

Key Terms

Dura mater The tough, fibrous outermost layer of the three meninges that surround the brain and spinal cord.

Hematoma A bruise or collection of blood within soft tissue that results in swelling.

(e.g., incontinence or inability to control the bladder or bowels).

Diagnosis

Neurologic assessment is the first step in determining the severity of a head injury. The patient's speech, eye-opening, and muscular responses are evaluated, along with the orientation (if conscious) to place, time, and commands to open eyes or the like. If the patient is unconscious, examination of the pupillary light reflex is important. An epidural or other hematoma increases intracranial pressure, which quickly has an effect on the third cranial nerve, which contains, among other nerve fibers, those that control constriction of the pupil. Pressure that blocks this nerve leads to fixed dilation of the pupil. Fixed pupil dilation in one or both eyes is a strong indicator that the patient may have an intracranial hematoma. To distinguish between epidural, subdural, and intracranial hematoma, computerized tomography (CT) or **magnetic resonance imaging (MRI)** is probably necessary. Surgeons determine if swelling on one side of the brain has shifted the midline of the brain. If a shift of more than 0.2 in (5 mm) is found, an emergency **craniotomy** (opening of the skull) may be performed.

Patients with spinal epidural hematoma may experience sudden onset of back or neck **pain** at the site of the bleed. Coughing or any other maneuver that increases pressure inside the torso may worsen the pain transiently. In children, the bleeding is more likely to be in the cervical (neck) region than in the thoracic (middle back) region.

When making the diagnosis of spinal epidural hematoma, physicians must decide whether the symptoms of spinal compression are being caused by a hematoma or by a tumor. CT or MRI are definitive in distinguishing between compression of the spinal cord caused by tumor or hematoma.

Treatment team

Treatment for hematoma is primarily surgical. A **neurologist** and a neurosurgeon will be essential members of

the treatment team, as will nursing staff, in the operating room and out of it, who are specially trained in head trauma care.

Treatment

Emergency care for spinal trauma consists of immobilizing the patient and administering high-dose corticosteroids. However, the highest priority for any intracranial or spinal hematoma is relief of the pressure by surgical drainage of the hematoma.

Recovery and rehabilitation

Epidural hematoma can result in permanent paralysis or other neurological deficits. If spinal cord compression due to hematoma is alleviated within 6–12 hours, permanent symptoms may be avoided. Prevention of brain damage depends more on preventing the brain from being deformed by the pressure of the hematoma than on relieving that pressure. Rehabilitation needs will depend on how much permanent damage, if any, has been caused.

Clinical trials

As of 2004, no **clinical trials** were being conducted for epidural hematoma patients in the United States.

Resources

PERIODICALS

Marsh, Cherly. "Surgical Management of Patients with Severe Head Injuries." *AORN Journal* May 1, 1996.

Sung, Helen Minjung. "How to Diagnose and Treat Acute, Nontraumatic Spinal Cord Lesions." *The Journal of Critical Illness* April 1, 2000.

Trask, Todd. "Management of Head Trauma (Critical Care Review)." *Chest* August 1, 2002.

OTHER

Epidural Hematoma Patient/Family Resources. April 26, 2004 (May 30, 2004). <<http://cchs-dl.slis.ua.edu/patientinfo/orthopedics/head/epidural-hematoma.htm>>.

NINDS Traumatic Brain Injury Information Page. National Institute of Neurological Disorders and Stroke. April 26, 2004 (May 30, 2004). <http://www.ninds.nih.gov/health_and_medical/disorders/tbi_doc.htm>.

Larry Gilman

Epilepsy

Definition

The words "epilepsy" and "epileptic" are of Greek origin and have the same root as the verb "epilambanein," which means "to seize" or "to attack." Therefore, epilepsy

means seizure, while epileptic means seized. In the modern understanding of epilepsy, it should not be considered a disease. Rather, it is a symptom indicating a medical condition in the brain that causes a potential for recurrent **seizures**. The condition of epilepsy has many causes and the kinds of seizures that occur can vary widely.

