NS @ 2–4 mL/kg/h	Mannitol 0.5–1 mg/kg is sometimes necessary to provide sufficient brain volume reduction to permit adequate surgical exposure.
Monitoring	Standard monitors (see p. B-1). Arterial line CVP line ± EMG and/or SSEP	Invasive monitoring will facilitate intraop and postop BP management. Sometimes EMG and/or SSEP monitoring of the facial nerve is performed.
Positioning	Lateral, "park bench" Pillow between legs and pad pressure points. eyes.	Patients usually will be positioned laterally in the "park-bench" position. Padding of the axillae and elbows and placing a pillow between the legs are necessary. A bean bag is often used to hold patient stable in the lateral position.

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Postoperative

Complications	PONV Bleeding Brain edema	PONV is common and usually requires multimodal treatment (see p. C-1). Major complications from this operation are uncommon, and postop recovery is usually uneventful. On rare occasion, significant brain edema or bleeding may be experienced.
Pain management	Codeine (30–60 mg im q 4 h)	

Tests	CT scan, if neurological recovery is delayed.

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Bifrontal Craniotomy for CSF Leak

Surgical Considerations

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Description: Trauma (44% accidental; 29% surgical) is the leading cause of CSF leaks. CSF leaks can also be caused by tumors (22%), congenital malformations, or as a result of transsphenoidal surgery. Most CSF leaks involve the floor of the anterior cranial fossa (e.g., cribriform plate: 35%), with drainage through the auditory canal (otorrhea), nasopharynx (rhinorrhea), sinus air cells, or other less common routes. CSF leaks can occasionally stop on their own or by management with a lumbar drain. However, to lessen the risk of meningitis, surgical repair is the usual treatment.

The surgical repair of CSF leaks has changed with the advent of the endoscope. The success rates of endoscopic closure are similar to open surgical approaches while avoiding the morbidity associated with craniotomies. Although it is common to attempt closure of a CSF leak endoscopically before proceeding on to an open repair, selected patients may benefit by proceeding directly to an open surgical approach.

An open surgical repair is typically done through a bifrontal craniotomy. The patient is placed in a supine position, and the head is stabilized with a Mayfield clamp. A bicoronal skin incision (Fig. 1.1-14) is made, and the pericranium is often turned as a separate flap and kept moist during the case. Next, burr holes are drilled and a bifrontal free-bone flap is elevated. Care is taken to avoid injury to the underlying superior sagittal sinus. If the frontal sinuses are entered, the mucosa is stripped and the sinus is packed with antibiotic-soaked *(Print pagebreak 48)* Gelfoam. The dura is opened over the inferiomedial frontal lobes and the sagittal sinus is ligated. An intradural exploration is undertaken to determine the site of the leak. Identification of the leak site is essential for successful outcome. Brain relaxation is usually necessary to reduce the need for mechanical retraction, and may require mannitol, hyperventilation, and/or the placement of a lumbar drain. The frontal lobes are elevated, and the olfactory tracts may be sacrificed. If the site of the leak can be determined, the dural repair can be performed using fascia lata, pericranium, bovine pericardium, or a

synthetic dural substitute. It usually is necessary to strip the dura off the anterior cranial fossa to complete the repair. Defects in the bone can be plugged with a variety of materials (e.g., pericranial flap, fat, muscle, bone, or wax). After the repair is complete, the dura can be closed and the bone flap replaced, often with a drain left in the epidural space. Additionally, tissue sealants such as fibrin glue can be sprayed over the dural suture line to help create a watertight seal. At this point, brain relaxation is no longer required. The wound is closed with stitches and skin is closed with staples.

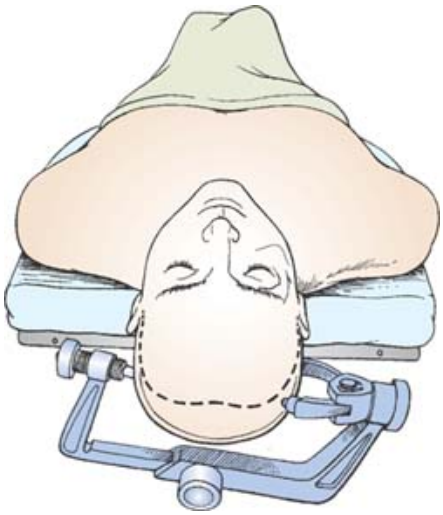

Figure 1.1-14. 14. Patient positioning for bifrontal craniotomy with bicoronal skin incision outlined. This position allows the frontal lobes to separate from the anterior cranial fossa with minimal retraction on the frontal lobes. (Reproduced with permission from Tindall GT, Cooper PR, Barrow DL: *The Practice of Neurosurgery.* Williams & Wilkins, Philadelphia: 1996.)

A combined approach may be used for CSF leaks 2° to a tumor that invades the dura and the cribriform plate. In these cases, the procedure often is performed with an otorhinolaryngologist. The intracranial part of this procedure is identical to the bifrontal craniotomy, except that bone and tumor are removed at the floor of the anterior cranial fossa. The extracranial part of this procedure is performed by the otorhinolaryngologist, who approaches the sinonasal region endoscopically or through a lateral rhinotomy or degloving gingival incision. The extracranial approach is chosen to complement the cranial exposure and a common space is created between the two operative fields. Closure involves isolating the two operative fields once again. The dural repair is as above. The mucosa of the nasal cavity is replaced with the use of a skin graft.

Alternate Procedure: Transnasal endoscopic repair of selected CSF leaks has been shown to be effective with significantly ↓ morbidity compared with craniotomy.

Usual preop diagnosis: CSF leak or rhinorrhea; fracture of the anterior cranial fossa; intracranial encephalocele; cribriform plate tumor; olfactory neuroblastoma (tumor of the olfactory epithelium). β-2 transferrin is a relatively sensitive and specific marker for CSF and can be used to establish the diagnosis.

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Summary of Procedures

Position	Supine
Incision	Bicoronal (Fig. 1.1-14); Transnasal for endoscopic
Special instrumentation	Operating microscope; endoscope; image-guidance system
Unique considerations	Brain relaxation desired; lumbar subarachnoid catheter; fascia lata graft
Antibiotics	Ceftriaxone 1–2 g iv q d (avoid Ca^{++} containing solutions)
Surgical time	2–3.5 h, depending on extent of leak or lesion
Closing considerations	Drain often left in epidural space. Anticipate that the application of a full head dressing will jostle patient and ETT →↑ BP + coughing/bucking.
EBL	75–500 mL

Postop care	ICU or close observation unit; Valsalva maneuver, coughing and vomiting can cause ↑↑↑ ICP → recurring CSF leak
Mortality	≤ 5%
Morbidity	Usually < 5% for all complications Infection (ascending meningitis) Recurrent CSF leak Neurologic disability Nerve injury Massive blood loss: venous sinus injury
Pain score	3–5

Patient Population Characteristics

Age range	15–65 yr
Male:Female	3:2
Incidence	Relatively uncommon neurosurgical procedure
Etiology	Traumatic (73%); congenital; neoplastic

Anesthetic Considerations

See Anesthetic Considerations following Transsphenoidal Resection of Pituitary Tumor, p. 54.

NB: Positive pressure mask ventilation in patient with an anterior CSF leak may produce severe pneumocephalus.

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(Print pagebreak 50)

Transoral Approach to the Cervicomedullary Junction and Odontoid

Surgical Considerations

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Steven D. Chang

Description: The transoral approach provides excellent access to the odontoid process of the C2 vertebral body, as well as the skull base just anterior to the brain stem (Fig. 1.1-15). This is important for conditions where there is pressure on the ventral brain stem or spinal cord → Sx: HA, impaired ambulation, hyperreflexia, paresthesias, neurogenic bladder, etc. Common indications for this approach include basilar impression (upward translocation of the odontoid process) (Fig. 1.1-16A); degeneration of the odontoid due to rheumatoid disease (Fig. 1.1-17A); fracture of *(Print pagebreak 51)* the odontoid, and resection of extradural tumors, such as chordomas and metastases. In these cases, the operation is performed through the oral cavity with an incision in the posterior wall of the pharynx. Special retractors hold the mouth open and keep the tongue out of the way. Fluoroscopic or image guidance helps the surgeon maintain proper trajectory. Intraop monitoring of evoked potentials (i.e., SSEP or BAER) and use of the operative microscope help avoid injury to the brain stem and spinal cord. The dissection is carried down to expose the anterior arch of C1 and body of C2. To decompress the region, the anterior arch of C1 and the odontoid process are removed. For basilar impression, a portion of the clivus may need to be removed to gain access to the dens (Fig. 1.1-16B). For rheumatoid arthritis, the pannus (thickened fibrous tissue) surrounding the dens is also removed (Fig. 1.1-17B). If an extradural tumor is found, it is resected in a piecemeal fashion. If a CSF leak occurs, the dura can be closed with fascia, muscle, fibrin glue, or thrombin-soaked Gelfoam. Upon completion of the decompression, the mucosa is closed. It may be necessary to fuse the occiput to the upper C-spine to correct any cervical instability. This may take place at the same time, or at a later date. C-spine precautions normally are used during and after the case. The patient may be in traction with tongs or a halter.

