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CHAPTER 1.2

Functional Neurosurgery

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Functional Neurosurgery—Introduction: The Surgical Treatment of Pain, Movement Disorders, and Epilepsy

Introduction

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Functional neurosurgery differs from most surgical subspecialties in that it aims to alter the function of the nervous system, rather than addressing anatomical abnormalities. In 2008, the three most common treatment areas are chronic pain, epilepsy, and movement disorders. Each of these disease states has a distinctive anesthetic requirement dictated by the specific pathophysiology and subsequent intraop objectives. Procedures may be performed under local anesthesia, conscious sedation, or GA, and can be divided into ablative (destructive) or augmentative (nondestructive) techniques. With the exception of the treatment of epilepsy and some specific chronic pain disorders, ablative techniques are now seldom performed because of their unpredictable duration of effect and irreversible nature of the lesion. Neuroaugmentation (electrical stimulation or continuous local drug administration) tends to produce more lasting benefits and, more importantly, can be changed to meet fluctuations in the patient's symptoms.

The key to successful functional neurosurgical therapies is target identification. Many targets can be identified anatomically on appropriate radiographic studies, whereas others may be difficult to visualize even with special techniques. However, even areas that can be targeted anatomically may vary in terms of their physiological function. Thus, it is important to incorporate physiological testing in many functional neurosurgical procedures. For pain management, this involves the identification of a target nucleus, as well as the appropriate somatotopic location *(Print pagebreak 71)* matching the patient's complaints. In the case of movement disorders, target location is often the region that is involved in the generation of tremor or abnormal movements. These regions may also be closely associated with areas involved in higher cognitive behavior. Thus, the need for physiological identification often dictates a specific anesthetic technique.

Stereotactic Procedures: Deep Brain Stimulation, Pallidotomy, and Thalamotomy

Surgical Considerations

Description: Many procedures are currently performed with **frame-based stereotactic techniques,** with intracranial access achieved via a burr hole or twist drill. There is a growing trend to use “frameless” stereotactic approaches, with two major systems currently in use. Real-time frameless localization can be achieved by replacing the stereotactic frame with rigidly attached skull markers (“fiducials”) that provide a fixed reference. Another innovative alternative is to produce a fixed trajectory guide using rapid prototyping techniques. Both of these approaches have been used successfully and appear to be equivalent in accuracy to a stereotactic frame.

Two approaches to identification of the functional target are used following initial stereotactic CT or MRI radiographic localization; both require patient cooperation. In one approach, the target is confirmed by assessing symptomatic resolution during high-frequency macrostimulation and by identifying surrounding structures using their characteristic stimulation-evoked responses. In the other approach, before stimulation testing, single-neuron recordings are performed to localize the appropriate target through somatotopic kinesthetic and/or somatosensory responses. In an awake patient, appropriate stimulation is delivered to the skin, or by passive and active movement of the joints. This technique utilizes specialized high-impedance microelectrodes and amplifiers

susceptible to interference from monitoring equipment, which may necessitate manual measurements of BP and clinical assessment of oxygenation. Anesthesia or sedation can modify neuronal activity significantly and, thus, interfere with functional mapping. For example, propofol has been shown to inhibit globus pallidus neurons for several minutes beyond its behavioral effects. Additionally, many of these agents have been shown to change evoked potential (EP) responses, raising the possibility that they could alter stimulation thresholds for either the internal capsule or optic tract.

Thermal ablation for pallidotomy or thalamotomy typically is performed with a radiofrequency (RF) lesion generator operating at 500 kHz, which may interfere with some types of monitoring equipment; however, the ablation process only lasts approximately 12–30 sec.

A variety of movement disorders are amenable to surgical treatment. They can be divided into akinetic (e.g., Parkinson's) and hyperkinetic (e.g., dystonias, tremor, spasticity) syndromes. Idiopathic **Parkinson's disease** is best treated with **deep brain stimulation (DBS)** targeted to a functional subcomponent of either the subthalamic nucleus or globus pallidus interna (posteroventral **pallidotomy,** or PVP, Fig 1.2-2). Tremor syndromes are treated by DBS, targeting the ventral intermedius thalamic nucleus (**thalamotomy** or stimulation), whereas dystonias are treated by stimulation of the globus pallidus interna. **Spasticity** is not treated stereotactically, but through the administration of intrathecal baclofen (GABA-like synaptic inhibition) in the lumbar cistern via an internalized pump system. Abrupt loss of intrathecal infusion of baclofen, however, can quickly lead to a serious withdrawal syndrome with dysautonomia, circulatory collapse, and death within hours. Spasticity refractory to intrathecal baclofen may be amenable to selective dorsal root rhizotomy performed through an open laminectomy.

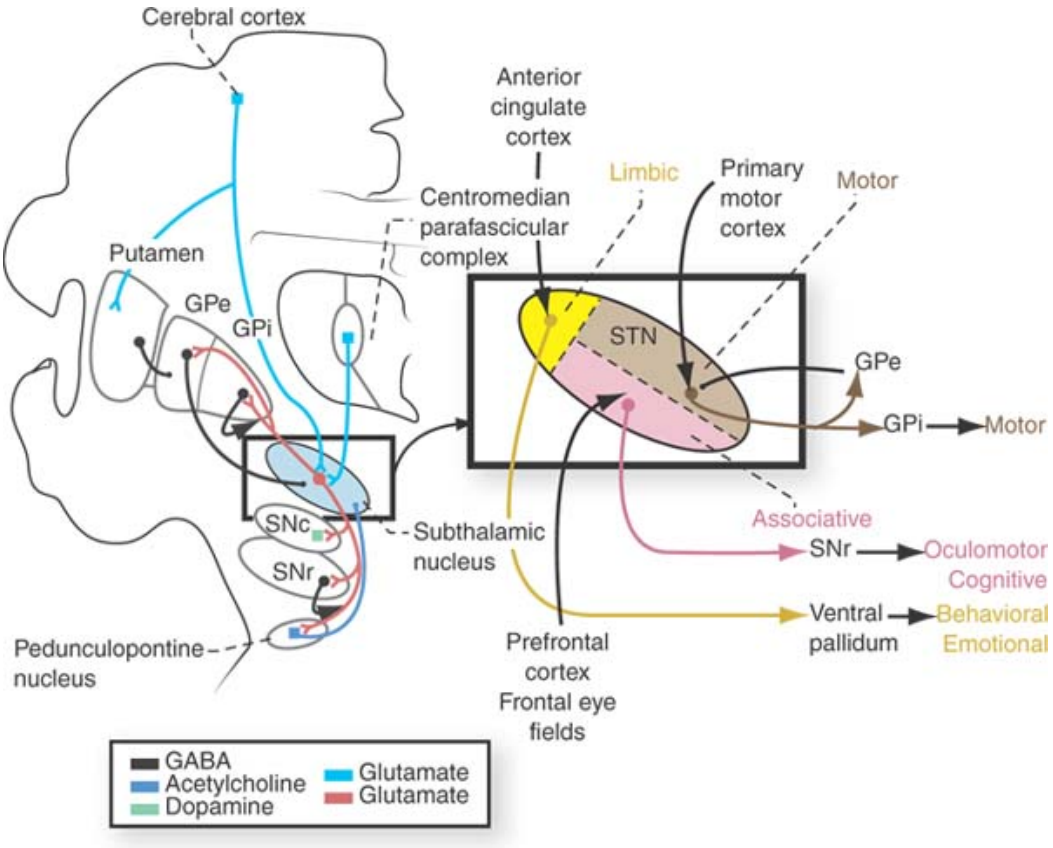

Figure 1.2-1. 1. Topographical anatomy of the ventrolateral thalamus. CM = centrum medianum; GPe = external segment of globus pallidus; GPi = internal segment of globus pallidus; Put = putamen; LPO = lateropolaris; VOA = ventrooralis anterior; VOP = ventrooralis posterior; VIM = ventrali intermedius; Vce and Vci = ventralis caudalis externus and internus. (Reproduced with permission from Grossman RG, Loftus CM: *Principles of Neurosurgery,* 2nd edition. Lippincott-Raven, 1999.)