Description

The word epilepsy is actually a descriptive term. It takes into account an individual's risk of recurrent seizures. However, when people are suffering from meningitis and have a seizure, they would not be considered to have epilepsy unless they had a seizure after the meningitis resolved. In this case, these individuals have a risk for recurrent seizures and, hence, epilepsy. If an individual over time does not have any seizures off medications, then it could be said that epilepsy has resolved or gone into remission.

For thousands of years, epilepsy was looked upon differently than most other medical problems. Because of this, epilepsy has been fraught with social stigmas, even up to today. The ancient Greeks knew about the condition that led to a sudden attack upon the unfortunate. Although Hippocrates, in roughly 400 B.C., referred to epilepsy as the sacred disease, he did so to emphasize the general public's superstitious view of the condition. Of course, it certainly was not an affliction sent from a deity, nor was it even a demon. Nevertheless, seizures, which manifest in unusual behaviors, mystified observers who considered this illness, from all others, as coming from another world.

The current understanding of epilepsy is a recent development. Previously, it was not even believed that the brain had electrical properties. It was not until the last few centuries that the brain was considered the seat of the mind; it was the heart or the lungs that were commonly regarded as the organ of thought. Physicians struggled with what to even call a seizure. In general, any behavior that resulted in a loss of consciousness or convulsions was labeled a seizure. It is likely that episodes of **fainting** were erroneously called seizures.

Finally, in 1873, an adequate definition for the term seizure finally came into existence. The famous English **neurologist** John Hughlings Jackson explained epilepsy as "a sudden, excessive, and rapid discharge of gray matter of some part of the brain" that would correspond to the patient's experience.

Demographics

More than 2.5 million Americans suffer from epilepsy, and more than another 50 million worldwide. Epilepsy is more common than **Parkinson's disease**, **multiple sclerosis**, **cerebral palsy**, and **muscular dystrophy**

Key Terms

Automatisms Movements during a seizure that are semi-purposeful but involuntary.

Gelastic seizures Seizures manifesting with brief involuntary laughter.

Gray matter The portion of the brain that contains neurons, as opposed to white matter, which contains nerve tracts.

Spike wave discharge Characteristic abnormal wave pattern in the electroencephalogram that is a hallmark of an area that has the potential of generating a seizure.

all combined. The risk of experiencing one seizure in the course of a lifetime, from any cause, is close to 10%. However, there is an approximately 1% chance of developing epilepsy in the general population before the age of 20. The risk increases to 3% by age 75. Of course, depending on the age group being studied, the cause of epilepsy will vary. The incidence of epilepsy is relatively constant among different ethnic groups and similar between genders. However, there may be variation in incidence in underdeveloped countries due to access to care and endemic illness that can cause seizures, such as neurocystercercosis in Latin American countries.

Causes and symptoms

Epilepsy has many causes that, in part, have an affect on the clinical presentation of symptoms. In order for epilepsy to occur, there must be an underlying physical problem in the brain. The problem can be so mild that an individual is perfectly normal other than seizures. The brain has roughly 50–100 billion neurons. Each neuron can have up to 10,000 contacts with neighboring neurons. Hence, trillions of connections exist. However, only a very small area of dysfunctional brain tissue is necessary to create a persistent generator of seizures and, hence, epilepsy. The following are potential causes of epilepsy:

- genetic and/or hereditary
- perinatal neurological insults
- trauma with brain injury
- **stroke**
- brain tumors
- infections such as meningitis and encephalitis
- multiple sclerosis
- ideopathic (unknown or genetic)

