

E.2. Diagnosis and Management of Surgically Treatable Pain Problems, Movement Disorders, and Epilepsy

a. Recognizing the Features of Trigeminal and Glossopharyngeal Neuralgia, Causalgia and Cancer Pain, Indications for Surgical Referral and Spectrum of Surgical Therapeutic Options.

i. Trigeminal Neuralgia:

Trigeminal neuralgia, also known as tic douloureux, is a sudden lancinating pain usually lasting for a few seconds that is confined to the distribution of one or more of the branches of the trigeminal nerve on one side of the face. This pain is often triggered by sensory stimulus. There is no neurologic deficit associated with this pain. The patient will oftentimes have a trigger point or position somewhere within the trigeminal nerve distribution that stimulates this pain. Facial pain that does not fit into this classification is usually referred to as atypical facial pain. A condition known as status trigeminus exists when the trigeminal neuralgia-like pain occurs as tic-like spasms in rapid succession.

The disease is not uncommon, with an incidence of approximately 4 per 100,000 people. Occasionally, there is an association with MS, with 18% of patients with bilateral trigeminal neuralgia having MS and 2% of patients with multiple sclerosis showing evidence of trigeminal neuralgia.

The exact pathophysiologic cause of trigeminal neuralgia is not known, but it is probably likely due to irritation or compression of the trigeminal nerve, particularly its large diameter myelinated A type fibers. This causes an abnormal transmission of sensory stimuli through the poorly myelinated A delta and C type pain fibers. Common causes include vascular compression of the trigeminal nerve at the root entry zone, posterior fossa tumor with compression of the nerve, or a multiple sclerosis plaque within the brain stem. Nearly half of autopsy specimens will show evidence of compression of the trigeminal nerve root entry zone in patients who have never had symptoms of trigeminal neuralgia, whereas nearly all patients with trigeminal neuralgia will show this type of compression. Most commonly the superior cerebellar artery is the culprit causing compression but other posterior fossa vessels may also cause compression. Patients with tumors can also show compression either of the nerve or the entry zone.

In order to definitively recognize trigeminal neuralgia, one rule out other causes of facial pain. Five other major causes must be considered in the differential diagnosis of trigeminal neuralgia including dental disease, disease in the orbit, temporal arteritis, tumor, and herpes zoster. In particular, herpes zoster pain tends to be continuous, rather than sudden and lancinating, and is accompanied by other signs. These include characteristic vesicles and crusting that usually follow the pain and pain noted in the distribution of the first division of the trigeminal nerve (V1). To delineate these causes, one must perform a detailed history and physical examination. This includes an accurate description of the distribution of the pain as well as the quality of the pain. The division or divisions of the trigeminal nerve that are involved should be characterized. Trigeminal neuralgia pain is characteristically paroxysmal (sudden) pain interrupted by pain free intervals. If pain is always present, then it is unlikely for the pain to be trigeminal neuralgia. If

the patient has been tried on medications, a full description of the dosages that have been used, the duration of their use, and any side effects must be noted.

On physical exam the patient should have a normal neurologic exam unless the patient has undergone surgery in the past which may have caused some neurologic deficit. If a neurologic deficit is present then one should look instead

For a brain tumor or other brain lesion as a cause of the abnormality. A simple series of tests to look at trigeminal nerve function include testing the corneal reflex, testing facial sensation in all three divisions of the trigeminal nerve, and testing the patient's bite (which assesses the motor function through the motor branch of the trigeminal nerve). To rule out orbital disease, one should assess extraocular movement function.

It is important to differentiate true trigeminal neuralgia pain and pain that is induced by a tumor, which is usually characterized by atypical facial pain. One of the atypical features of tumor-related pain is that the pain is usually constant and neurologic abnormalities are often present, usually of sensory type. True trigeminal neuralgia patients are typically older individuals with whereas tumor patients can be of significantly younger age.

Trigeminal neuralgia is usually treated by medications. In fact, the most common medication, Carbamazepine (Tegretol), will give acceptable relief in 69% of patients. Patients on this medication should be checked for a relative leukopenia. Baclofen and gabapentin can be used if these medications are not effective. Other medications can be tried including phenytoin and clonazepam but if these medications show poor benefit for the patient surgical therapy is considered.

Surgical referral is recommended for patients that are refractory to medical management or when the side effects of the medications exceed the risks and the drawbacks that would be noted for surgery.