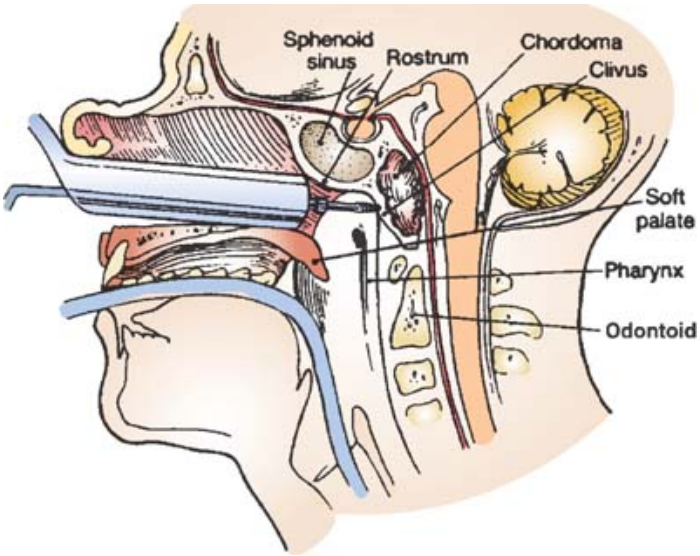

Figure 1.1-15. 15. Inferior exposure of the clivus and chordoma. (Reproduced with permission from Grossman RG, Loftus CM: *Principles of Neurosurgery,* 2nd edition. Lippincott-Raven, Philadelphia: 1999.)

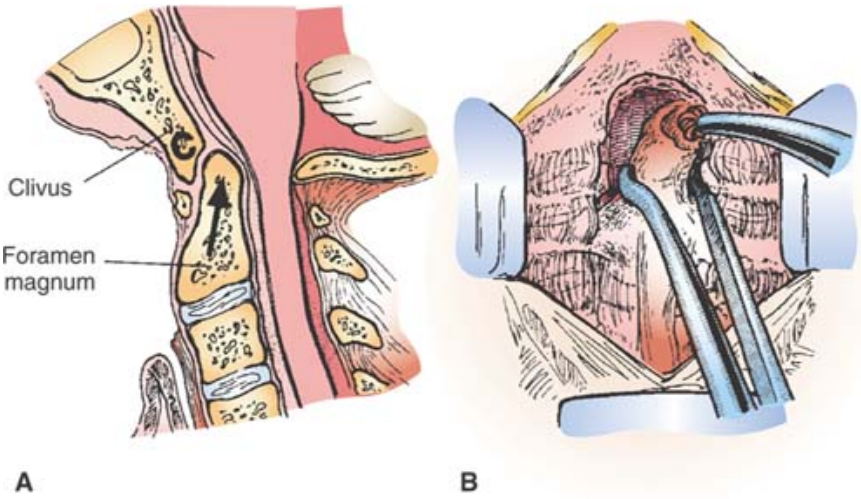

Figure 1.1-16. 16. A. Basilar impression. The dens has moved superiorly into the foramen magnum (*arrow*). **B.** Removal of the dens with a drill. The arch of C1 and a portion of the clivus have been removed. (Reproduced with permission from Donald PJ: *Surgery of the Skull Base.* Lippincott-Raven, Philadelphia: 1998.)

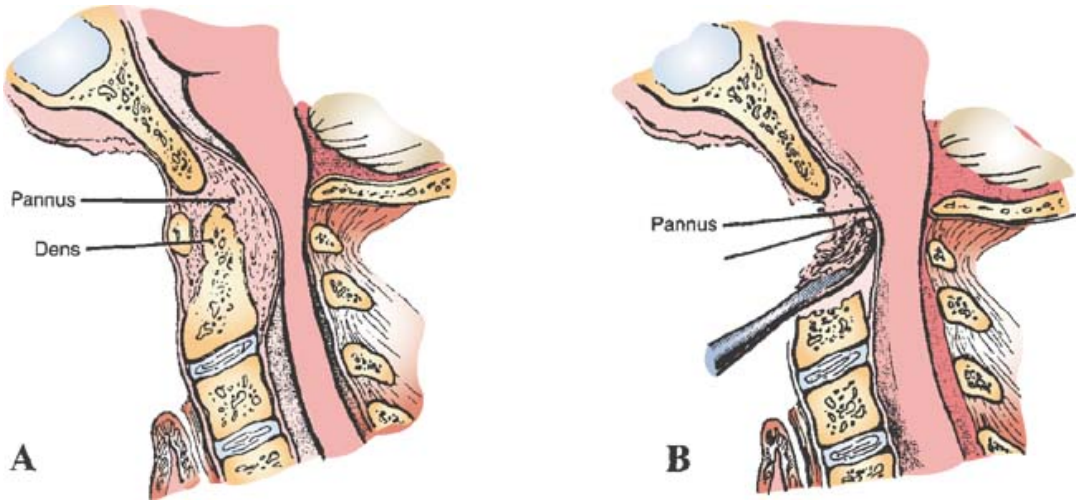

Figure 1.1-17. 17. A. Pannus formation in a patient with rheumatoid arthritis. **B.** Removal of pannus. (Reproduced with permission from Donald PJ: *Surgery of the Skull Base.* Lippincott-Raven, Philadelphia: 1998.)

Variations in procedure: A fiber optic nasal intubation or tracheostomy may be necessary to gain adequate exposure and maintain ventilation. It also may be necessary to split the soft palate, hard palate, tongue, and/or mandible to obtain adequate exposure.

Usual preop diagnosis: Basilar impression (platybasia); odontoid fracture; rheumatoid arthritis with atlantoaxial instability and anterior impingement of the cord; chordoma; metastatic tumor

Summary of Procedures

Position	Supine; head in traction or pins
Incision	Back of oropharynx
Special instrumentation	Operating microscope; I.I.; intraop monitoring = SSEP or BAER; micro drill; intraoral retractors for exposure; image-guided navigation system (optional)
Unique considerations	FOL may be necessary. ETT must be taped securely in a location satisfactory to surgeon. Anode or RAE tube may be helpful. Occasionally, a **tracheostomy** is performed in advance.
Antibiotics	Ceftriaxone 1g iv (avoid Ca^{+}containing solutions)
Surgical time	2.5–3 h
Closing considerations	Patient may remain intubated postop. C-spine precautions may be necessary.
EBL	25–250 mL
Postop care	ICU or close observation unit; monitor airway for swelling (for stridor).
Mortality	0–3%
Morbidity	All < 5%: Infection CSF leak Neurological Massive blood loss
Pain score	2–4

(Print pagebreak 52)

Patient Population Characteristics

Age range	18–85 yr (usually 20–60 yr)
Male:Female	1:2
Incidence	Rare
Etiology	Neoplastic; traumatic; congenital; degenerative
Associated conditions	Rheumatoid arthritis (atlantoaxial subluxation in 25%; basilar compression in 8%); traumatic injury

Anesthetic Considerations

See Anesthetic Considerations following Transsphenoidal Resection of Pituitary Tumor, p. 54.

Suggested Readings

1. Hadley MN, Spetzler RF, Sonntag VKH: The transoral approach to the superior cervical spine. *J Neurosurg* 1989; 71(1):16–23.

2. Vangilder JC, Menezes AH: Craniovertebral abnormalities and their neurosurgical management. In *Operative Neurosurgical Techniques: Indications, Methods, and Results.* Schmidek HA, Sweet WH, eds. WB Saunders, Philadelphia: 2000, 1934–45.