Any of the above conditions have the potential for causing the brain or a portion of it to be dysfunctional and produce recurrent seizures. Regardless of the exact cause, epilepsy is a paroxysmal (sudden) condition. It involves the synchronous discharging of a population of neurons. This is an abnormal event that, depending on the location in the brain, will correspond to the particular symptoms of a seizure. The International League Against Epilepsy (ILAE) issued a classification of types of seizures. The list gives the kind of seizures that can occur. Individual seizure types are based on the clinical behavior (semiology) and electrophysiological characteristics as seen on an electroencephalogram (EEG). Generalized seizures included in the list are:

- tonic-clonic seizures (includes variations beginning with a clonic or myoclonic phase)
- clonic seizures, including without tonic features and with tonic features
- typical absence seizures
- atypical absence seizures
- myoclonic absence seizures
- tonic seizures
- spasms
- myoclonic seizures
- eyelid myoclonia, including without absences and with absences
- myoclonic atonic seizures
- negative **myoclonus**
- atonic seizures
- reflex seizures in generalized epilepsy syndromes

Focal seizures included in the ILAE list are:

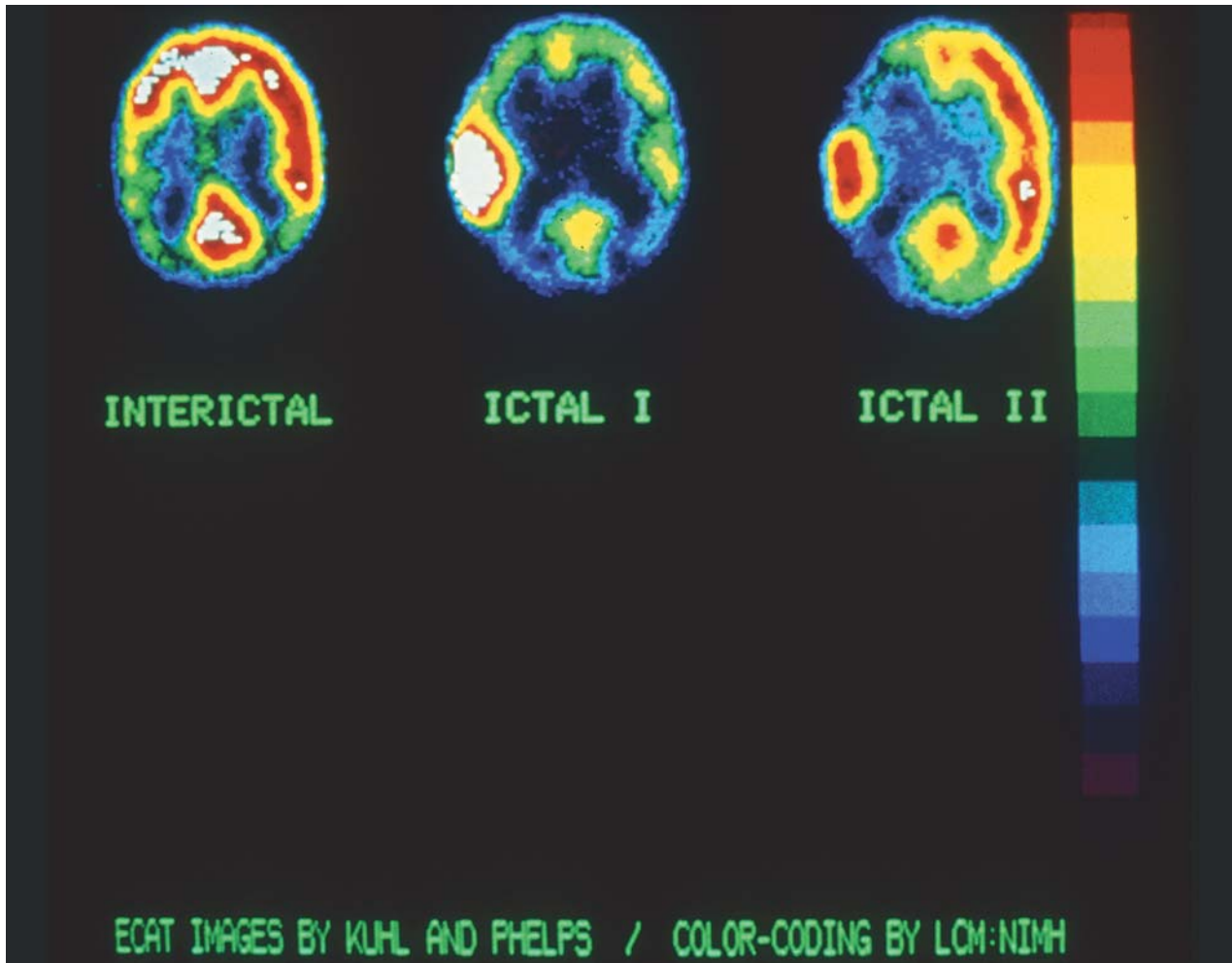
- focal sensory seizures with elementary sensory symptoms (e.g., occipital and parietal lobe seizures) and experiential sensory symptoms (e.g., temporo-parieto-occipital junction seizures)
- focal motor seizures with elementary clonic motor signs, asymmetrical tonic motor seizures (e.g., supplementary motor seizures), typical (temporal lobe) automatisms (e.g., mesial temporal lobe seizures), hyperkinetic automatisms, focal negative myoclonus, and inhibitory motor seizures
- gelastic seizures
- hemiclonic seizures
- secondarily generalized seizures
- reflex seizures in focal epilepsy syndromes