Surgical methods for the treatment of trigeminal pain are numerous and deal with different portions of the trigeminal nerve.

aa. Extracranial Procedures

□ One can attempt to block the transmission of the pain signal using a percutaneous trigeminal rhizotomy. The object of this procedure is to attempt a selective destruction of the a delta and c fibers which mediate nociception and to preserve the a alpha and beta fibers which mediate touch and other sensory parameters. The techniques most widely utilized include radiofrequency thermocoagulation, glycerol injection into Meckel's cave, and percutaneous microcompression via inflation of a Fogarty catheter balloon.

□ Rarely, peripheral nerve ablation or neurectomy may be performed. These procedures are usually reserved for elderly patients whose remaining life span may not exceed the time to recurrence of the pain. This recurrence of pain is secondary to nerve regeneration and occurs within 18-36 months, but it may be followed by a repeat of that procedure if necessary.

bb. Intracranial Procedures

□ One common procedure is the microvascular decompression (MVD), in which a microsurgical exploration of the root entry zone is performed via a craniectomy of the posterior fossa bone. The vessel that is impinging on the nerve

is displaced using a nonabsorbable insulator such as Teflon felt or Ivalon sponge to absorb the pulsations of the vessel impinging on the nerve.

□ A newer, less invasive means of treatment is stereotactic radiosurgery. In this treatment, a small collimator size (4 mm) is used to place a 70-90 Gy lesioning dose on the portion of the trigeminal nerve as it enters the brain stem in the root entry zone. All efforts are made to keep the 80% isodose curve outside of the brain stem.

□ Infrequently, an extradural subtemporal approach to section the trigeminal nerve or an open intradural exposure of the nerve followed by sectioning may be performed.

ii. Glossopharyngeal Neuralgia

Glossopharyngeal Neuralgia is a much less common form of pain compared to trigeminal neuralgia but is also a severe lancinating type of pain. The pain is in the distribution of the glossopharyngeal and vagus nerves and includes pain radiating to the throat and the base of the tongue, occasionally to the ear, as well as to the neck. Sometimes this pain is associated with coughing or salivation. In rare instances the patient may have evidence of cardiac arrest, convulsions, syncope, or hypertension. The pain may be triggered by talking, chewing, and swallowing, all tasks that utilize the IXth and Xth cranial nerve reflex arcs.

One may try to treat initial pain using cocaine over the tonsillar pillars and fossa but usually patients with glossopharyngeal neuralgia will require surgical treatment. Surgical treatment includes either microvascular decompression or sectioning of the glossopharyngeal nerve via either an extra or intracranial approach. The intracranial approach may provide better results. In the intracranial approach, the patient will undergo sectioning of all of the preganglionic glossopharyngeal nerve fibers as well as the upper one-third or two fibers (whichever is larger) of the vagus nerve. Occasionally patients may have problems either with their cardiovascular system or with their swallowing and therefore require monitoring, particularly over the first 24 to 48 hours in order to treat any vagus nerve complication. Unlike trigeminal neuralgia, medications do not appear to be effective and this particular pain syndrome seems to require surgical management for long term benefit.

iii. Causalgia

Causalgia is also known as reflex sympathetic dystrophy and, more recently, as complex regional pain syndrome (CRPS). Patients with complex regional pain syndrome (CRPS) usually show some manifestation of a partial peripheral nerve injury including autonomic dysfunction, severe burning or gnawing type of pain, and trophic changes in the involved extremity. Previously the pain had been divided into minor and major causalgia forms, the minor form usually occurring after nonpenetrating trauma and major causalgia occurring after high velocity missile injuries in victims of war.

Theories regarding the pathogenesis of complex regional pain syndrome initially invoked ideas of electrical transmission between sympathetic nerves and afferent pain fibers. More recently it has been postulated that norepinephrine released at

sympathetic terminals, together with hypersensitivity secondary to denervation or sprouting, induces the complex regional pain syndrome.

Most commonly, patients present with burning pain in a single limb, most prominent in the distal extent of that extremity. Most patients will show the onset within 24 hours or less of the actual time of injury though it may take days or weeks to develop. Allodynia is often present and is defined as pain induced by a non-noxious stimulus. In patients with CRPS, allodynia is a common finding.

On examination, patients may show either vasodilatory findings, with a warm and pink extremity, or vasoconstrictor findings, with a cold, mottled, blue skin change, along with their other neurologic findings. The patient may show trophic changes including stiff joints, tapering fingernails, long coarse hair or loss of hair, dry scaly skin, and alterations in their sweating ranging from anhidrosis to hyperhidrosis.