Transsphenoidal Resection of Pituitary Tumor

Surgical Considerations

Edward R. Laws
Gordon T. Sakamoto
Lawrence M. Shuer
Steven D. Chang

Description: The transsphenoidal approach to the sella turcica is a direct procedure used to gain access to the pituitary gland and sella region and is associated with relatively fewer complications than a craniotomy. The procedure usually is performed transnasally; although a sublabial transeptal approach is out of favor it may be used in patients with large tumors. An otorhinolaryngologist may participate in obtaining the exposure, which typically involves creating a tunnel to the sphenoid sinus. Once the sphenoid sinus is reached, it is entered by removing the face and rostrum. The thin bone of the lateral wall of the sphenoid sinus protects portions of the optic nerve and carotid artery. The sella is entered by removing a portion of the sellar floor (Fig. 1.1-18A). Under image guidance and with the aid of the operating microscope and/or endoscope, the surgeon can operate safely upon the pituitary gland, respecting the midline and avoiding injury to the carotid arteries in the cavernous sinus on either side of the exposure. After the sellar floor is removed, the dura is opened and the tumor is removed with a series of microdissectors and curettes. Following tumor removal, if there is a CSF leak, the surgeon may harvest fat, muscle, or fascia from the thigh or abdomen to pack into the sella to reduce free intrasellar space and serve as a graft to prevent a leak (Fig. 1.1-18B). Resorbable materials, such as Gelfoam and fibrin glue, also may be used to obliterate dead space within the sella. The *(Print pagebreak 53)* floor of the sella can be reconstructed with bone salvaged from the exposure or artificial material. During the closure, the sphenoid sinus also may be packed and sealed with the same materials used to close the sella. The nostrils may or may not be packed, depending on the details of the technique used.

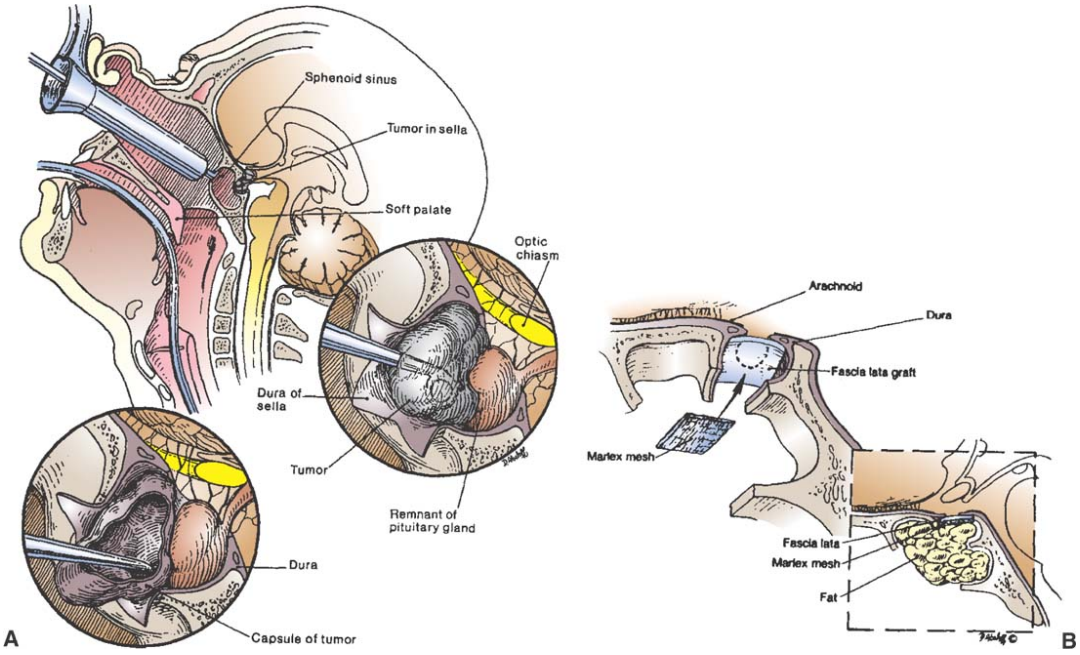

Figure 1.1-18. 18. The transnasal/transsphenoidal removal of a pituitary tumor. **A.** The rostrum of the sphenoid is removed (*upper*). The floor of the sella is removed, the dura opened, and the tumor removed using microcurettes (*middle and lower*). **B.** Fat taken from the subcutaneous tissue at the time of the fascial resection is utilized to fill the sphenoid and hold the graft material in position. (Reproduced with permission from Grossman RG, Loftus CM: *Principles of Neurosurgery,* 2nd edition. Lippincott-Raven, Philadelphia: 1999.)

Alternative Procedure: Endoscopic transsphenoidal approach has several advantages (improved surgical view and access, fewer complications and more rapid recovery).

Usual preop diagnoses: Pituitary tumor; prolactin-secreting tumor; growth hormone-secreting tumor (acromegaly); ACTH-secreting tumor (Cushing's disease); visual compromise, intrasellar tumor; craniopharyngioma; meningioma; Forbes-Albright syndrome (galactorrhea-amenorrhea syndrome)

Summary of Procedures

Position	Supine, head elevated 20–30°
Incision	Endonasal; + abdomen (or thigh) for fat graft; occasionally sublabial, maxillary gingiva;
Special instrumentation	Operating microscope; operating endscope; micro drill; neuronavigation (image guidance)
Unique considerations	Throat packs may be used but are usually unnecessary. ETT must be taped securely in a location away from the surgeon (usually left side of mouth). Anode or RAE tube may be helpful but is not essential. The generous use of topical vasoconstrictors and the injection of epinephrine-containing solutions may →↑ BP and dysrhythmias. Dissection in the nasal cavity can be painful →↑ BP and ↑ ICP. There is a risk of VAE following inadvertent entry into the cavernous sinus. Valsalva maneuver, jugular compression, or injection of saline through a lumbar intrathecal catheter may be requested to facilitate tumor exposure. Injuries to sphenopalatine or carotid arteries are rare but may be dramatic when they occur.
Antibiotics	Ceftriaxone 1 g iv (avoid Ca^{++} containing solutions)
Surgical time	2.5–3 h
Closing considerations	Possible abdominal or thigh-fat graft. Closure quite fast. Avoid straining and bucking if there has been an intraoperative CSF leak. Remove throat pack before operation.

EBL	25–250 mL
Postop care	Close observation of fluid and electrolytes require frequent monitoring as the patient may develop diabetes insipidus (DI), usually transient, following this procedure.
Mortality	1%
Morbidity	Anterior pituitary insufficiency: 19.4% DI: 0.5–31%; SIADH: 9–25% Sinusitis: 8.5% Septum perforation: 3% CSF leak: 3.5% Epistaxis: 3.0% Loss of vision: 1.8% Meningitis: 1.5% Carotid artery injury: 1.1% stroke (rare) **Prolactin Secreting:** Few anesthetic implications beyond possible mass effect and ↑ ICP **ACTH Secreting:** HTN is very common and is often associated with diastolic dysfunction. OSA is common and may have implications for postop airway management. Hyperglycemia is common and should be treated if blood glucose > 180 mg/dL. ↑ cortisol → fragile skin and veins → difficult iv access. **Growth Hormone Secreting:** Hypertrophy of the mandible, tongue and other facial tissues is common and complicates airway management. Hoarseness suggests laryngeal stenosis or recurrent laryngeal nerve dysfunction. OSA is very common with its attendant risk of postop airway compromise. HTN and diastolic dysfunction (with a need for ↑ filling pressure) are very common. The incidence of intraop ventricular ectopy and SVT is higher in these patients. ECG changes (e.g., ST/T wave changes, conduction abnormality) are common. **Pituitary Apoplexy:** Acute hemorrhagic infarction of the pituitary → severe HA, shock, coma, death. Urgent surgical decompression required.
Pain score	2–4

(Print pagebreak 54)

Patient Population Characteristics

Age range	18–85 yr (usually 30–50 yr)
Male:Female	2:3 (microadenomas)
Incidence	16.7% (autopsy studies); 1.5/100,000/yr diagnosed
Etiology	Neoplastic; traumatic
Associated conditions	Cushing's disease (ACTH); acromegaly (GH); amenorrhea/galactorrhea (prolactin); hyperthyroidism (TSH); hypergonadotropism (FSH; LH); multiple endocrine neoplasia I

Anesthetic Considerations

(Procedures covered: bifrontal craniotomy for CSF leak; transoral approach to cervicomedullary junction, odontoid; transsphenoidal resection of pituitary tumor)