In 1989, the International League Against Epilepsy also issued the following classification of epilepsies and epileptic syndromes:

- benign familial neonatal seizures
- early myoclonic encephalopathy

- Ohtahara syndrome
- migrating partial seizures of infancy (syndrome in development)
- West syndrome
- benign myoclonic epilepsy in infancy
- benign familial and non-familial infantile seizures
- Dravet's syndrome
- HH syndrome
- myoclonic status in nonprogressive encephalopathies (syndrome in development)
- benign childhood epilepsy with centrotemporal spikes
- early onset benign childhood occipital epilepsy (Panayiotopoulos type)
- late-onset childhood occipital epilepsy (Gastaut type)
- epilepsy with myoclonic absences
- epilepsy with myoclonic-astatic seizures
- **Lennox-Gastaut syndrome**
- Landau-Kleffner syndrome (LKS)
- epilepsy with continuous spike-and-waves during slow-wave sleep (other than LKS)
- childhood absence epilepsy
- progressive myoclonus epilepsies
- idiopathic generalized epilepsies with variable phenotypes include juvenile absence epilepsy, juvenile myoclonic epilepsy, and epilepsy with generalized tonic-clonic seizures only
- reflex epilepsies
- idiopathic photosensitive occipital lobe epilepsy
- other visual sensitive epilepsies
- primary reading epilepsy
- startle epilepsy
- autosomal dominant nocturnal frontal lobe epilepsy
- familial temporal lobe epilepsies
- generalized epilepsies with **febrile seizures** plus (syndrome in development)
- familial focal epilepsy with variable foci (syndrome in development)
- symptomatic focal epilepsies
- limbic epilepsies
- mesial **temporal lobe epilepsy** with hippocampal sclerosis
- mesial temporal lobe epilepsy defined by specific etiologies
- neocortical epilepsies
- Rasmussen syndrome

Classifying epilepsy can help in the evaluation and management of patients with seizure disorders. The combination of seizure type(s), etiology (cause), age of onset,



PET scans of a human brain during the stages of an epileptic seizure; the middle image represents the most severe period of the seizure. (© Photo Researchers, Inc. Reproduced by permission.)

family history, and other medical or neurological conditions can be used to identify an epilepsy syndrome. Classification helps clinicians and researchers understand the broader picture of seizure disorders. On a practical level, syndrome identification can help in planning the management of patients. Syndrome classification schemes are revised periodically as individual components of particular categories are better understood.