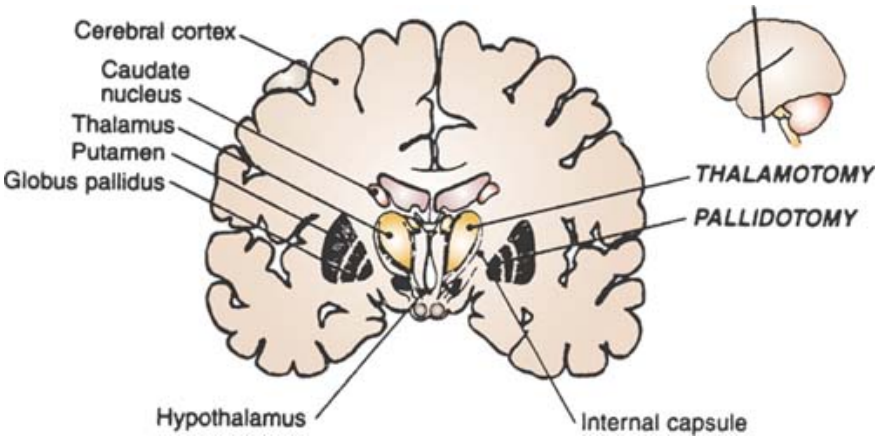

Figure 1.2-2. 2. Anatomical locations for therapeutic lesions (thalamotomy and pallidotomy) for the surgical treatment of Parkinson's disease. Inset shows the plane of the coronal section through the diencephalon, identifying the lesions. (Reproduced with permission from Mason LJ, Cojocaru TT, Cole DJ: Surgical intervention and anesthetic management of the patient with Parkinson's disease. In *International Anesthesiology Clinics: Topics in Neuroanesthesia.* Jaffe RA, Giffard RG, eds. Little, Brown, Boston: 1996; 4(34):141.)

For **DBS,** mapping and implantation of the intracranial stimulating electrode is performed under local anesthesia to permit monitoring of behavioral and physiologic responses. A 2–3 cm linear incision (burr-hole access) or stab wound (twist-drill access), generally is placed near the coronal suture and 10–50 mm from the midline in the frontal bone. This can be accomplished under propofol sedation for patient comfort. Subsequently, minimal or no sedation is used, as patient cooperation is necessary during the functional mapping component of the case. If single-neuron recordings are used, propofol should be discontinued at least 20 min in advance of mapping, because it can produce prolonged suppression of target neuronal activity. If sedation is needed, use a low-dose (0.01–0.05 mcg/kg/min) remifentanil infusion. Local anesthetics should not contain epinephrine. Avoid central-acting β-blockers (e.g., propranolol), which suppress tremor activity in target neurons. Sedation with meperidine in patients taking *(Print pagebreak 72)* selegiline is contraindicated. No dopaminergic antagonist (e.g., metoclopramide, droperidol) should be given to Parkinsonian patients or patients with dopamine-responsive dystonia. To enhance single-cell responses, withhold medications prescribed for target symptoms for 8–24 h. In some cases (e.g., Parkinson's disease), this may result in a rebound HTN requiring active treatment; however, patients will resume their medications postop (often in the post-anesthesia recovery area) and the HTN will resolve. The **implantable pulse generator (IPG)** may be placed in an infraclavicular subcutaneous pocket at the time of the intracranial electrode implantation. Implantation of the IPG and subcutaneous tunneling of the electrode lead can be done under MAC, but is best tolerated under GETA. To facilitate intubation, the calvarial wound is closed temporally and the stereotactic localizing apparatus is removed. Closure consists of a single, interrupted suture for stab wounds or two-layer suture/staple closure for a burr hole. IPG pockets are closed in two layers. The subcutaneous layer is closed with absorbable sutures, and the skin is closed with staples or sutures.

Usual preop diagnosis: Medically intractable idiopathic Parkinson's disease with disabling L-Dopa-induced dyskinesia, bradykinesia, or rigidity; severe fluctuations in medication responses; dystonia musculorum deformans; post-CVA dystonia; occasionally, torticollis; tremor; movement disorders

Summary of Procedures

Position	Supine
Incision	Linear (burr holes); stab-wound (twist drill)
Unique considerations	Minimal/no sedation. BP ↑ in patients who have their antihypertensive medications withheld. Manual vital sign monitoring as electronic monitors may interfere with single-cell recordings.
Antibiotics + other meds	Cefazolin 1 g; dexamethasone 6–8 mg
Surgical time	3–6 h
EBL	25–150 mL
Postop care	Continue antibiotics, Parkinson's medications. BP may ↓. Maintain MAP <90 mmHg to prevent postop bleeding. ICU vs. step-down.

Mortality	<0.5%
Morbidity	Overall: 8–27% Infection Hardware failure: Rare Intracranial hemorrhage Cognitive disturbances
Pain score	2–3

Patient Population Characteristics

Age range	12–80 yr
Male:Female	1:1
Incidence	Becoming common
Etiology	Idiopathic; genetic
Associated conditions	Dystonia

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Anesthetic Considerations

Parkinson's disease is the most common movement disorder, affecting 1% of the population > 60 yr. It is caused by the loss of dopaminergic neurons in the substantia nigra → ↓ dopamine (dopamine/acetylcholine imbalance) in basal ganglia → movement disorder. Medical treatment is directed primarily to restoring dopamine levels by increasing the availability of the dopamine precursor (L-dopa), inhibiting liver dopa decarboxylase (carbidopa, usually given in combination with L-dopa as Sinemet), by releasing endogenous dopamine (amantadine [Symmetrel]) and by blocking MAO-B (selegiline [Eldepryl]). Medical treatment also may include dopamine agonists (pergolide [Permax]; bromocriptine [Parlodel]), and acetylcholine antagonists (amantadine [Symmetrel], benztropine [Cogentin]) to correct the dopamine/acetylcholine imbalance. Patients presenting for surgery have failed medical therapy and will have been taken off their antiparkinsonian medications 8–24 h before surgery. This will maximize their symptoms to help assess treatment effects intraop; thus, preop assessment on the day of surgery will be difficult.