(Print pagebreak 55)

Preoperative

Endocrine	Tumors of the pituitary gland are either nonfunctional or secretory. If nonfunctional, they will produce Sx either by their mass effect on adjacent pituitary tissue, or because of extension outside of the sella turcica. Rarely, the mass effect may cause the clinical picture of panhypopituitarism requiring preop treatment with thyroxine, glucocorticoid, and vasopressin. Functional tumors commonly secrete prolactin (→ lactation), ACTH (→ adrenal hyperplasia), or growth hormone (→ acromegaly). Hyperprolactinemia may occur with any tumor 2° loss of inhibition of prolactin secretion. **Tests:** Preop endocrine studies, including serum and urinary levels of pituitary, thyroid, and adrenal hormones; appropriate replacement therapy established before proceeding with surgery. "Stress-dose" hydrocortisone (e.g., 100 mg iv q.8.h) is often unnecessary; treatment should be guided by symptoms and periop cortisol levels. Dexamethasone will not interfere with cortisol assays.
Respiratory	No special requirements, unless patient has acromegaly, in which case large facial features, long neck, large tongue, and redundant soft tissue in the oropharynx may make mask fit and ET intubation difficult. If these patients evidence hoarseness or inspiratory stridor, they should have a full clinical and radiological evaluation of the upper airway. **Tests:** As indicated from H&P.
Cardiovascular	No special requirements unless patient has acromegaly, in which case they may have HTN, ischemic heart disease, and diabetes. **Tests:** As indicated from H&P.
Neurological	Secretory tumors of the pituitary are usually small, confined to the sella, rarely cause ↑ ICP (2° direct tumor effects or by obstruction of 3rd ventricle), and commonly produce Sx of endocrine dysfunction early in their growth. In contrast, nonfunctional pituitary tumors may not produce Sx until they extend beyond the boundaries of the sella, causing HA's (the most common presenting symptom) or compression of the optic chiasm, producing visual field defects (temporal or bitemporal hemianopsia). **Tests:** A CT or MRI will delineate the site and size of the tumor, especially if iv contrast material, such as gadolinium, is administered to enhance the margins of the tumor.
Musculoskeletal	If growth hormone is the primary secretant, patient will exhibit Sx of acromegaly, including large hands, feet, head, and tongue. Cushing's myopathy (UE proximal muscle weakness) may affect apparent recovery from muscle relaxant. **Tests:** As indicated from H&P.
Laboratory	Metabolic panel, HCT, and others as indicated from H&P. ↓ Na^+ may indicate DI; ↑↑ Ca^{++} may indicate MEN I.
Premedication	If ↑ ICP is not a concern, patients may benefit from midazolam 1–2 mg iv. In patients with ↑ ICP consider pretreatment with mannitol (e.g., 0.25 gm/kg 20 min before induction). Acromegalic patients may develop airway obstruction following even small doses of sedatives or narcotics.

Intraoperative

Anesthetic technique: GETA is required for this operation, since the surgical approach is through either the nose or the mouth

above the maxillary gum line and behind the nose. A lumbar drain may be placed to permit intraop management of ICP and access to the tumor.

Induction	If a difficult intubation is anticipated (e.g., severe acromegalic patients), orotracheal intubation will need to be accomplished before induction of GA. LMA placement may be difficult and awake FOL is often the best choice (see p. B-5). Because pituitary tumors generally are confined to the sella turcica and, hence, ICP usually is not increased, a standard induction technique with standard ETT is appropriate (see p. B-2). An oral RAE tube may be preferred by oral surgeons. If increased ICP is of concern, induction should be similar to that used for patients with other kinds of brain tumors (see Anesthetic Considerations for Craniotomy for Tumor, p. 31). Because the surgeon will be working from the patient's right side, the ETT must be positioned at the far left side of the mouth. The surgeon may infiltrate the nasal mucosa with lidocaine and epinephrine →↑ BP + dysrhythmias → possible myocardial ischemia.
Maintenance	Standard maintenance with sevoflurane (or propofol infusion) supplemented with remifentanil (see p. 1539). Generally, no further NMBs are administered beyond ET intubation. With adequate anesthesia, and the head in Mayfield clamp, patient movement of any consequence is highly unlikely. Ventilation is controlled with $PaCO_2$ maintained in the normal range. Hyperventilation is not desirable, because it makes it more difficult to access the tumor in the sella and establish that it has been removed in its entirety. Following the resection, the surgeon may request a Valsalva maneuver to check for CSF leak.
Emergence	If the patient evidences normal emergence from anesthesia, the ETT should be removed before vigorous coughing ensues. Before removing the ETT, however, make certain that all blood accumulated in the back of the throat is suctioned out, and that oropharyngeal packs which may have been placed in the back of the throat have been removed. An OG tube should be used to suction out the stomach. The surgeon may have packed the nose at the end of operation, forcing the patient to be an obligatory mouth breather. If there is any question about airway patency because of a large tongue, small mouth, or soft-tissue redundancy in the oropharynx, the ETT should be left in place until patient is fully awake from anesthesia. Prophylactic antiemetics (e.g., metoclopramide 20 mg and ondansetron 4 mg) should be given 30 min before extubation.
Blood and fluid requirements	Minimal blood loss usual Potential large blood loss IV: 16–18 ga × 1 NS @ 4–8 mL/kg/h
Monitoring	Standard monitors (see p. B-1). ± Arterial line ± UO ± CVP line ± precordial Doppler
Positioning	and pad pressure points. eyes. Shoulder roll Table turned 90–180°
Complications	Carotid injury VAE
	Blood loss is minimal, unless the surgeon inadvertently enters the internal carotid artery or cavernous sinus. Additional iv access may be necessary. Monitor for VAE in semisitting position if > 30°. Ulnar artery flow may be compromised in acromegaly—document Alan test before placing radial artery catheter. CVP line should be placed in patients at significant risk for VAE or as medically indicated. The surgeon will use an operating microscope or endoscope, which means that the anesthesiologist will be positioned at the side or foot. Anesthetic hoses and intravascular lines must be long enough to be accessible. Potential for large volume hemorrhage and intraop stroke. Deliberate hypotension may facilitate repair. Carotid compression may →↓ CBF → ischemia.

(Print pagebreak 56)

Postoperative

Complications	Hypopituitarism Diabetes insipidus (24–48h postop) CSF leak SIADH Cranial nerve injury	Replacement therapy with steroids is necessary in hypopituitary patients until normal pituitary function returns. Occasionally, patients will develop DI postop → polyuria and ↓ urine specific gravity < 1.005. Rarely, this may occur in the PACU necessitating fluid replacement and/or DDAVP.
Pain management	Codeine (30–60 mg im q 4 h)	HA is the most common complaint.
Tests	CT scan Serum cortisol Visual acuity/fields	If a patient exhibits any delay in emergence from anesthesia and surgery, or any new neurologic deficits emerge postop, a CT scan is ordinarily obtained.

(Print pagebreak 57)

Suggested Readings

1. Burton CM, Nemergut EC: Anesthetic and critical care management of patients undergoing pituitary surgery. *Front Horm Res* 2006; 34:236–55.

2. Chan VWS, Tindal S: Anesthesia for transsphenoidal surgery in a patient with extreme giantism. *Br J Anaesth* 1998; 60:464–8.

3. Ciric I, Ragin B, Baumgartner C, et al: Complications of transsphenoidal surgery: Results of a national survey, review of the literature, and personal experience. *Neurosurgery* 1997; 40(2):225–36.

4. Elias JW, Laws ER Jr: Transsphenoidal approaches to lesions of the sella. In *Operative Neurosurgical Techniques: Indications, Methods, and Results.* Schmidek HA, Sweet WH, eds. WB Saunders, Philadelphia: 2000, 373–84.

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8. Semple PL, Laws ER: Complications in a contemporary series of patients who underwent transsphenoidal surgery for Cushing's Disease. *J Neurosurg* 1999; 91:175–9.

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Ventricular Shunt Procedures

Surgical Considerations

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Description: Many conditions exist where it is necessary to shunt CSF from the ventricles to another body cavity where it can be

absorbed readily. The most common condition is hydrocephalus, where there is dilation of the ventricular system due to an obstruction in the flow of CSF or decreased absorption of CSF by the arachnoid villi. Hydrocephalus is commonly treated by diverting CSF to the peritoneal cavity via a ventriculoperitoneal shunt (**VP shunt**). Most shunt systems have a one-way, pressure-dependent valve to regulate the flow of CSF. These valves usually have a preset pressure setting (e.g., low, medium, high) at which they open and allow CSF to flow. Newer valves have externally adjustable opening pressures, flow-regulating devices, and/or antisiphon systems to prevent overshunting. Additionally, antibiotic impregnated catheters can be used to reduce the risk of infection.