The term idiopathic refers to a cause that is suspected to be, if not genetic, then unknown. Cryptogenic is a term that suggests that an underlying cause is suspected, but not yet fully understood. Symptomatic is a term that is applied to epilepsies that are a result of understood underlying pathologies.

The management and prognosis vary considerably among these differing syndromes. Epilepsies that have a genetic basis can be inherited or occur spontaneously. A

detailed family history can often identify other family members who have had seizures. However, because seizures are common, it is possible to have more than one family member with epilepsy, though the etiologies may not be related. To say that a particular type of epilepsy is genetic does not mean that it is necessarily transmitted by heredity. Often, disorders can have a genetic cause, but be spontaneously occurring in only one member of a family. In this case, there may simply be a random mutation in that particular person's genes.

There are several mechanisms in which epilepsies can be inherited. So-called simple Mendelian inheritance occurs with benign familial neonatal convulsions and autosomal dominant nocturnal frontal lobe epilepsy. On the other hand, complex inheritance mechanisms can involve more than one gene, or a gene mutation in combination with environmental or acquired factors such as juvenile

myoclonic epilepsy. As the genetics of the epilepsies become better understood, the classification scheme will evolve.

With epilepsy, symptoms vary considerably depending on the type. The common link among the epilepsies is, of course, seizures. The different epilepsies can sometimes be associated with more than one seizure type. This is the case with Lennox-Gastaut syndrome.

Diagnosis

Arriving at a diagnosis of epilepsy is relatively straightforward: when people suffer two or more seizures, they would be considered to have epilepsy. However, diagnosing the specific epilepsy syndrome is much more complex. The first step in the evaluation process is to obtain a very detailed history of the illness, not only from the patient but from the family as well. Since seizures can impair consciousness, the patient may not be able to recall the specifics of the attacks. In these cases, family or friends that have witnessed the episodes can fill in the gaps about the particulars of the seizure. The description of the behaviors during a seizure can go a long way to categorizing the type of seizure and help with the overall diagnosis. Moreover, in the initial visit with the physician, the entire history of the patient is obtained. In a child, this would include birth history, complications, if any, maternal history, and developmental milestones. At any age, so-called co-morbidities (other medical problems) are considered. Medications that have been taken or currently being prescribed are documented.

A complete physical examination is performed, especially a neurological exam. Because seizures are an episodic disorder, abnormal neurological findings may not be present. Frequently, people with epilepsy have a normal exam. However, in some, there can be abnormal findings that can provide clues to the underlying cause of epilepsy. For example, if someone has had a stroke that subsequently caused seizures, then the neurological exam can be expected to reveal a focal neurological deficit such as weakness or language difficulties. In some children with seizures, there can be a variety of associated neurologic abnormalities such as **mental retardation** and cerebral palsy that are themselves non-specific but indicate that the brain has suffered, at some point in development, an injury or malformation. Also, subtle findings on examination can lead to a diagnosis such as in **tuberous sclerosis**. This is an autosomal dominantly inherited disorder associated with **infantile spasms** in 25% of cases. On examination, patients have so-called ash-leaf spots and adenoma sebaceum on the skin. There can also be a variety of systemic abnormalities that involve the kidneys, retina, heart, and gums, depending on severity.

In the course of evaluating epilepsy, a number of tests are typically ordered. Usually, magnetic resonance image (**MRI**) of the brain is obtained. This is a scan that can help in finding many known causes of epilepsy such as tumors, strokes, trauma, and congenital malformations. However, while MRI can reveal incredible details of the brain, it cannot visualize the presence of abnormalities in the microscopic neuronal environment. Another test that is routinely ordered is an electroencephalogram (EEG). Unlike the MRI scan, this can be considered a functional test of the brain. The EEG measures the electrical activity of the brain. Some seizure disorders or epilepsies have a characteristic EEG with particular abnormalities that can help in diagnosis. Other tests that are frequently ordered are various blood tests that are also ordered in many medical conditions. These blood tests help to screen for abnormalities that can be a factor in the cause of seizures. Occasionally, genetic testing is performed in those instances where a known genetic cause is suspected and can be tested. A major concern in the course of an evaluation of epilepsy is to identify the presence of life-threatening causes such as brain tumors, infections, and cerebrovascular disease. Also, an accurate diagnosis can expedite the most effective treatment plan.