Preoperative

Respiratory	Autonomic dysfunction → esophageal dysfunction →↑ risk of aspiration. Patients typically have ↓ vital capacity 2° rigidity, dyskinesia →↓ respiratory function and ↓ cough. Laryngospasm and respiratory failure may occur following withdrawal of antiparkinsonian medications. **Tests:** CXR; consider ABG; PFTs may be difficult to obtain.
Cardiovascular	Autonomic dysfunction → orthostatic hypotension. Dopamine replacement therapy → cardiac dysrhythmias, ↓ BP, and hypovolemia. In patients taking selegiline (MAOI): avoid meperidine →↑↑ BP, rigidity, agitation; sympathomimetics → exaggerated ↑ BP.
Neurological	The primary Sx of Parkinson's disease include rigidity, tremor, bradykinesia, muscle weakness. Secondary symptoms include dementia, depression, and speech difficulty. Patients may alternate between a state of immobility and one of exaggerated tremor (which may interfere with intraop monitoring).
Renal	Urinary retention is common.
Gastrointestinal	Autonomic dysfunction → gastroparesis, ↑ incidence of reflux. Poor nutrition. Pharyngeal muscle dysfunction → dysphagia.
Laboratory	As indicated from H&P.

Premedication	None. These patients may have received small doses of fentanyl and/or midazolam to facilitate placement of the stereotactic frame. For frameless procedures, fiducial markers or mounting screws will have been placed before surgery.

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Intraoperative

Anesthetic technique: MAC, with minimal or no sedation, as patient cooperation and typical neuronal activity are essential for the success of the procedure.

Induction	Low-dose remifentanil (0.01–0.05 mcg/kg/min) or propofol (25–75 mcg/kg/min) may be used for sedation during burr-hole placement only. If propofol is used, it should be discontinued 20–30 min before electrophysiologic recording or mapping. In the event of an airway emergency in framed procedures, a means of releasing the patient from the stereotactic frame must be readily available.	
Maintenance	BP control is important to decrease the risk of intracranial hemorrhage during passage of the recording and stimulating electrodes. If not otherwise contraindicated, MAP should be kept 10–20% below normal for that patient. This usually can be accomplished without an arterial line by using an infusion of NTG titrated to effect or by using small doses of atenolol (0.5–1 mg increments iv). Atenolol is preferred over other parenteral β-blockers, because its CNS penetration is limited → minimal effect on central tremor.	
Emergence	Postop BP control can be continued by using atenolol or labetalol titrated to effect at the end of the procedure. The patient is usually transported to the PACU, then to the neurology ward for postop monitoring. Antiparkinsonian medication should be given in the PACU.	
Blood and fluid requirements	Minimal blood loss IV: 18–20 ga ×1 NS/LR @ 1–2 mL/kg/h	For unilateral procedures, IV should be placed in the ipsilateral arm (relative to side of surgery).
Monitoring	Standard monitors (see p. B-1)	Exaggerated tremors and/or neurologic testing in these patients may interfere with monitoring. It may be helpful to place the BP cuff on a leg (less tremor). For unilateral procedures, monitors should not be placed on the contralateral arm, which must be kept free for testing.
Positioning	and pad pressure points. stereotactic frame clearance.	Patient comfort may be improved by placing pillows under the knees to relieve lower back strain. A massage therapist can greatly improve patient comfort during the procedure.
Complications	Intracerebral hemorrhage Loss of airway in stereotactic frame	Dx: ↓ mental status and hemiparesis. CT scan usually required to confirm Dx. Emergency craniotomy may be necessary. Rx: remove stereotactic frame and secure airway.

Postoperative

Complications	Intracranial hemorrhage Motor deficit Visual field deficit Aphasia	Intracranial hemorrhage may require emergency craniotomy. The surgeon should be notified immediately if any new deficits are noted.
Tests	None	Unless otherwise indicated.
Pain management	Usually not necessary	

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Surgical Analgesics: Spinal Cord Stimulation, Intrathecal Pumps, and Cortical Stimulation

Surgical Considerations

Description: Chronic pain arises from a variety of etiologies. It can involve both neuropathic and nociceptive processes and occur in a variety of anatomical distributions (e.g., radicular versus a hemibody pattern). Therapeutic interventions are dictated by the pathophysiology of the pain, its qualitative nature, etiology, and the patients' prognosis. Common procedures for chronic pain are directed at the spinal cord, and may consist of epidural or intrathecal medications or electrical stimulation.

For **chronic spinal cord stimulation** (e.g., postlaminectomy syndrome) an epidural electrode is implanted either percutaneously or via an open laminectomy. **Percutaneous electrodes** are easier to implant and have less associated surgical pain. The surgical electrode (implanted via laminectomy) confers greater mechanical stability in the epidural space. Additionally, given its larger contact size, it can generate higher current densities with less drain on the implanted system. For percutaneous electrodes, a small skin incision is made two to three vertebral levels caudal to the target region of the spinal cord. For **"surgical paddle" electrodes,** the skin incision is made one to two levels caudal to the target zone of the spinal cord, and a laminectomy is performed to provide access to the epidural space. Incisions are closed in the standard fashion.

In either technique, patient cooperation is required during intraop test stimulation. The procedures are done in the prone or lateral position with consequent implications for airway management in the sedated patient. Localization of the electrodes is accomplished initially based on radiographic criteria; however, these localizations are only approximate, and it is recommended that the electrode placement be confirmed by intraop stimulation. The patient needs to be sufficiently alert to communicate the quality, distribution, and intensity of the stimulation-induced paresthesias. To assess efficacy, the electrode may be externalized and percutaneous stimulation used for assessment. If there is ≤50% reduction in pain, the patient may return to the OR for internalization of the implantable pulse generator (IPG). Postop, these patients can have an exacerbation of pain, particularly if neuropathic in nature.

They may require iv lidocaine or ketamine infusions to return them to their preop baseline, even with a functioning and appropriately located stimulating electrode.

For **pain of central origin** (e.g., poststroke, multiple sclerosis, and trigeminal nerve pathology), surgical procedures are directed at the thalamus and cortex. Thalamic interventions include stereotactic insertion of stimulating electrodes into sensory thalamus. **Thalamic DBS** requires awake mapping of the somatotopic representation of the affected body part through either microelectrode mapping or stimulation-induced paresthesias. For medically intractable neuropathic pain syndromes, **epidural motor cortex stimulation** has shown great promise. Functional mapping of the motor cortex for epidural stimulation can be done with anatomical and physiological mapping under GA via a craniotomy, with the anesthetic tailored to minimize interference with EP recording. The incision consists of a 5–10-cm linear or 5 × 10 cm trapezoidal incision placed over and paralleling the motor cortex.

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Identification of the appropriate location on the motor strip can be done initially with epidural mapping using SSEPs elicited from the part of the body where the pain originates. In cases of denervation syndromes, SSEP may not be present and the minimum intensity point for epidural stimulation-induced movements is used. In rare cases, the surgeon may elect to perform the surgery awake to facilitate mapping. To assess efficacy, the electrode may be externalized and percutaneous stimulation assessed for overall therapeutic efficacy. If there is ≤ 50% reduction in pain, the patient will return to the OR for internalization of the IPG. Closure consists of calvarial reconstruction with externalization of leads. The craniotomy incision is closed in the standard fashion. If a percutaneous trial is done, the IPG implant requires a second GETA with reopening of wounds.

The surgical treatment of trigeminal neuralgia is **microvascular decompression of the trigeminal nerve** in the prepontine cistern (see p. 44). In patients who are poor surgical candidates, treatment is accomplished with ablative procedures aimed at the gasserian ganglia, using hemolytic, mechanical, or RF techniques. Patients who fail gasserian ganglion interventions or have atypical facial pain are candidates for **thalamic DBS** or **motor cortex stimulation.** Surgical treatments for trigeminal neuralgia, however, may become obsolete, given the advent of **stereotactic radiosurgical ablative techniques.** These systems utilize multiple beams of radiation focused on the nerve to achieve pain relief equivalent to microvascular decompression without the attendant complications of an invasive neurosurgical procedure.