To insert a VP shunt, the patient is positioned so that the cranial incision and abdominal incision are aligned in the same plane. The scalp is shaved (usually over the frontal or parietal region), and a continuous surgical field is created from head to abdomen. The cranial incision is made over the intended region of cannulation of the ventricle, and a burr hole is made in the cranium. A subgaleal pocket is created for the valve, usually behind the ear. A separate incision is made in the abdomen, and the dissection is carried down to the level of the peritoneum. A catheter is then passed subcutaneously from the abdominal incision to the cranial incision with a special tunneling instrument (Fig. 1.1-20A). Alternatively, the catheter can be threaded from the cranial incision to the abdominal incision. It may be necessary to use one or more incisions between the head and the abdomen to thread the catheter. The valve is connected to the peritoneal catheter and placed in the subgaleal pocket. A ventricular catheter is then inserted into the ventricle (Fig. 1.1-20B) and a small amount of CSF is drained to check placement and patency of the catheter. The ventricular catheter is then connected to the valve, and CSF flow through the entire shunt system is checked by draining some CSF from the distal end of the peritoneal catheter. The distal end is then placed into the peritoneal cavity and all wounds are closed.

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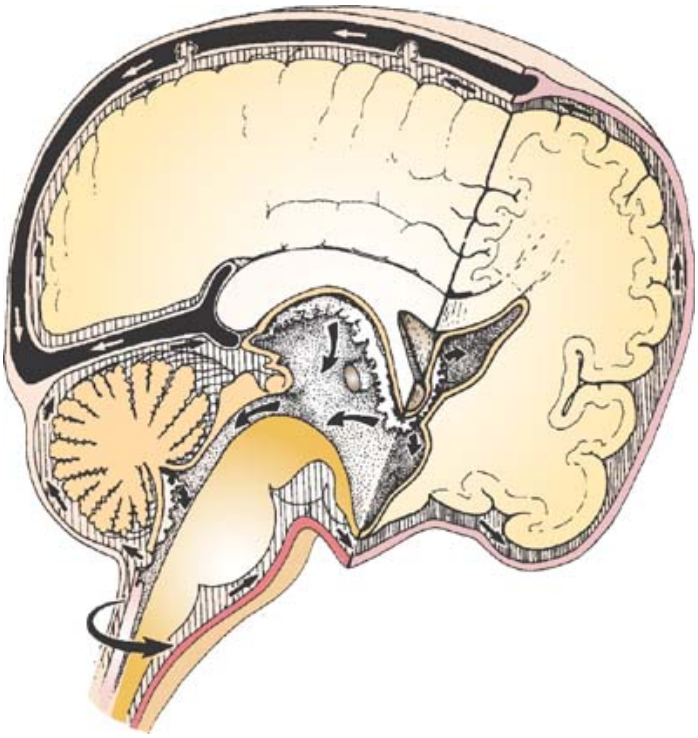

Figure 1.1-19. 19. Pathways of CSF flow and potential sites of obstruction. (Reproduced with permission from Tindall GT, Cooper PR, Barrow DL: *The Practice of Neurosurgery,* Vol III. Williams & Wilkins, 1996. Orig. from Scott RM: *Hydrocephalus. Concepts in Neurosurgery,* Vol III. Williams & Wilkins, Philadelphia: 1990.)

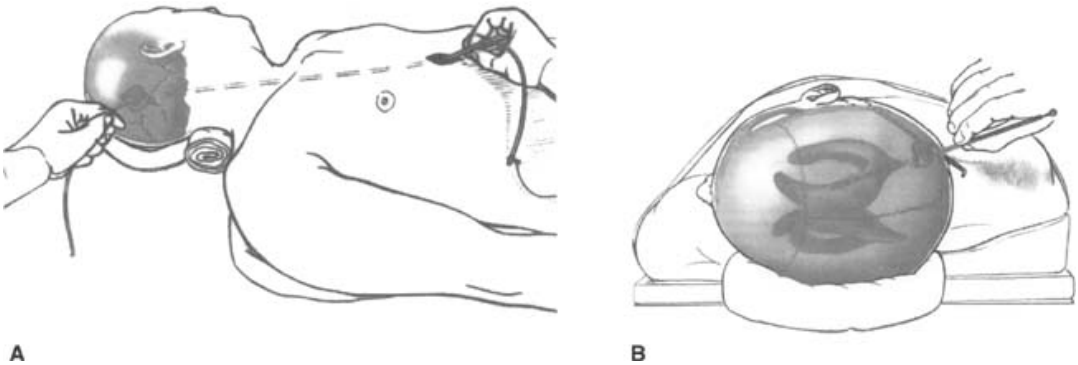

Figure 1.1-20. 20. A. The catheter is threaded from the abdominal incision to the cranial incision. **B.** Insertion of the ventricular catheter. (Reproduced with permission from Meyer FB: *Atlas of Neurosurgery: Basic Approaches to Cranial and Vascular Procedures.* Churchill Livingstone, 1999).

Any component of the shunt may malfunction; thus, it may be necessary to test each component at the time of revision to identify the problem. To test the patency of the ventricular catheter, it is disconnected from the valve and CSF should flow freely through it. A saline-filled manometer can be attached to the valve to measure the opening pressure of the valve. The manometer should drain spontaneously until the opening pressure of the valve is reached. An *(Print pagebreak 59)* elevated opening pressure may indicate a valve malfunction or a distal occlusion. If a distal occlusion is suspected, gentle irrigation may clear it, but externalization may be necessary.

Variant procedure or approaches: Endoscopy, fluoroscopy, ultrasound, image-guided navigation, or intraoperative MRI may be used to help place the catheter into the ventricle. The ventricular catheter can be placed in either lateral ventricle; occasionally, both lateral ventricles are cannulated. This procedure also is used to shunt the 4th ventricle and sometimes, subarachnoid cysts and subdural hygromas. The distal end alternatively may be placed in the pleural cavity or the right atrium (ventriculoatrial or **VA shunt**). If the distal end of the catheter is placed into the pleural cavity, a Valsalva maneuver is performed upon closure to reinflate the lung. To place the distal end into the atrium, a vein is cannulated in the neck (usually IJ, EJ, or facial vein), and the catheter is fed into the atrium under fluoroscopic guidance. It may be necessary to inject radiopaque contrast to verify proper placement. Rarely, the distal end of the catheter can be placed in an intracranial venous sinus, the gallbladder, or the ureter.

Endoscopic Third Ventriculostomy (ETV): Endoscopic third ventriculostomy is being used with increasing frequency to treat both obstructive and communicating forms of hydrocephalus. Head fixation is generally not needed and the procedure involves a small skin incision over the frontoparietal lobe to gain access to the skull. A small burr hole is then made. The dura is opened over the burr hole, permitting the insertion of the endoscope into the lateral ventricle. Under endoscopic guidance, the tuber cinereum is then fenestrated with a balloon tip catheter. CSF is diverted through the membranous floor of 3rd ventricle into the basal cisterns. The endoscope is then removed and the wound is closed.

Usual preop diagnosis: Hydrocephalus; obstructive or communicating hydrocephalus; aqueductal stenosis; Dandy-Walker malformation (cystic dilation of the 4th ventricle and incomplete formation of the cerebellar vermis); occult hydrocephalus; normal-pressure hydrocephalus; subarachnoid cyst; subdural hygroma; pseudotumor cerebri.