The symptoms of epilepsy are dependent in part on the particular seizures that occur and other medical problems that may be associated. Seizures, themselves, can take on a variety of features. A simple sustained twitching of an extremity could be a focal seizure. If a seizure arises in the occipital lobes of the brains, then a visual experience can occur. Aura is a term often used to describe symptoms that a person may feel prior to the loss of consciousness of a seizure. However, auras are, themselves, small focal seizures that have not spread in the brain to involve consciousness. Smells, well-formed hallucinations, tingling sensations, or nausea have each occurred in auras. The particular sensation can be a clue as to the location in the brain where a seizure starts. Focal seizures can then spread to involve other areas of the brain and lead to an alteration of consciousness, and possibly convulsions. In certain epilepsy syndromes such as Lennox-Gastaut, there can be more than one type of seizure experienced, such as atonic, atypical absence, and tonic-axial seizures.

Treatment

One challenge in predicting the course of epilepsy is that for any type, there can be a variable response to treatment. Sometimes, seizures may play a rather small role in the manifestation of a medical condition. For example, a severe head injury could result in seizures that readily respond to medication, but severe neurological impairments and disabilities may still be present. On the other hand, a

different head injury may result in relatively mild neurological problems, but there may be seizures that are severe and be resistant to medications.

Whatever the case, the ultimate goals when treating epilepsy are to:

- strive for complete freedom from seizures
- have little to no side effects from medications
- be able to follow an easy regimen so that compliance with treatment can be maintained

Up to 60% of patients with epilepsy can be expected to achieve control of seizures with medication(s). However, in the remaining 40%, epilepsy appears to be resistant, to varying degrees, to medications. In these cases, the epilepsy is termed medically intractable.

Generally, the choice of medication is somewhat trial and error. There are, however, a number of considerations that guide the choice of treatment. Each medication has a particular side effect profile and mechanism of action. Some medications seem to be particularly effective for certain epilepsy syndromes. For example, juvenile myoclonic epilepsy responds well to valproic acid. On the other hand, ethosuxamide is primarily used for absence seizures.

As with any medication, individuals can have very different experiences with same drug. Consequently, it is difficult to predict the efficacy of treatment in the beginning. A key concept of treatment is to first strive for monotherapy (or single drug therapy). This simplifies treatment and minimizes the chance of side effects. Sometimes, however, two or more drugs may be necessary to achieve satisfactory control of seizures. As with any treatment, potential side effects can be worse than the disease itself. Moreover, there is little point in controlling seizures if severe side effects limit quality of life. If a seizure disorder is characterized by mild, focal, or brief symptoms that do not interfere with day-to-day life, then aggressive treatments may not be justified. Epilepsy medications do not cure epilepsy; the medications can only control the frequency and severity of seizures. A list of the most commonly used medications in the management of epilepsy includes:

- phenobarbital
- phenytoin (Dilantin, Phenytek)
- clonazepam (Klonopin)
- ethosuxamide (Zarontin)
- carbamazepine (Tegretol, Carbatrol)
- divalproex sodium (Depakote, Depakene)
- felbamate (Felbatol)
- gabapentin (Neurontin)

- lamotrigine (Lamictal)
- topiramate (Topamax)
- tiagabine (Gabatril)
- zonisamide (Zonegran)
- oxcarbazepine (Trileptal)
- levetiracetam (Keppra)