There are no good tests at this time that will confirm or exclude the diagnosis of CRPS. In order to assess improvement one can only assess the subjective impression of improvement by the patient. Medications have been tried but are largely ineffective. These medications include the use of tricyclic antidepressants as well as pain medications. One-fourth or less of patients will show satisfactory long lasting relief after a series of sympathetic blocks. Surgical sympathectomy, usually involving an exposure (if the pain is in the upper extremity) to the upper portion of the thoracic chain has, in some studies, been shown to significantly relieve pain. Spinal cord stimulation, which is the most recent treatment being utilized, also seems to have some success in treating causalgia. Its effectiveness is usually tested preoperatively by a spinal cord stimulator trial. A needle is utilized for percutaneous placement of a lead electrode over the spinal cord. It is placed in the cervicothoracic area for the upper extremity symptoms and in the thoracolumbar region (most commonly the lower thoracic area) lower extremity symptoms.

iv. Cancer Pain

Cancer pain may be extremely difficult to treat. Many malignant tumors may cause pain, particularly if the cancer itself is localized peripherally. Cancer within the CNS, particularly the brain and spinal cord, tends not to show pain symptomatology except for headache, and can be treated with other means.

Patients with cancer pain are usually referred when opioid (narcotic) medication management has been found to be ineffective. These patients may undergo a trial of either an IV narcotic pain medication regimen or, more recently, a morphine pump infusion. The intrathecal morphine pump utilizes a subcutaneously placed pump, usually in the abdominal region, which slowly and continuously instills the narcotic within the CSF, usually into the lumbar cistern. This has been found to be quite effective in many types of cancer pain and has become a widely used treatment option. More aggressive and invasive surgical techniques may also be utilized but have found significantly greater disfavor with the advent of the intrathecal morphine pump. These other procedures include cordotomy (either open or percutaneous), dorsal root entry zone lesioning, and commissural myelotomy to treat bilateral pain. One may also consider deep brain stimulation in the periaqueductal or periventricular gray matter for treatment of cancer pain.

Medial thalamotomy has been tried but is controversial. Stereotactic mesial encephalotomy has even been utilized for head, neck, facial, and upper extremity pain. All of these more invasive techniques have found much less favor since the introduction of intrathecal morphine pump treatment.

b. Recognize movement disorders amenable to surgical intervention, including Parkinson's disease, dystonia, spasticity and hemifacial spasm, indications for surgical referral and the spectrum of surgical therapeutic options

i. Parkinson's disease

Parkinson's disease is characterized by a triad of bradykinesia or akinesia, tremor, and cogwheel rigidity. Prior to the early 1950's the only treatment that showed some benefit was ligation of the anterior choroidal artery. The results were quite variable, however. It was soon discovered that most patients with favorable outcomes had lesions that included the globus pallidus. Because of this, techniques for lesioning this site developed and this lesioning provided benefit. Initially, anterior and dorsal pallidotomy became the accepted procedure. This mainly improved rigidity but had minimal effect on tremor or bradykinesia. The thalamus then became a lesion target, particularly the ventral lateral thalamus. This site of treatment provided marked improvement in tremor on the contralateral side. It could not be performed bilaterally because of an unacceptably high incidence of complications with the bilateral procedure, including dysarthria and gait disturbance, as well as an unacceptable level of memory disturbance.

When L-dopa was introduced in the late 1960's, surgical treatment for Parkinson's disease diminished significantly. However, in the early 1990's a resurgence of surgical treatment occurred because it was found that medications only provided benefit for several years. Tissue transplantation developed as one method of treatment (e.g. with adrenal medullary tissue or fetal tissue), but showed only modest benefits. Lesioning procedures, including the posterior ventral lateral pallidotomy, were later re-explored. They showed significant benefit in end-stage patients and became the accepted form of surgical treatment in the early and mid-1990's. Patients showed some improvement, particularly in the dyskinesias or abnormal movements induced by their medication, as well as some improvement in their rigidity and tremor. Much less improvement was seen in bradykinesia, akinesia, or gait disturbance.

More recently, deep brain stimulation has been utilized as a treatment for Parkinson's disease. Criteria for referral include Parkinson's disease symptoms that do not improve with maximal medical treatment or if unacceptable side effects result when control is obtained. Usually patients will do well with medical treatment for the first several years but require surgical treatment to provide improvement after that period. It is possible that patients should be referred earlier for surgical treatment because of preliminary evidence that deep brain stimulation performed bilaterally may actually reduce the severity, progression, and course of the disease.