For pain unresponsive to spinal cord stimulation or because of unacceptable side effects of parental medications, continuous intrathecal administration of analgesics can be accomplished with an **implantable medication delivery system.** An incision is made over the L3–L4, L4–L5, or L5–S1 spinous process and a second incision is placed in the RLQ of the abdomen. A tunnel tool is used to bring the catheter from the lumbar spinal region to the abdomen. This tunnel is rarely tolerated without GETA or deep sedation. A reservoir or continuous-delivery pump is then placed in the abdomen and attached to the catheter. Both incisions are closed in two layers.

Intrathecal narcotics (typically morphine), can produce satisfactory results, since therapeutic drug concentrations can be achieved at the level of spinal opiate receptors without influencing higher CNS opiate systems. This therapeutic intervention can be assessed through a percutaneous catheter trial. Patients who experience ≤ 50% reduction in pain are candidates for a **totally implanted pump system.** These are low-morbidity procedures (5–10%) with infections and hardware failures constituting the greatest problems. Abrupt cessation of medications—through either patient noncompliance with refill schedules (10–12 wk) or hardware failure—however, can lead to a serious withdrawal syndrome.

Usual preop diagnosis: Chronic pain

Summary of Procedures

Position	Prone/lateral
Incision	2–3 levels above target area (spinal cord stimulation) + laminectomy; L3, 4, 5 (intrathecal pump) + 2nd incision RLQ of abdomen.
Unique considerations	Patient communication important; therefore, avoid oversedation. Copious use of local anesthetics (avoid overdosing).
Antibiotics	Cefazolin 1 g
Surgical time	1.5–3 h
EBL	25 mL
Postop care	May have ↑ pain

Mortality	0.5%
Morbidity	Overall: 5–10% Infection Hardware failure Withdrawal Sx
Pain score	3–8

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Patient Population Characteristics

Age range	20–75 yr
Male:Female	1:1
Incidence	Common
Etiology	Iatrogenic; neuropathic; traumatic
Associated conditions	Opiate dependency

Anesthetic Considerations

Preoperative

Patients with chronic pain often present unique challenges to the anesthesiologist and surgeon. They may be intolerant of any additional pain (e.g., iv placement, positioning on the OR table, postop pain) and seemingly immune to typical doses of systemic analgesics while remaining vulnerable to opioid overdose. In most cases, patients should continue their usual pain medications and co-analgesics until 2 h before surgery. Typically any transdermal fentanyl patch should be left in place unless its location will be subject to intraoperative heating, compression, or other manipulation. A detailed H/o of patient pain, effective (and ineffective) pain management techniques and drugs, and the current medication regime (including the use of both over and under the counter drugs and herbals) is essential.

Respiratory	Chronic pain →↓ physical activity →↓ respiratory reserve.
Cardiovascular	Ventricular dysrhythmias have been associated with methadone at doses > 200 mg/day. Chronic pain →↓ physical activity →↓ cardiovascular reserve.
Neurological	A careful neurologic assessment is essential to detect pre-existing deficits.
Psychological	These patients often benefit from a detailed explanation of the surgical and anesthetic procedures including expectations and contingency plans for intraop pain management if an awake procedure is planned. These patients may have a higher incidence of co-existing psychiatric disease and often have high scores on standardized measures of hysteria and hypochondria. Patients on antipsychotic medication may develop neuroleptic malignant syndrome.
Gastrointestinal	Chronic opioid use may →↓ gastric emptying and ↓ GI motility with chronic constipation. Inactivity 2° chronic pain → obesity.
Laboratory	As indicated from H&P. For patients on anticonvulsants, drug levels are useful to exclude toxic effects.
Premedication	Chronic opioid use may make the patients at risk for gastric aspiration 2° ↓ bowel motility. Consider metoclopramide 10–20 mg iv 30 min preinduction with Na citrate 30 mL po on transport to the OR. Midazolam iv titrated to effect can be beneficial.

Intraoperative

Anesthetic technique: The placement of intrathecal pumps and cortical stimulators is often best accomplished under GA using anesthetic techniques appropriate for spinal or intracranial procedures. Accurate electrode placement may require an awake patient during at least a portion of the procedure. For intracranial electrode placement, see the anesthetic technique for awake craniotomy (p. 34). These same general principles can also be used for spinal cord electrode placement, although regional techniques are usually simpler and just as effective.

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Thoracic epidural	Following the application of standard monitors and with an iv catheter in place, the patient should be seated carefully on the edge of the operating table. Provide support for feet and arms as necessary. The epidural injection should be at the level of the surgery (usually 2–3 levels below the electrode target site). A single bolus injection of 10 mL of 0.75% bupivacaine usually provides excellent surgical anesthesia without compromising the ability to map the more rostral portion of the spinal cord. It is important to avoid oversedation and to minimize the use of systemic analgesics in order to ensure accurate spinal cord mapping.	
Blood and fluid requirements	IV: 18 ga ×1 NS/LR @ 2–4 mL/kg/h	After replacement of preop deficit
Monitoring	Standard monitors (see p. B-1).	
Positioning	Prone and pad pressure points. eyes	The patient should self-position on bolsters (hips and upper chest) or Wilson frame.
Complications	Spinal cord injury	

Postoperative

Complications	Spinal cord injury Epidural hematoma	Prompt postop neurologic assessment is essential.
	Respiratory distress Drug withdrawal symptoms	May occur in response to the patient's usual opiate dose if the spinal stimulation has produced significant pain relief.
Tests	None unless otherwise indicated	Surgeons may request specific tests.
Pain management	Typically complex and multimodal	Patient often intolerant of postop pain while remaining susceptible to narcotic overdose. Acute pain service consult recommended.

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Vagal Nerve Stimulation

Surgical Considerations

Description: The surgical treatment of medically intractable epilepsy consists of either **surgical resection** of the epileptic site (see p. 81) or **vagal nerve stimulation (VNS).** For patients who are not candidates for resective surgery, VNS can be a viable surgical strategy. Although it is a low-morbidity procedure, its efficacy also is low. VNS tends to produce a 50% or greater reduction of Sz in 48% of patients after 18 mo of stimulation. *(Print pagebreak 79)* By contrast, **temporal lobectomy** for patients with medial temporal lobe epilepsy has an 80–85% chance of making the patient seizure-free, while using only one anticonvulsant medication, or none at all. Surgery for **extratemporal epilepsy** originating in neocortex has a 50% chance of making the patient seizure-free, in the absence of a structural lesion. (If an anatomical abnormality is present, then that figure increases to 75%.) In general, the complication rate is 7%, dependent on the exact location of the Sz focus and its relationship to eloquent cortex.

Left vagal nerve stimulation is also used to treat some types of medically refractory **depression**. As with its use in epilepsy, the exact mechanism of action remains unclear.

Two incisions are used for VNS: one left anterior cervical, placed approximately at the C6–C7 level, with a second incision in the left infraclavicular region for placement of the implantable pulse generator (IPG). Most surgeons perform the procedure through a carotid-type incision and place the IPG caudally through blunt dissection. The left vagus nerve is isolated, and the electrode assembly is wrapped around the nerve below the origin of the superior and inferior cervical cardiac branches. During surgery, the interface between the vagal nerve and the electrode is tested with electrical stimulation. Fortunately, this is associated with a very low incidence of bradycardia and extremely rare reports of asystole. All resolve with cessation of stimulation and administration of atropine. The surgeon should inform the anesthesiologist when vagal stimulation is about to begin. The incisions are closed using the standard technique; however, because many patients are developmentally delayed, the final skin layer is often closed with a subcutaneous technique that does not require subsequent suture removal.