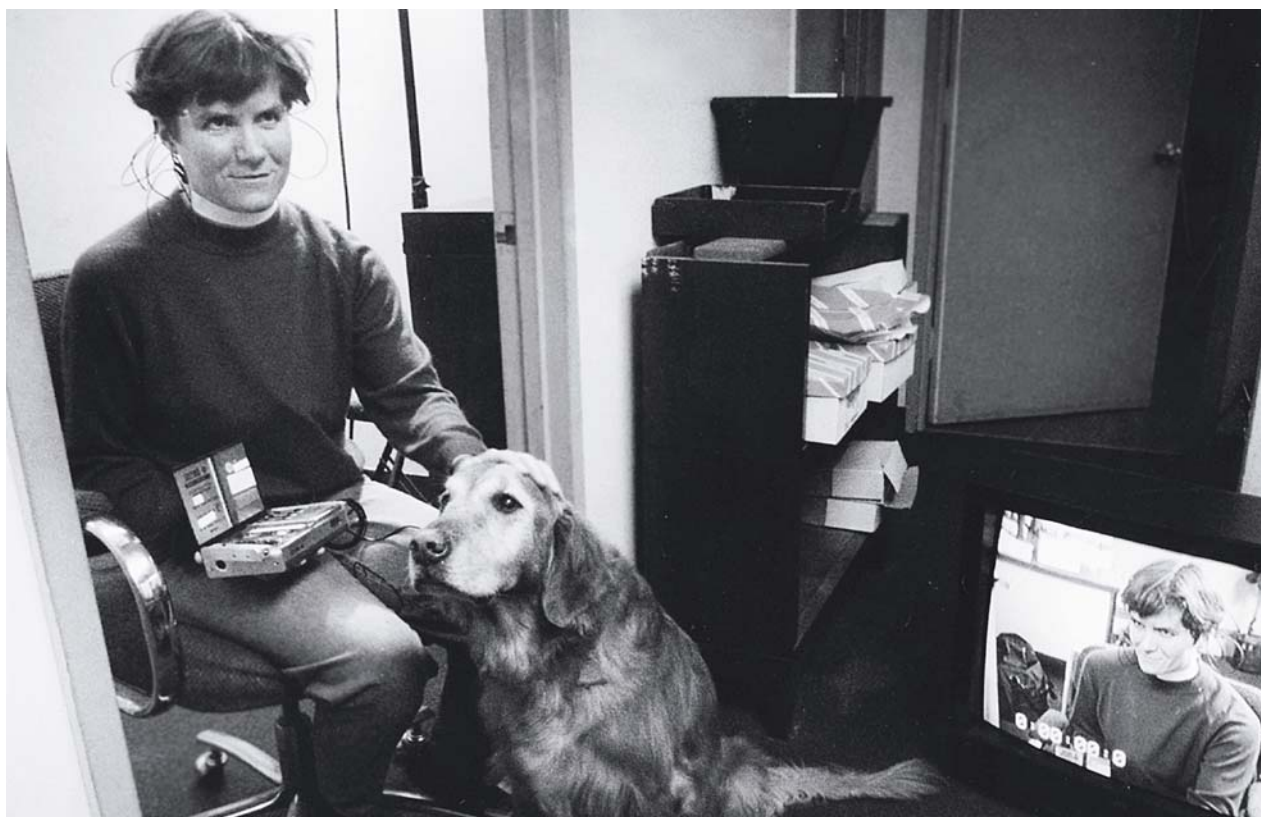
It has been found that the initial, thoughtfully chosen medication can be expected to make almost 50% of patients seizure free for extended periods of time. If the initial drug fails, another well-chosen drug may make an additional 14% of people seizure free. If that drug fails, then the likelihood of rendering someone with epilepsy seizure free is poor. This does not mean that trying more medications or combinations of them may not be successful, but rather, these statistics give the neurologist and the patient an understanding of the realities of epilepsy treatment. In cases where medications do not fully control epilepsy, it is recommended that a more extensive evaluation at a comprehensive epilepsy center be conducted where an epileptologist (a specialist in epilepsy) will more thoroughly assess the particular aspects of the seizures. When medications are clearly ineffective, the other types of therapy that can be considered are the ketogenic diet, brain surgery, and vagal nerve stimulation.