Deep brain stimulation for Parkinson's disease is almost always now directed at the subthalamic nucleus. The procedure may be performed either by staged

unilateral procedures or occasionally as a bilateral procedure. The staged unilateral procedure appears to have lower risk for the patient, particularly in postoperative recovery, but do require a second operative setting. The bilateral procedure has the advantage of combining the surgical treatments all in a single day but patients may show some difficulty postoperatively because of the micro-lesioning effect obtained bilaterally at the time of implantation.

ii. Dystonia

Treatment for dystonia has mainly evolved from treatment already developed for Parkinson's disease. Dystonia, however, differs in its clinical presentation from Parkinson's disease. Very rigid muscle contraction or spasm in multiple muscle groups creates a distorted posture. Many patients with dystonia will note severe pain secondary to the dystonic muscle contractions. Preliminary evidence suggests that pallidotomy procedures may be of significant benefit. Additionally, current work is underway exploring deep brain stimulation directed at both the globus pallidus and the subthalamic nucleus. At present, however, there is no generally accepted surgical treatment for dystonia.

iii. Spasticity

Spasticity is an uninhibited reflex arc between alpha motor neurons and Ia afferents for muscle spindles resulting in a hypertonic state. Oftentimes patients will show clonus and may show involuntary movements as well. The lack of inhibition of the reflex arc results from a lesion in the upper motor neuron pathways causing absence of inhibitor influence on the motor neurons, including both the alpha motor neurons, as well as the gamma motor neurons (intrafusal fibers). Many types of CNS injury may cause spasticity, but the most common are cerebral injury (e.g. stroke), spinal cord injury (e.g. trauma to the spinal cord), congenital abnormalities (e.g. cerebral palsy or spinal dysraphism), and multiple sclerosis.

Spasticity is noted on physical examination by hyperactive muscle stretch reflexes, increased resistance to passive movement, and, occasionally, simultaneous activation of opposing antagonistic muscle groups. Patients oftentimes have pain accompanying this rigidity. In addition to causing pain, spasticity may disrupt activities of daily living by making the patient unable to sit in a wheelchair with or without special modification, or even lay comfortably in bed. Some patients will develop a spastic bladder with spontaneous emptying and low capacity. Ulcers may also develop because of characteristic postures that develop including scissoring of the legs or hyperflexion of the thighs. In cases of spasticity following spinal cord injury, the onset of the spasticity may be delayed anywhere from days to months after the initial injury, particularly in cases where spinal shock or hypotonia and hyporeflexia exist in the early period. Some level of spasticity may actually be helpful in these patients, usually to provide support when sitting in the wheelchair and to help in bracing the patient, but in other respects the spasticity is very limiting for the patient and requires medical and/or surgical treatment.

Spasticity is graded by a score known as the Ashworth score. This is a useful score graded on 1-5 with 1 showing no increase in tone and 5 showing the effected muscle as being rigid in flexion or extension.

Medical treatment for spasticity includes stretching, to prevent muscle and joint contractures, removal of inciting stimuli, and use of various medications. The most common medications utilized include baclofen which activates gaba-b receptors, diazepam, which activates gaba-a receptors, and dantrolene, which reduces calcium flux through calcium channels within the sarcoplasmic reticulum. Often, however, these medications have significant undesirable side effects culminating in the need for surgical treatment.

Patients are referred for surgical treatment when they are noted to be refractory to medical treatment or when the side effects of the medication become intolerable. Orthopedic procedures, including tendon releases and myotomies, are sometimes performed, particularly for fixed deformities. Neurosurgical interventions include nonablative procedures, such as the intrathecal baclofen pump, and ablative procedures, including injections, rhizotomies, neurolyses, and neurectomies. By far the most common procedures now utilized are the selective dorsal rhizotomy and the intrathecal baclofen pump.

Selective dorsal rhizotomy utilizes intraoperative EMG and electrophysiologic stimulation to evaluate the reflex arc involved in the spasticity. This procedure acts by interrupting the afferent component of the pathologic reflex arc. Particularly in children with cerebral palsy, this operation has been noted to produce improvement, both in ambulatory and nonambulatory patients. It usually does not provide much improvement in upper extremity, trunk, or head and neck-related spasticity.