Usual preop diagnosis: Partial onset seizures refractory to medication; chronic or recurrent depression refractory to other medical treatment

Summary of Procedures

Position	Supine
Incision	Anterior cervical (C6–7) + infraclavicular pocket (for IPG)
Unique considerations	Bradycardia → asystole during stimulation phase
Antibiotics	Cefazolin 1 g
Surgical time	1.5 h
EBL	<50 mL
Postop care	Observe for ↑ Sz activity postop × 8 h
Mortality	<0.5%
Morbidity	Hoarseness: 50%, may be 2° nerve injury or fatigue, device malfunction Cough: 40% in first 3 mo after surgery Infection: 2% Dysphagia during stimulation →↑ aspiration risk Dyspnea during stimulation; patients with OSA may have ↑ apneic events Nerve/vascular injury (similar to carotid surgery)
Pain score	1–2

Patient Population Characteristics

Age range	12–80 yr (≤18 yr for treatment of depression)

Male:Female	1:1
Incidence	>30,000 patients treated worldwide thru 2007
Etiology	Idiopathic, typically
Associated conditions	Developmental delay; severe depression

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Anesthetic Considerations

Preoperative

Antiepileptic medication, such as phenytoin, will abolish seizure disorders in most patients, but some develop intolerable side effects; others are refractory to medical therapy. Surgical ablation of the Sz focus may be the only effective therapy for some patients. For patients who are not candidates for ablative procedures, the placement of a vagal nerve stimulator may be a viable alternative. These patients typically take multiple antiepileptic medications that should be continued throughout the morning of surgery. These same general considerations are also applicable to patients with refractory depression.

Neurologic	The only common neurological finding in epilepsy patients is a Hx of uncontrollable Sz, either focal or generalized. Obtain a description of Sz and prodromal Sx.
Psychiatric	Medications used to treat depression (e.g., MAOI, SSRI) may have significant interactions with other drugs (e.g., meperidine → serotonin syndrome)
Gastrointestinal	Abnormal liver function may be associated with use of valproate and carbamazepine. **Tests:** LFT and others as indicated from H&P.
Hematologic	Phenytoin/phenobarbital →↓ Hct; carbamazepine/valproate/ethosuximide/primidone →↓ Plt; carbamazepine/primidone →↓ WBC. **Tests:** CBC and others as indicated from H&P.
Laboratory	Tests as indicated from H&P.
Premedication	Standard premedication (see p. B-1) is usually appropriate.

Intraoperative

Anesthetic technique: GETA. Chronic use of antiepileptic medications may cause hepatic enzyme induction → accelerated drug metabolism; resistance to muscle relaxants including succinylcholine.

Induction	Standard induction (see p. B-2). Once anesthesia is induced, a nondepolarizing NMR is administered.	
Maintenance	Standard maintenance (see p. B-2). Generally, no further NMBs are administered beyond those used for tracheal intubation. Hyperventilation may promote Sz activity. Sevoflurane and enflurane can induce epileptiform EEG activity.	
Emergence	No special considerations. Prophylactic antiemetic (e.g., ondansetron 4 mg) should be given 30–60 min before extubation.	
Blood and fluid requirements	Minimal blood loss IV: 18 ga NS/LR @ 4–6 mL/kg/h	Possibility of injury to carotid artery or jugular vein may result in significant blood loss.
Monitoring	Standard monitors (see p. B-1). EEG monitoring not necessary	O_2sat and ETCO$_2$monitoring will indicate the adequacy of ventilation and oxygenation.
Positioning	and pad pressure points. eyes.	Head turned to the right with neck extension
	Asystole	Rarely (<1%), the SA node is innervated by the left vagus nerve. In those patients, vagal

Complications	Bronchospasm Sz Vascular injury	stimulation may result in severe bradycardia or asystole. Rx: stop stimulation; atropine 0.5 mg iv ± CPR. Bronchospasm may occur as a result at vagal nerve stimulation.

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Postoperative

Complications	Seizure Vocal cord paralysis Hoarseness Peritracheal hematoma	Sz precautions may be necessary. Monitor carefully for altered mental status. Airway obstruction may occur.
Pain management	Usually mild discomfort	Rx: See p. C-2.
Tests	No routine tests	

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Epilepsy Surgery

Surgical Considerations

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Gary Heit

Description: In the United States, the prevalence of epilepsy is 5–20/1,000 (0.5–2%), meaning that at least 1.5 million people have epilepsy. In childhood, the incidence and prevalence is higher, with 90% of all new cases occurring before the age of 20. Intractable epilepsy is defined as persistent seizure activity of such frequency or severity that prevents normal function and/or development. This diagnosis is made only after an adequate trial of anticonvulsant medication(s), with therapeutic levels, has been documented. Of all those with epilepsy, 10–20% prove to be intractable; it is estimated that 20–30% of patients with intractable epilepsy may benefit from a surgical procedure.

The causes of epilepsy are varied, ranging from idiopathic to neoplastic. Epilepsy surgery is most beneficial in patients with partial epilepsy 2° a structural lesion. Most commonly, this lesion is located in the temporal lobe, and the most common operation is a **temporal lobectomy,** in both children and adults. Cerebral dominance and, hence, the location of speech, must be determined using a preop Wada test (intracarotid amobarbital injection to localize language function), although functional MRI mapping of speech centers may eventually replace this invasive test. Studies to define the epileptogenic focus include simultaneous recordings of

video/EEG and high-resolution MRI and PET scans. Temporal lobe surgery may involve removal of only the structural lesion and associated epileptogenic cortex, cortical resection alone, excision of the amygdala and hippocampus, or removal of the entire anterior temporal lobe, with the extent of posterior resection dependent on dominance. Depending on the involved center, intraop electrocorticography may be used, requiring neurolept anesthesia (tranquilizer, opiate and N_2O). In addition, the speech center may need to be identified intraop, necessitating an awake procedure. These differing options will significantly alter the choice of anesthesia and must be established before surgery.

For a standard **temporal lobectomy,** the patient is placed supine on the operating table with the head turned 90° and held with pin fixation. A "question mark" temporal incision is often used, and hemostasis is achieved with skin clips. *(Print pagebreak 82)* A flap—either a free temporal bone flap or an osteoplastic flap, based on the temporalis muscle—is elevated with a high-speed craniotome. A **subtemporal craniectomy** allows visualization of the entire anterior temporal lobe. The dura is opened, widely exposing the anterior 6–6.5 cm of the temporal lobe. Labbe's vein must be preserved. At this point, surface and/or depth electrocorticography may be employed and **inhalation anesthetics must not be used.** After mapping the lesion, amygdala and hippocampus or anterior temporal lobe is removed. Temporal lobectomy involves resection of both the lateral and medial temporal structures, and is commonly performed in two steps. Often an operating microscope will be used to completely resect medial structures, including the uncus and hippocampal formation. Injury to the brain stem, 3rd and 4th cranial nerves, and either the middle cerebral or posterior cerebral arteries can occur; these are known complications of this surgery. Closure of the dura, bone flap, and scalp concludes the surgery.