Summary of Procedures

	VP Shunt	VA Shunt	ETV
Position	Supine, with head turned to contralateral side		
Incision	Scalp, either coronal and retroauricular, or parietal; + neck and abdomen	Scalp, either coronal and retroauricular or parietal; + neck	
Special instrumentation	Ventricular endoscope (optional)	+ I.I.	
Unique considerations	Patient to be treated as if there is ↑ ICP.		
Antibiotics	Ceftriaxone 1–2 g iv q d (avoid Ca^{++} containing solutions)		
Surgical time	1 h		
EBL	5–25 mL		
Postop care	PACU → room; usually kept flat for 24 h.		
Mortality	< 1%		
Morbidity	Infection: < 15% Neurological: Intracranial bleed: < 1% Subdural hematoma: < 1% Hardware failure: < 1%		Basilar artery injury ↑↑ ICP →↓↓ HR or asystole

Pain score	4–6	2–4

(Print pagebreak 60)

Patient Population Characteristics

Age range	Newborn-elderly
Male:Female	1:1
Incidence	Common
Etiology	Congenital (3–4/1000 live births); acquired; neoplastic; infectious; posthemorrhagic
Associated conditions	Subarachnoid hemorrhage; myelodysplasia; spina bifida (95% incidence); intraventricular hemorrhage; intraventricular tumor

Anesthetic Considerations

Preoperative

Ventricular shunts are inserted to ameliorate hydrocephalus or cyst formation, which may be either congenital or acquired.

Cardiovascular	↑ ICP →↑ BP + ↓↓ HR (Cushing's response) **Tests:** As indicated from H&P.
Neurological	The most common presenting Sx is HA. If hydrocephalus is severe, Sx of ↑ ICP (> 15 mmHg) (e.g., N/V, drowsiness, papilledema, Sz, and focal neurological defects) develop.
Laboratory	Tests as indicated by H&P.
Premedication	Usually not required; should be avoided in patients with ↑ ICP.

Intraoperative

Anesthetic technique: GETA

Induction	If ↑ ICP, iv induction with STP (2–5 mg/kg) or propofol (1–2 mg/kg) is preferred, because of their ability to decrease cerebral blood volume and, hence, to decrease ICP. ET intubation is accomplished with the use of a NMB (e.g., vecuronium [0.1 mg/kg]; or rocuronium [0.6–1 mg/kg]).
Maintenance	Isoflurane ≤ 1% or sevoflurane < 2%, inspired with 50:50 N_2O/O_2 mixture. Depending on duration of operation, additional doses of vecuronium (0.1 mg/kg) or rocuronium (0.2 mg/kg) may be needed. Maintain normal temperature in children by keeping OR warm (78F) and using a forced-air warming blanket. Ventilation should be controlled. TV and frequency should be adjusted such that the $PetCO_2$ = 35–40 mmHg. Hyperventilation and hypocarbia are undesirable because they make cannulation of the ventricle(s) more difficult. Maintain normotension. During endoscopic 3rd ventriculostomy (ETV), puncture of the floor of the 3rd ventricle may be associated with significant ↓↓ HR or asystole. The surgeon should be immediately informed if bradycardia occurs.
Emergence	ETT is removed at the conclusion of the anesthetic. Prophylactic antiemetic (e.g., metoclopramide 10–20 mg and ondansetron 4 mg) should be given 30 min before extubation.
Blood and fluid requirements	IV: 18–20 ga × 1 NS @ 4–6 mL/kg/h Administer crystalloid, usually NS. Blood is rarely, if ever, necessary.
Monitoring	Standard monitors (see p. B-1). Invasive monitors as indicated

Positioning	Table turned 180° and pad pressure points. eyes.	Supine with a bolster under the shoulder on the operative side. The head, chest, and abdomen are prepped, so all anesthesia equipment and lines must be at the sides of the patient.
Complications	Valve malfunction ↓↓ HR or asystole Basilar artery injury	Major intraop complications from this operation are rare. ETV may be associated with severe bradycardia and injury to the basilar artery or its branches.

(Print pagebreak 61)

Postoperative

Pain management	Children < 2 yr: Tylenol suppositories (10–15 mg/kg q 4 h) Adults: meperidine 10–20 mg iv prn

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Craniocervical Decompression (Chiari Malformation)

Surgical Considerations

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Description: Historically, congenital hindbrain abnormalities characterized by cerebellar descent were known collectively as **Arnold-Chiari malformations**; now they are known as **Chiari malformations**. In these abnormalities, portions of the cerebellum protrude through the foramen magnum and may compress the brain stem and upper cervical spinal cord. Frequently, the malformation is accompanied by **syringomyelia**, a condition in which CSF is located abnormally within the spinal cord. In **Type I Chiari** malformation, the cerebellar tonsils are herniated through the foramen magnum. In **Type II Chiari** malformation, the tonsils, vermis, 4th ventricle, pons, and medulla are displaced caudally through the foramen magnum. Type II Chiari malformations are usually found in infants and are associated with myelomeningoceles. In **Type III Chiari** malformations there is a defect in the

back of the head or neck through which the cerebellum and/or brainstem is herniated. Patients with **Type IV Chiari** malformations usually do not survive past infancy.

In order to make room for the brainstem and caudally displaced cerebellum, decompression of the craniocervical junction is necessary. This procedure may be performed in either the prone, 3/4 prone, or seated position. The head is placed in Mayfield pin fixation. A midline incision is made, and the dissection is carried down to the skull and the posterior arch of C1. Paramedian burr holes are placed to aid in the suboccipital craniectomy. The craniotome is then used to remove an approximately 4 cm by 3 cm bone flap that includes the foramen magnum. Care is taken to avoid injury to the underlying venous sinuses when performing the craniectomy.

The posterior arch of C1 is removed and as many upper cervical lamina as are needed to fully decompress the Chiari malformation are removed. Next, any constricting fibrous bands are cut and the dura is opened. Any subarachnoid adhesions should be lysed to allow the free flow of CSF. The tonsils are dissected apart under microscopic guidance *(Print pagebreak 62)* to gain an opening to the 4th ventricle (Figure 1.1-21A,B). The cerebellar tonsils can be reduced with electrocautery or by resection in order to facilitate the outflow of CSF from the 4th ventricle. A stent or a shunt may be placed in the 4th ventricle and brought out to the subarachnoid space to ensure adequate drainage of the 4th ventricle. The dura is closed loosely with autologous pericranium, fascia lata, or bovine pericardium (Fig. 1.1-21C). This creates a widely patent cisterna magna and prevents constriction at the cervicomedullary junction.

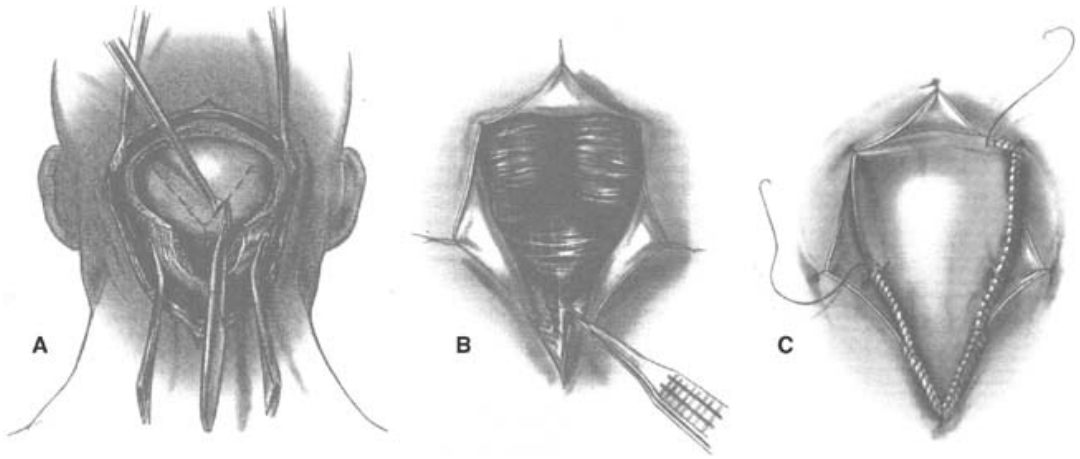

Figure 1.1-21. 21. A. A Y-shaped dural incision is made through the occipital craniectomy and laminectomy. **B.** Opening the arachnoid mater to gain access to the cerebellar tonsils and fourth ventricle. **C.** Loose closure of dura with graft. (Reproduced with permission from Meyer F: *Atlas of Neurosurgery: Basic Approaches to Cranial and Vascular Procedures.* Churchill Livingstone, 1999.)