The intrathecal baclofen pump, on the other hand, provides improvement in all of these respects. The device consists of an implanted pump that delivers an intrathecal infusion of baclofen through a catheter in the lumbar region. The dosage can be adjusted to maximize improvement in spasticity without reducing tone to such a degree that the patient is handicapped by hypotonia. Prior to placement of a baclofen pump a baclofen trial is performed in which incremental test doses of 50, 75, or 100 ugs of intrathecal baclofen are infused via lumbar puncture or a temporary catheter. If a positive improvement in Ashworth score is noted, the patient then becomes a candidate for placement of the pump itself. Intrathecal baclofen pump implantation is a minor procedure performed as an outpatient, but does require continued follow-up for refills of the pump and adjustments of the rate of infusion to maximize benefits.

iv. Hemifacial spasm

Hemifacial Spasm is a condition in which spasmodic, involuntary, usually intermittent, usually unilateral, and painless contractions of the facial muscles occur. In rare instances, the contractions may be limited to just one portion of the facial nerve distribution. Patients may occasionally have excessive lacrimation. Usually the orbicularis oculi start the contractions, but they usually progress to involve the entire half of the face. The condition usually increases in frequency until the ability to see out of the affected eye is impaired. It is critical to distinguish this from facial myokymia, a continuous facial spasm. One must also distinguish hemifacial spasm from blepharospasm, a bilateral spasmodic closure of the orbicularis oculi muscles. It is interesting to note that hemifacial spasm and palatal

myoclonus are the only involuntary movement disorders that can still persist during sleep. In other types of movement disorders, the symptoms dissipate with the induction of sleep. Hemifacial spasm shows a predilection for the left side, usually appears sometime after the teenage years, and is more common in women. Alteration of cranial nerve VIII function is noted frequently as well.

The pathogenesis of the disease appears to be compression of the facial nerve at the root entry zone by an artery or other vascular structure. Most commonly this artery is the anterior inferior cerebellar artery but other veins or arteries may cause compression as well. Typically the vessel that contacts the root entry zone causes other symptoms as well (e.g. compression of the vestibular nerves causes vertigo, cochlear nerve compression causes tinnitus or hearing loss). Occasionally, benign tumors, cysts, multiple sclerosis, or bony abnormalities may cause hemifacial spasm due to irritation of the facial nerve. In hemifacial spasm the facial motor nucleus may be involved secondarily as the result of the root entry zone compression via a phenomena similar to kindling.

Patients should undergo an MRI scan to rule out a posterior fossa mass such as a tumor or AVM. Occasionally the offending vessel may be seen on these studies. Diagnostic work-up otherwise will be negative. Medical management is usually limited to botulinum (Botox) injections. Botulinum toxin injections may be effective in temporarily treating hemifacial spasm and particularly blepharospasm, but, in general, hemifacial spasm is a surgically treated disease. Because of this, patients with this disease, with limitation of vision or significant limitation of their activities of daily living, will present for surgical treatment.

There are a number of options for surgical treatment. Ablative procedures are effective but they will leave the patient with facial paresis or paralysis. The current procedure of choice is microvascular decompression (MVD), in which the offending vessel is physically moved off of the nerve and cushioning material similar to that utilized in the treatment of trigeminal neuralgia is interposed. Though it is common for a patient to show mild hemifacial spasm immediately after decompression, the symptoms usually have significantly improved by 2-3 days following the procedure. Facial weakness, remarkably, is a rare postoperative complication but hearing decrease may occur in as many as 10-33% of patients. Some patients will not resolve the hemifacial spasm even after microvascular decompression. Approximately 10% of the patients will show recurrence of symptoms.

c. Understanding the general classification of seizure disorders, definition of intractable epilepsy, and the broad categories of surgical intervention for epilepsy including invasive electrodes, resective and disconnective surgery.

i. Classification of seizure disorders:

Seizures are defined as sudden cerebral neuronal discharges that result in alterations of motor function, behavior, or consciousness. This discussion, of course, will be limited and cannot cover all aspects of seizure diagnosis and treatment, but will give brief descriptions of each of the major types.

Seizures are usually divided into either generalized or partial seizures. Generalized seizures involve bilateral symmetric synchronous activation of both cerebral hemispheres with evidence of a non-focal onset and loss of consciousness at the very start of the seizure. Generalized seizures account for approximately 40% of seizures.