Variant procedure or approaches: There are four common variant procedures. The first is sectioning of the corpus callosum, known as a **corpus callosotomy.** This is commonly used for patients with atonic seizures or partial seizures with secondary generalization. Either the anterior two-thirds or the entire corpus callosum is divided in the midline. The approach is the same as any transcallosal, intraventricular procedure, and uses a bifrontal, paramedian scalp incision and elevation of free-bone flap adjacent to the midline in the region of the coronal suture. Injury to the sagittal sinus is possible and may result in massive VAE or hemorrhage. In addition, numerous bridging veins across the interhemispheric fissure must be preserved to avoid venous congestion and possible infarction. The right cerebral hemisphere is gently retracted from the falx, exposing the paired anterior cerebral arteries and underlying corpus callosum. If an anterior two-thirds transection is performed, an intraop x-ray is required to determine the posterior border.

The second variant involves either a **frontal, temporal,** or **occipital craniotomy** for resection of a structural, epileptogenic focus, such as a tumor or AVM. This procedure may use **stereotaxic localization** and the resultant craniotomy may be performed with image guidance, or in a stereotaxic head frame, which affects the method of intubation. The subsequent craniotomy is similar to the excision of any structural lesion, with the exception of intraop electrocorticography of surrounding cortex, which if used will affect the choice of anesthetic.

The third variant is most common and consists of a diagnostic procedure involving placement of **surface and/or depth electrodes.** This may be performed with or without stereotaxic localization. Often only burr holes, outlining the future craniotomy flap, are used. After placement, the electrodes are externalized, and postop the patient's naturally occurring Sz are recorded, in conjunction with video monitoring, to register the clinical presentation with the onset of ictal activity. This recording/observation period may last for several days. The patient is then returned to the OR and the epileptogenic focus resected as described earlier.

The fourth alternative is **selective amygdalohippocampectomy,** a variation of the standard anterior temporal lobectomy. In this procedure, the surgeon makes a cortical incision in the anterior temporal lobe and exposes and resects the amygdala and hippocampus, sparing the remaining portions of the temporal lobe. This procedure is sometimes used on the dominant side of the brain in an effort to lower the risk of postop speech and language dysfunction.

Usual preop diagnosis: Temporal lobe epilepsy; partial epilepsy; intractable epilepsy

Summary of Procedures

Position	Supine, rarely prone for occipital lesions; table turned 180°
Incision	Temporal question mark, reverse question mark, paramedian, frontal, or occipital
Special instrumentation	Operating microscope; Cavitron; bipolar cautery; surface electrode grids and strips; depth electrode
Unique considerations	Electrocorticography requiring neuroleptic anesthesia; awake procedures for mapping of temporal and/or frontal speech areas; stereotaxic craniotomy and lesionectomy

Antibiotics	Ceftriaxone 1 g iv (avoid administration with calcium-containing solutions)
Surgical time	3 h
EBL	Minimal for diagnostic procedures; 250–500 mL with craniotomy (adults)
Postop care	After craniotomy, ICU for 12–24 h
Mortality	<1%
Morbidity	Hemiplegia Dysphasia Ophthalmoplegia Brain stem injury
Pain score	2–4

Patient Population Characteristics

Age range	New onset Sz: pediatric and geriatric populations
Male:Female	1:1
Incidence	180,000/yr (new cases of epilepsy): 10% eventually present for surgery/yr
Etiology	Idiopathic (mesial temporal sclerosis); infectious (brain abscess, encephalitis); traumatic (glial scar); vascular (AVM, infarct); neoplastic (glioma, hamartoma, ganglioglioma); congenital (cortical dysplasia)
Associated conditions	Tuberous sclerosis; Sturge-Weber syndrome; infantile hemiplegia; encephalitis; hemimegalencephaly

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Anesthetic Considerations

Preoperative

Epilepsy is a common neurologic disorder among young adults. Antiepileptic medication, such as phenytoin, will abolish seizure disorders in most patients, but some develop intolerable side effects to such medications; others are refractory to medical therapy. Surgical ablation of the seizure focus may be the only effective therapy for these patients if they are to become self-sufficient. Several operations may be done, the most common being placement of surface or depth electrodes to determine the focus of the Sz, with subsequent temporal lobectomy for removal of the focus. In some cases, the lesion may be very focal and amenable to stereotactic localization and removal. At some centers both seizure mapping and subsequent resection are carried out during the course of an awake craniotomy (see p. 34). If wake-up testing is planned, the procedure should be explained to the patient in detail including what the patient should expect to hear and feel during the test.

Neurological	Usually the only neurological finding is a Hx of uncontrollable seizures, either focal or generalized. Obtain a description of Sz and prodromal Sx. Sz medications should be continued through the morning of surgery. **Tests:** A preop Wada test (intracarotid injection of sodium amytal) and/or fMRI mapping is performed to determine whether the area of proposed surgery has any cerebral dominance or speech function.
Gastrointestinal	Abnormal liver function may be associated with valproate and carbamazepine use. **Tests:** LFT and others as indicated from H&P.

Hematologic	Phenytoin/phenobarbital →↓ Hct; Carbamazepine/valproate/ethosuximide/primidone →↓ Plt; Carbamazepine/primidone →↓ WBC **Tests:** CBC and others as indicated from H&P.
Laboratory	Tests as indicated from H&P.
Premedication	Standard premedication (see p. B-1) is usually appropriate.

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Intraoperative

Anesthetic technique: GETA (± wake-up testing) or local anesthesia (awake craniotomy). The placement of surface or depth electrodes is done under GETA, as is a temporal lobectomy in the nondominant hemisphere. If the seizure focus is in the dominant hemisphere and/or if there is any question about possible injury to speech or motor areas, the procedure may be performed under local anesthesia (awake craniotomy, see p. 34), with intraop localization of the seizure focus.

Induction	Standard induction (see p. B-2). If a difficult intubation is anticipated (e.g., stereotactic frame), orotracheal intubation is best accomplished before induction of GA. An awake fiber optic intubation is the best technique (see p. B-5). After anesthesia is induced, a nondepolarizing neuromuscular relaxant is administered.
Maintenance	Standard maintenance (see p. B-2). Generally, no further NMBs are administered beyond that used for tracheal intubation. With adequate anesthesia, and the head fixed in the Mayfield-Kees skeletal fixation, patient movement of any consequence is highly unlikely. Furthermore, it is useful to see movement of an extremity as an indicator of inadequate depth of anesthesia.
Emergence	No special considerations. Prophylactic antiemetic (e.g., ondansetron 4 mg iv, ±metoclopramide 10–20 mg iv) should be given 30 min before extubation. Surgeon should infiltrate wound with 0.5% bupivacaine (max = 30 mL) or 0.75% ropivacaine (max = 40 mL) with epinephrine to improve post-op analgesia.