Usual preop diagnosis: Chiari Malformation I or II, Arnold-Chiari malformation; syringomyelia

Summary of Procedures

Position	Prone or sitting
Incision	Midline posterior, posterolateral thigh for fascia lata graft (optional)
Special instrumentation	Operating microscope
Unique considerations	Risk of air embolus; brain stem manipulation can cause BP and pulse instability; dural patch used to expand dura at foramen magnum
Antibiotics	Ceftriaxone 1–2 g iv q d (avoid Ca^{++}containing solutions)
Surgical time	2.5–3.5 h
Closing considerations	BP control to ensure adequate hemostasis
EBL	25–250 mL
Postop care	ICU or constant observation unit; neurological function monitored.
Mortality	0–3% (usually respiratory arrest)

Morbidity	Respiratory depression: 14% up to 5 d postop All others < 5%: Infection Neurological: dysphagia and ↓ gag reflex Aseptic meningitis CSF leak Massive blood loss; vertebral artery or transverse sinus injury
Pain score	5–7

(Print pagebreak 63)

Patient Population Characteristics

Age range	Infant–70 yr; Chiari I: average age = 41 yr
Male:Female	1:1
Incidence	Relatively rare neurosurgical procedure
Etiology	Congenital; acquired, S/P lumboperitoneal shunting
Associated conditions	Hydrocephalus; syringomyelia; scoliosis; myelodysplasia

Anesthetic Considerations

See Anesthetic Considerations for Cervical Neurosurgical Procedures, p. 106.

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Stereotactic Neurosurgery

Surgical Considerations

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Description: Stereotaxis applies simple rules of geometry to radiographic images to allow precise localization within the brain. Such techniques can be applied to a variety of neurosurgical procedures, providing up to 1-mm accuracy. This precision makes it possible to perform certain intracranial procedures less invasively. Methods include both frame-based and frameless navigation systems, as well as specific stereotactic applications for functional neurosurgery and radiosurgery.

Frame-based stereotaxy: Stereotactic localization was developed using frames; however, with the widespread use of image-guided, ‘frameless’ stereotactic systems, frame-based stereotaxy is rarely used. These procedures begin with the attachment of a frame to the patient's head using four screws that anchor it to the skull. This is typically done outside the OR, using local anesthetic and, sometimes, light sedation. In the cooperative adult, frame application takes only 5–10 min; however, GA typically is used for children. Once the frame is in place, access to the patient's airway is restricted. A key for emergency removal of the frame must be kept with the patient at all times. With the stereotactic frame in place, CT, MRI, and/or cerebral angiography are used for target

identification and localization. During imaging, a set of fiducials (radiographically visible markers) is attached to the frame. These fiducials provide the geometric reference points needed for localization. Data from imaging studies are used to calculate the spatial coordinates of the target(s). In nearly all cooperative patients, there is no need for sedation or analgesia during this stage of the procedure.

Frame-based stereotaxy is used most frequently for brain biopsies; however, drainage of cystic lesions or placement of a catheter or depth electrodes also can be performed. These common procedures involve relatively minor surgery, usually requiring only light sedation and local anesthesia. After prepping and draping, specialized operating tools are *(Print pagebreak 64)* attached to the stereotactic frame. These instruments provide spatial guidance throughout the surgical procedure. A burr or twist drill hole is the usual form of intracranial access. Maximal sedation is needed during drilling and dural opening. Anesthetic considerations include ensuring adequate oxygenation and ventilation, which may be compromised by the sterile drapes. Additionally, sedation should be light enough at times to allow the neurosurgeon to test gross neurologic function, such as extremity movement, both during and immediately after biopsy. At the completion of the case, the frame is removed and the patient is brought to the recovery room.

Image-guided "frameless" stereotaxy: Over the last several years, techniques have been developed to provide neurosurgeons with a real-time computer display of position and trajectory. Spatial information is displayed on high-speed computer work stations as two-dimensional images and three-dimensional renderings obtained from CT and/or MRI. These devices have the added advantage of being frameless; instead of a frame providing a system of reference, small markers (fiducials) are affixed to the scalp and forehead with adhesive. Advances in image-guided navigation also allow the incorporation of ultrasound and endoscopy to provide intraoperative updating of the preoperative radiographic images.

Image-guided surgery begins with an imaging study performed with fiducials in place. The remainder of the case is performed in the OR, usually under GA. After the patient's head is appropriately fixed and positioned, the locations of the fiducials are entered into a computer, using any one of several different commercially available digitizing techniques. The most common system uses triangulation of infrared light from LEDs to determine the position of a pointer in space. The computer calculates the position of the pointer with respect to the patient and displays on the monitor the images from the scan with a representation of the pointer superimposed. Because the process of localization assumes a constant frame of reference, it is important that fiducials do not move between the time of imaging and registration in the OR. A further refinement, the dynamic reference frame, may be affixed to the craniotomy headrest to allow intraoperative repositioning of the patient. The OR must be set up to accommodate the computer and monitor as well as to allow a direct line-of-sight between the operative site and an array of infrared sensors. (See Fig. 1.1-22.) Care must be taken to avoid patient movement during the initial registration of the fiducials, because this induces a component of error with respect to the accuracy of the navigation system. The camera system can be repositioned during the case and still maintain accuracy.

The spatial information provided to neurosurgeons by image guidance can be used to access deep or critically located lesions through the most direct trajectory and a smaller craniotomy. These techniques have been used primarily during craniotomy for tumor or vascular malformation; however, image guidance also can be used for brain biopsy, *(Print pagebreak 65)* shunt placement, cyst drainage or other smaller procedures. As with most craniotomies, GA is used. Maneuvers that alter the spatial relationship of the intracranial contents, such as hyperventilation, diuresis or CSF drainage, will result in brain shift relative to the presurgical images and should be avoided if possible, particularly during the early stages of surgery.

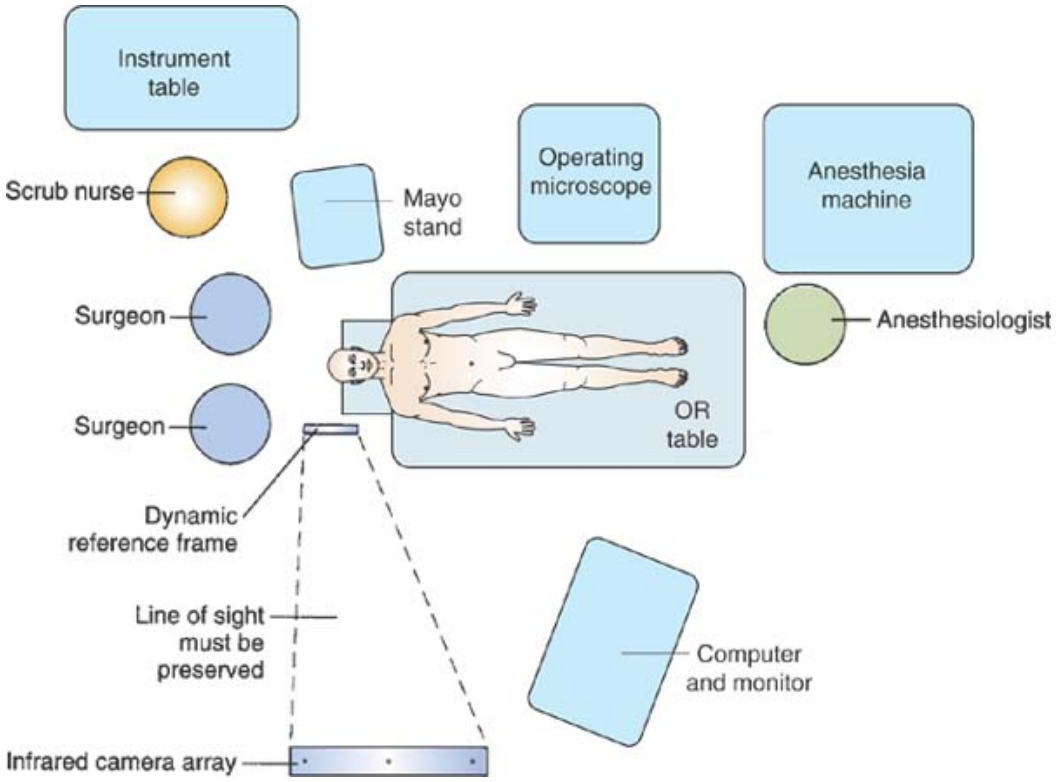

Figure 1.1-22. 22. OR layout for frameless stereotactic surgery.