There are six major types of generalized seizures:

- ☐ Clonic seizures, characterized by symmetric bilateral synchronous semirhythmic jerking of the extremities
- ☐ Tonic seizures, which include sudden sustained increase in tone often accompanied by a characteristic cry or grunt as air is forced through the adducted vocal cords
- ☐ Generalized tonic clonic seizures, which usually involve a generalized seizure that evolves from tonic to clonic motor activity
- ☐ Absence seizures (previously called petit-mal seizures), in which there is impaired consciousness but only mild or no motor movement
- ☐ Myoclonic seizures, characterized by shock-like body jerks
- ☐ Atonic seizures (sometimes called drop attacks), which involve sudden brief loss of tone that may cause falls.

Partial seizures, on the other hand, involve only one hemisphere or one focal center in the brain at the onset. When partial seizures exist, one should conduct a thorough evaluation for a potential structural lesion. Surgery is most useful in the treatment of this type of seizure. Partial seizures are divided into simple partial, complex partial, and partial seizures with secondary generalization.

- ☐ Simple partial seizures have no impairment of consciousness and may involve either motor signs, autonomic signs, or sensory symptoms
- ☐ Complex partial seizures involve an alteration of consciousness, usually with a loss of consciousness and automatisms (e.g. chewing, picking with the fingers, or lipsmacking) and, oftentimes, an autonomic aura, e.g. gastric discomfort.
- ☐ Partial seizures may generalize, with simple partial or complex partial seizures evolving to generalization, or, in some instances, with a simple partial seizure evolving to a complex partial seizure that then evolves to a generalized seizure.

Some patients may have seizures due to a known etiology, also sometimes known as symptomatic or secondary seizures. Etiologies include stroke, tumor, and mesial temporal sclerosis. Other seizures, sometimes referred to as primary seizures or idiopathic seizures, may occur without an underlying cause, e.g. juvenile myoclonic epilepsy.

ii. Intractable epilepsy

Twenty percent of patients with epilepsy will continue to have seizures even with antiepileptic medication. Surgery becomes a consideration for these patients under the classification of medically refractory seizures. Usually the seizure disorder must be severe, with the seizures being medically refractory with several trials of tolerable medication for at least one year, with significant disability to the patient. In functional terms, the patient has usually been tried on two different

monotherapy antiepileptic drugs at high doses and has had one attempt at polytherapy.

iii. Surgical intervention for epilepsy:

aa. Invasive electrodes:

Invasive techniques are sometimes required to definitely identify a seizure focus. These techniques include subdural strip electrode placement as well as depth electrode placement. Subdural strip electrode placement involves creation of small burr holes through which electrodes are then passed in a subdural location over the brain surface to determine, in an invasive fashion, and with more precision, the actual seizure focus. Depth electrodes may be placed, usually stereotactically, to localize the seizure focus as well. In cases where the general region of a seizure focus is known but the exact location is not, subdural grid electrodes may be placed via craniotomy. These grids also allow mapping to be performed, either in surgery, or in an epilepsy unit, in order to evaluate motor function, language function, and other vital centers that may be near the epileptogenic focus.

bb. Resective surgery:

By far the most common epilepsy operation is temporal lobectomy with resection of the amygdala and hippocampus. Usually, if surgery is performed on the dominant hemisphere, the temporal lobe is removed from the temporal tip to a point approximately 4-5 cm posteriorly. If resection is proceeded beyond this distance, speech centers may be involved. Nondominant temporal lobe resections can go further back, usually 6-7 cm. Intraoperative electrocorticography is used in some centers to guide the resection. Patients undergoing such a temporal lobectomy in an epilepsy center can usually expect a 60-70% incidence of seizure control (defined as the absence of seizure activity) with or without medications. Other patients may experience a reduction in seizure frequency, even if not rendered totally seizure-free.

cc. Disconnective surgery:

Disconnection surgeries are usually utilized when eloquent brain is involved, or to separate the electrical activity of the two cerebral hemispheres. Disconnective surgery includes corpus callosotomy (particularly useful when drop attacks are the most disabling seizure type), hemispherectomy (utilized for unilateral seizures with wide spread hemispheric lesions and profound contralateral neurologic deficit), and multiple subpial transections (MST). In MST, the cortex is transected at 5 mm intervals, interrupting the horizontal spread of seizures, while sparing the vertically oriented functional fibers. Most recently, the vagus nerve stimulator has been used to interrupt the spread of seizure activity. This device consists of a pulse generator coupled to an electrode system around the left vagus nerve. The exact method of inhibition of seizures is unclear, but it has been noted to have significant benefit in both focal seizures as well as generalized seizures.