Wake-up testing: At some institutions, an asleep-awake-asleep technique is used in which the patient is placed under GETA for positioning and craniotomy. After surgical exposure of the seizure area, the patient is allowed to awaken to assess neurologic function while areas of the brain are stimulated. When the seizure focus has been adequately delineated, GA is reinstituted for the remainder of the operation. An excellent anesthetic technique under these circumstances includes the use of sevoflurane (± 50% N_2O) for amnesia, and remifentanil (0.05–2 mcg/kg/min) by continuous infusion for analgesia. The advantage of these drugs is that they are quickly eliminated, allowing for rapid emergence for the awake component of the procedure, followed by rapid reinduction of anesthesia when the testing period is over. To allow the patient to talk during the awake portion of the procedure, the ETT must be removed. This is best accomplished by inserting a small or medium-sized tube changer through the ETT before it is removed. The tube changer can then be used as a guide for reinsertion of the ETT, and it will not prevent normal vocalization by the patient during the awake phase. Alternatively, an LMA can be used to secure the airway initially, removed for the awake testing and reinserted for closure.

Blood and fluid requirements	Moderate blood loss IV: 18 ga ×2 NS @ 4–6 mL/kg/h	Two iv cannulae are useful: one for fluid administration, another for infusion of anesthetic and other drugs. Alternatively, one peripheral iv and a CVP cannula are inserted. Blood transfusions are seldom needed.
Monitoring	Standard monitors (see p. B-1). Arterial line ± CVP line UO	O_2sat and $ETCO_2$monitoring will indicate the adequacy of ventilation and oxygenation in awake patients.

Positioning	Table rotated 180° and pad pressure points eyes	Semisitting position with head held in Mayfield-Kees 3-pin clamp and rotated laterally with a roll under the shoulder on the operative side. Anesthetic hoses and intravascular lines must be long enough to be accessible.
Complications	Anxiety Agitation Sz	Local anesthetic toxicity may produce agitation and seizures. Iced NS should be immediately available to flood cortical surface for Sz control.

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Postoperative

Complications	Seizure Bleeding Cerebral edema	Monitor carefully for altered mental status. May require urgent reexploration and removal of electrode grid if present.
Pain management	Codeine 30–60 mg im q 4 h PCA (selected patients)	Avoid oversedation. LA infiltration of the incision effectively reduces postop pain. Morphine PCA (e.g., 1.5 mg, 8 min lockout, 40 mg/4 h max) combined with ondansetron 4 mg iv q 4 h
Tests	CT scan	If a patient exhibits any delay in emergence from anesthesia and surgery, or any new neurologic deficits appear, a CT scan is invariably obtained.

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Surgery for Spasticity

Surgical Considerations

Description: Neurologic conditions associated with spasticity of the extremities include spinal cord injury, multiple sclerosis, stroke, etc. The spasticity is often managed with oral medications. An alternative approach is to infuse baclofen (GABA-agonist that decreases frequency and amplitude of tonic impulses to the muscle spindles) into the subarachnoid space via an implanted pump. When these therapeutic modalities fail, it may be necessary to perform surgery. Some of the procedures are destructive in that a lesion is placed in certain nerves or the spinal cord to destroy the reflex arc that is contributing to the spasticity. There are both percutaneous and open techniques for placing the lesion. In the **open procedures,** a **laminectomy** is performed (see Posterior Lumbar Spine Surgery, p. 119). In a case where a **myelotomy** is to be performed, the lower spinal cord is exposed and incised at the appropriate location to interrupt the reflex arc. In certain cases, selective electrical stimulation can be performed on isolated dorsal rootlets from the involved extremity. Abnormal responses in the extremities are monitored via EMG and direct observation. When abnormal responses are detected, that particular rootlet is divided (**dorsal rhizotomy**). This procedure is somewhat tedious and requires that stable anesthetic conditions be maintained so that appropriate monitoring can be carried out during the procedure. The surgeon usually must sacrifice 40–60% of the dorsal rootlets in cases of spastic diplegia found in cerebral palsy.

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Variant procedure or approaches: Percutaneous **RF rhizotomy** is another procedure used for spasticity. A thermal lesion is placed in the appropriate dorsal roots with a RF generator. Needles are passed into the neural foramen via a posterolateral trajectory for levels L1–L5, and then from a midline approach to the S1 root. The needle position is determined by A-P and lateral fluoroscopy and by stimulus mapping. Stimulating current is delivered via an electrode passed through the needle. A low-level stimulating current causes muscle twitching in the appropriate leg if the needle is in the proper location. Once placed and verified, the RF generator is used to produce the lesion. At the termination of this procedure, the patient's legs should be flaccid. Patients with spinal cord injury producing anesthesia below T10 may not require additional anesthetic for the procedure.

Usual preop diagnosis: Spasticity; multiple sclerosis; spinal cord injury

Summary of Procedures

	Open Rhizotomy or Myelotomy	RF Rhizotomy
Position	Prone	
Incision	Midline	Needles placed in lumbar region
Special instrumentation	EMG monitor; nerve stimulator	RF generator; fluoroscope
Unique considerations	Anesthetic that allows EMG recordings	May not require anesthesia if anesthetic below waist (2° spinal cord surgery).
Antibiotics	Ceftriaxone 1 g iv (avoid administration with calcium-containing solutions)	None
Surgical time	4 h	2 h
Closing considerations	Surgeon may wish to test dural closure with Valsalva maneuver.	
EBL	50–250 mL	Negligible
Postop care	Head flat; PACU room	
Mortality	<1%	
Morbidity	All <5%: CSF leak Infection Hemorrhage Neurological impairment	
Pain score	4	2

Patient Population Characteristics

Age range	3–55 yr

Male:Female	3:2
Incidence	Relatively uncommon neurosurgical procedure
Etiology	Hyperactivity of gamma stretch reflex
Associated conditions	Multiple sclerosis; cerebral palsy; spinal cord injury

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Anesthetic Considerations

See Anesthetic Considerations for Surgical Correction of Spinal Dysraphism, Pediatric Neurosurgery, p. 1162.

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Percutaneous Procedures for Trigeminal Neuralgia

Surgical Considerations

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Description: Three percutaneous procedures are commonly used to treat trigeminal neuralgia (a well-defined pain disorder of the face). Each involves placing a needle percutaneously from the cheek into the foramen ovale at the base of the skull under a light iv anesthetic. For **glycerol injection,** it is necessary for the surgeon to verify that the needle is placed in the cistern of the trigeminal (AKA Gasserian) ganglion. This usually is done via I.I. or x-ray films with contrast instilled into the cistern by the surgeon. There should be free flow of CSF through the needle. The patient is then placed in the seated position with head flexed and sterile glycerol is injected into the cistern (a potentially painful event). The patient is taken to the recovery room with the head still flexed for 1 h. The glycerol damages neurons in the ganglion, which usually causes mild sensory loss and relieves the tic pain in most cases. **Percutaneous balloon compression** of the ganglion is a procedure done in some centers for this condition. The needle is placed similarly to the previously mentioned procedure, but, in this case, a balloon catheter is placed through the needle, and the balloon is inflated after it is successfully located in the Gasserian ganglion cistern. The patient may be kept sedated throughout this procedure. Compression of the ganglion will relieve the pain in many patients.

Radiofrequency (RF) rhizotomy is the most common percutaneous procedure used for trigeminal neuralgia. This procedure differs from glycerol injection in that a RF generator is used to place a thermal lesion in the appropriate portion of the Gasserian ganglion. The needle is actually an insulated electrode with a portion of the tip exposed. The proper needle position is determined by applying stimulating current, with the patient awake, while assessing patient's responses. Multiple brief periods of anesthesia will be required to adjust the needle position or to lesion the nerve. It is important for the patient to awaken quickly and be able to cooperate with the stimulus localization throughout this procedure. This same approach may be used to place a stimulating electrode in the ganglion with the leads subsequently tunnelled to an implantable pulse generator. Direct stimulation has been shown to provide pain relief in many patients.