Radiosurgery: This technique uses focused radiation to ablate small or residual tumors and vascular malformations. It can also be used to treat some pain, psychiatric and movement disorders. In contrast to other stereotactic operations, radiosurgery is performed outside the OR, using a linear accelerator or other specialized irradiation device. Cooperative adults need little or no sedation during the procedure. Children, however, almost always require GA from the beginning of frame placement through the actual treatment, which may last from 5–8 h. Consequently, anesthesia must be administered at several locations in the hospital, as well as during transport between these sites. Ideally, GA is initiated in the imaging suite just prior to frame placement. After transport to the recovery unit, the child is maintained under anesthesia while radiosurgical treatment is being planned, which takes from 1–3 h. The patient is then transported to the radiosurgery suite, still anesthetized, and treatment is performed. Depending on complexity, radiosurgery itself may last from 1–2 h. At the completion of treatment, the stereotaxic frame is removed and the child is awakened.

Newer, frameless radiosurgical systems can obviate the need for GA and most children can be set up and treated with iv sedation alone. However, the set-up and treatment are usually performed on different days and the child can go home while the treatment plan is created. These systems also allow for fractionation of the total dose, which may spare damage to surrounding critical structures.

Functional Neurosurgery: (See p. 70 for additional discussion of these procedures) Surgery using precisely placed lesions (e.g., **pallidotomy**, **thalamotomy**) or deep-brain stimulators for the treatment of movement disorders, pain, and certain psychiatric syndromes are encompassed by the term "**functional stereotaxy**." These techniques depend on absolutely precise localization of specialized instruments. These surgeries traditionally have required the use of a stereotactic frame. However, advances in imaging and image-guided navigation have obviated the need for a stereotactic frame. Concurrent neuroanatomic localization using electrophysiologic techniques is routinely used, regardless of the stereotactic system employed. In order to preserve neural potentials and allow the patient to cooperate with neurologic testing, GA is not used. Frameless procedures begin with placement of the fiducials (small screws) and imaging. Typically the fiducials are implanted in the patient's skull using local anesthetic and a special screwdriver. Then the patient undergoes a CT scan, which is uploaded into the image-guided navigation system. The surgeon can then plan the trajectory in the computer. In the OR, the patient's head is placed in Mayfield pins, and the imaged-guided navigation system is set up and the fiducials are registered. A small skin incision is made, and a burr hole is placed with the patient under iv sedation. The patient is then awakened and must remain awake and cooperative throughout a several-hour procedure, which may require patient participation in various neurologic tests. The recording electrodes are positioned using the image-guided navigation system and the electrophysiologic monitoring. After the proper electrophysiologic position is obtained, the recording electrode is removed and the deep brain stimulator is placed in the exact same position and depth. After the deep-brain stimulator is placed, a subgaleal pocket may be created to house the leads until they can be connected to the pulse generator. Frame based procedures begin with the attachment of a rigid frame to the patient's skull with local anesthesia. The stereotactic frame provides an immobile reference which aids in targeting. The remainder of the procedure is similar to the frameless procedure previously described.

Usual preop diagnosis: Brain disease requiring biopsy; brain tumor; vascular malformation; Parkinson's disease; hydrocephalus; pseudotumor cerebri; pain syndromes; psychiatric syndromes; cyst drainage

Summary of Procedures

	Frame-Based Stereotaxy	Frameless Stereotaxy	Radiosurgery	Functional Neurosurgery
Position	Supine or prone	Sitting, supine, prone, or lateral	Supine or prone	Supine, head fixed to floor stand
Incision	0.5–2 cm, scalp	1–12 cm, scalp	None	1–3 cm, scalp
Special instrumentation	Stereotactic treatment arc	Sensor array, pointer, computer, monitor, dynamic reference frame	Linear accelerator (LINAC) Cyberknife	Electrophysiologic monitoring equipment, image-guided navigation, C-arm
Unique considerations	Key for emergency: removal of frame	1. Minimize brain shift. 2. No patient movement during registration. 3. Clear line-of-sight between camera and dynamic reference frame.	Extended anesthesia in several different locations	Patient must be awake, able to cooperate with testing.
Antibiotics	Ceftriaxone 1g iv (avoid Ca++ containing solutions)	Ceftriaxone 1g iv (avoid Ca++ containing solutions)	None	Ceftriaxone 1g iv (avoid Ca++ containing solutions)
Surgical time	0.5–1.5 h	2–6 h	5–8 h	2–6 h
EBL	< 10 mL	20–1000 mL	None	< 100 mL
Postop care	PACU	ICU	PACU	Neuroobservation unit
Mortality	< 0.5%	1%	None	< 1%
Morbidity	Overall: < 1% Symptomatic intracerebral hemorrhage	InfectionAir emboliHemorrhageStrokeSeizuresCerebral edema	N/V 2–3%	N/ V: 1–10% Intracerebral hemorrhageNew neurologic deficits
Pain score	2	4	2	2

(Print pagebreak 66)

Patient Population Characteristics

Age range	All ages		0–12 yr	18–90 yr
Male:Female	1:1			
Incidence	Uncommon	Unusual		Rare
Etiology	Tumor; infection; AIDS; demyelinating syndromes	Tumor; vascular malformations; epilepsy		Parkinson's disease; pain syndromes; psychiatric syndromes

Anesthetic Considerations

Preoperative

Neurosurgical procedures are performed using stereotactic control when the lesion is small and/or is located deep within brain tissue, or as a means of obtaining a biopsy of a lesion for diagnosis. For example, focal, deep-seated AVMs may be resected under

stereotactic, or image-guided, control. These patients are often otherwise healthy.

Neurological	Neurological Sx vary (depending on site and size of the lesion) and they should be carefully documented. In addition to the usual tests, a CT or MRI scan is obtained preop with the frame in place to determine stereotactic coordinates. Once the coordinates are established, the frame or fiducial markers must not be moved until the operation is complete.
Laboratory	Tests as indicated from H&P.

(Print pagebreak 67)

Intraoperative

Anesthetic technique: GETA or MAC. In adults, fiducial markers or a stereotactic frame are placed before surgery and the patient is taken to the radiologic suite for CT/MRI scan to determine stereotactic coordinates. The patient is then brought to OR with the frame or fiducial markers in place. The key for removing the stereotactic frame must be readily available in the event of an airway emergency. Biopsies generally are done under local anesthesia with MAC. If a complete resection is planned (e.g., AVM resection), GETA is used. In children, it is usually necessary to induce GA before placing the frame, thus necessitating the maintenance of GA during the CT/MRI scan. The child is then moved to the OR, still anesthetized, and the operation is completed.

Induction	If MAC is planned, $O_2$by nasal prongs is administered, and the patient is lightly sedated with combinations of propofol 25–50 mcg/kg/min ± midazolam 1–4 mg/kg in divided doses to provide amnesia ± remifentanil 0.02–0.05 mcg/kg/min to provide analgesia. It is important that the patient be able to communicate with the surgeon as needed throughout the operation. For functional neurosurgery (e.g., pallidotomy), sedation should be minimized to preserve normal electrophysiological activity essential for mapping. If GETA is needed in a framed stereotactic procedure, FOL is necessary before inducing anesthesia because the frame precludes intubation by direct laryngoscopy (see p. B-5). After ET intubation is established, anesthesia may be induced with STP 2–5 mg/kg or propofol 1–2 mg/kg, followed by a nondepolarizing NMB to facilitate positioning of patient.
Maintenance	If GA is used, maintenance is the same as for a tumor (see Anesthetic Considerations for Craniotomy for Tumor, p. 31) or AVM (see Anesthetic Considerations for Craniotomy for Intracranial Vascular Malformations, p. 19). If children are to be transported from the site of placement of the stereotactic frame to the radiologic suite and then to the OR, it is best to use a propofol infusion (e.g., 75–150 mcg/kg/min) with spontaneous or controlled ventilation to assure adequate ventilation and oxygenation during transport and study. Opiates and nondepolarizing NMBs should not be administered until the child is in the operating suite.
Emergence	ETT generally is removed at the conclusion of the operation. Prophylactic antiemetic (e.g., metoclopramide 10–20 mg and ondansetron 4 mg) in adults should be given 30 min before extubation.
Blood and fluid requirements	IV: 16–18 ga × 2 (adults); 20–22 ga (children) NS @ 4–6 mL/kg/h Blood loss is minimal because the volume of tissue removed is small.
Monitoring	If local anesthesia: standard monitors (see p. B-1). If GA: Arterial line CVP line UO
Positioning	and pad pressure points. eyes.

Postoperative

Complications	Bleeding	Focal bleeding may occur postop, causing onset of a neurological deficit.
Pain management	Vicodin (1–2 mg po q 4 h prn)	
Tests	CT or MRI scan, if a new neurological deficit occurs.	

(Print pagebreak 68)

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