Usual preop diagnosis: Trigeminal neuralgia; tic douloureux

Summary of Procedures

	Glycerol Injection	Balloon Compression	RF Rhizotomy
Position	Supine		
Incision	Needle placed lateral to mouth on cheek		
Special instrumentation	I.I.		+ RF generator
Unique considerations	Hypertensive response to needle placement		+ Requirement for periodic deep sedation with rapid awakening for RF lesioning.
Antibiotics	Usually none		
Surgical time	0.5 h	1 h	1–1.5 h
EBL	None		
Postop care	Seated position with head flexed for 1 h	PACU	PACU
Mortality	1%		
Morbidity	Usually < 5%: Infection Complete facial numbness (anesthesia dolorosa) Extraocular muscle paresis (diplopia) CSF leak Carotid puncture Facial hematoma		
Pain score	3–7	2–4	2–4

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Patient Population Characteristics

Age range	40–85 yr (usually 60–70 yr)
Male:Female	1:2
Incidence	Trigeminal neuralgia: 4.3/100,000/yr [25/100,000/yr > 70 yrs]
Etiology	Vascular compression of trigeminal nerve; multiple sclerosis plaque; cerebellopontine angle tumor; AVMs
Associated conditions	HTN; multiple sclerosis; rheumatoid arthritis; Charcot-Marie-Tooth disease; Sjogren syndrome

Anesthetic Considerations

Preoperative

Trigeminal neuralgia, or tic douloureux, is a condition that develops in adults usually > 60 yr old. It is more common in women in whom an intermittent, severe, lancinating pain arises over the maxillary and/or mandibular divisions of the trigeminal nerve. The ophthalmic division of the trigeminal nerve is rarely involved. The pain is unilateral and often can be precipitated by stimulating a trigger point, such as by rubbing the cheek, mastication, or brushing the teeth. The cause of this condition is not known. Medical management consists of therapy with carbamazepine (Tegretol) or oxcarbazepine (Trileptal), anticonvulsants, and analgesics specific for this condition. The failure rate for medical treatment is 50–60%. Stereotactic radiosurgery (dose = 60 Gy) has been shown to have long-term effectiveness in 50% of patients who failed drug therapy. Surgery is considered when medical or radiotherapy management fails to control pain or complications of drug therapy develop (anemia, bleeding disorders, dizziness, etc.). One of three types of percutaneous procedures is performed: either **glycerol injection, RF rhizotomy** of the symptomatic branches of the trigeminal ganglion or direct ganglionic stimulation. If these treatments fail, microvascular decompression of the trigeminal nerve is considered.

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Respiratory	No common associations or special considerations, unless patient has a longstanding Hx of smoking and has COPD. **Tests:** As indicated from H&P.
Cardiovascular	Most patients have Hx of idiopathic HTN and take antihypertensive medications. Good control of BP preop is important because most patients become hypertensive during the operative procedure. Intraop HTN is often unavoidable because of the surgical need to have the patient awake during much of the procedure. **Tests:** As indicated by H&P.
Neurological	The presenting symptom is pain in the maxillary and/or mandibular division of the trigeminal nerve, unaccompanied by motor or sensory deficits. (Dx should be questioned in the presence of sensory deficits).
Laboratory	As indicated from H&P. CBC/platelets if Rx carbamazepine. Anticonvulsant drug levels.
Premedication	Midazolam (1–2 mg iv) ± glycopyrrolate 0.2 mg iv shortly before induction of anesthesia to minimize oral secretions while the surgeon is working in the mouth, positioning the needle.

Intraoperative

Anesthetic technique: GA, regardless of which percutaneous technique is to be used. O_2 by nasal cannula should be administered before induction of anesthesia. To keep the cannula out of the surgeon's field, it must be taped above the eye on the side of operation.

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Induction	Because the surgeon will want the patient awake to check for pain distribution as soon as the needle or electrode is in place, the induction drug must be potent, but short-acting. Methohexital (0.5–1 mg/kg) is preferred for this purpose, but propofol (1–2 mg/kg) alone or at a reduced dose in combination with remifentanil (0.5 mcg/kg) has been used successfully. The drug should be injected by bolus into a rapidly flowing iv to achieve a high concentration in the brain quickly. Continuous infusion of the drugs is not satisfactory, because it fails to achieve a high brain concentration quickly, and its continuous administration prolongs the time before the patient is sufficiently able to communicate with the surgeon. In experienced hands, the needle is placed within a matter of 2–3 min. If difficulty is encountered in placing the needle, additional bolus doses of induction drug may be needed. Patients often develop apnea for 1–2 min, followed by partial or total airway obstruction, while the surgeon has his hand in the mouth positioning the needle. Prior to induction of anesthesia, therefore, it is essential that the anesthesiologist optimize ventilation and oxygenation by asking the patient to take a series of deep breaths through the nose with the mouth closed. This will ↑ the O_2 level and ↓ the CO_2 level in the lungs before induction. To maintain adequate spontaneous ventilation and oxygenation, it is usually necessary to institute forward displacement of the mandible while the surgeon inserts the needle. Another alternative is to insert a nasal airway. If **glycerol injection** is used, needle position is verified by radiological imaging, using a radio-opaque dye. Patient is awakened and placed in a seated position with head flexed. The

		glycerol is injected, causing severe pain. Patient is then moved to recovery room still in seated position with the head forward to keep the glycerol localized to the region of the trigeminal ganglion. If **RF rhizotomy** stimulator electrode placement is performed, the patient is awakened and the position of the electrode is verified by stimulating the ganglion with heat or directly and determining the site of pain. The patient is then reanesthetized with a smaller bolus of the same drug to permit electrode adjustment or local heating for 1 min. This procedure may be repeated several times. Each time, the patient needs less anesthetic, because of both the cumulative effects of the drug, and the fact that the ganglion is becoming damaged by the heat and manipulation.
Emergence		Following glycerol injection patients are maintained in a seated position with an ice pack on the cheek at the site of needle insertion to minimize postop bleeding and swelling. These patients often complain of pain in the face, requiring opiate analgesics. Following RF rhizotomy, the face is usually numb, which is the end point for concluding the operation.
Blood and fluid requirements	IV: 18–20 ga ×1 NS/LR @ 4–6 mL/kg/h	
Monitoring	Standard monitors (see p. B-1).	HTN patients may require an arterial line.
Control of BP	Labetalol Clonidine patch SNP infusion may be necessary	Patients almost invariably become hypertensive during this therapy. Attempts to lessen these episodes with a clonidine patch preop or use of intermittent doses of labetalol prophylactically or therapeutically are only partially successful.
Positioning	Table turned 180° and pad pressure points. eyes.	Patient in reclining position with head in the midline.
Complications	Failure of needle placement Bleeding Respiratory arrest Oculocardiac reflex	Major complications from this operation are uncommon, but include: (1) Failure to identify the foramen ovale, through which the needle must be inserted to reach the trigeminal ganglion. If the needle cannot be placed within 30–40 min, the procedure usually is aborted until another day. (2) Bleeding into cheek from puncture of a branch of the facial artery. (3) Apnea from spillover of the neurolytic solution into circulating CSF, presumably affecting the respiratory center in the 4th ventricle.

Postoperative

Pain management	Parenteral opiates (see p. C-2).

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