Ketogenic diet

The ketogenic diet is based on high-fat, low-carbohydrate, and low-protein meals. The ketogenic diet is named because of the production of ketones by the breakdown of fatty acids. The most common version of the diet involves long-chain triglycerides. These are present in whole cream, butter, and fatty meats.

The ketogenic diet is administered with the support of a nutritionist with experience in this treatment modality. It is mostly used in children with medically intractable epilepsy and whose diet can be controlled. The ketogenic diet can be considered a pharmacologic treatment. As such, there are potential side effects that limit its tolerance. This includes hair thinning, lethargy, weight loss, kidney stones, and possibly cardiac problems. Sugar-free vitamin and mineral supplementation is necessary. The diet may not be appropriate for certain individuals, particularly in children, who may have certain metabolic diseases.

Overall, the diet has been very helpful in the control of seizures in many patients. Roughly 50% of patients can hope to achieve complete control of seizures, 25% of the patients see improvements, and another 25% are non-responders. There are some patients who have an improvement in behavior. If the diet is well tolerated with good results, then it can be maintained for up to two years, followed by a careful gradual transition to regular meals.



Elizabeth Rudy, who suffers from epilepsy, sits hooked up to brain wave monitor. Her left hand strokes her seizure-predicting dog, Ribbon. (A/P Wide World Photos. Reproduced by permission.)

Epilepsy surgery

Epilepsy surgery is an option in the attempt to either cure or significantly reduce the severity of medically resistant cases. It is thought that up to 100,000 patients in the United States could be potential candidates for a surgical treatment. However, only about 5,000 cases are performed throughout the United States annually. This is likely due to several factors, including the belief that any brain surgery is a last resort, the lack of awareness or understanding of the benefits of surgery, and the false hope that some medication will come along that will be effective.

There are several kinds of surgery that are available depending on the nature of the seizure disorder. A list of operations that are utilized regularly for epilepsy include:

- lobectomy
- lesionectomy
- corpus collosotomy
- multiple subpial transection
- hemispherectomy

The type of surgery that is performed depends on the nature of the individual seizure disorder. If a seizure can

be localized to a particular area in the brain, then this abnormal region can potentially be surgically removed. Epileptic brain tissue is abnormal and its removal can provide a chance of a cure. Generally, surgery should be a consideration when the risk and benefits of it outweigh the long-term risks of uncontrolled epilepsy.

The approach taken in any brain surgery for epilepsy is highly individualized and great care is taken to avoid injury to essential brain tissue. The most common epilepsy surgery performed is the temporal lobectomy. Brain tumors are frequently associated with seizures. In many cases, surgery to remove the tumor is planned so that regions that may be causing seizures are removed as well. However, in many cases, epilepsy surgery cannot be done.

Vagus nerve stimulation

Another non-medicinal approach to treating epilepsy is a novel method that became available in July 1997. The Food and Drug Administration (FDA) approved the use of the vagal nerve stimulator (VNS) as add-on therapy in patients who experience seizures of partial onset. The VNS is designed to intermittently deliver small electrical stimulations to a nerve in the neck called the vagus nerve.

There are two vagal nerves, one on each side of the neck near the carotid arteries, making a pair of cranial nerves (there are 12 different paired cranial nerves). The vagus nerve carries information from the brain to many parts of the thoracic and abdominal organs. The nerve also carries information from these same organs back to the brain. VNS takes advantage of this fact and, by intermittent stimulation, there is an effect on many brain areas that can be involved in seizures.

About 50% of patients experience at least 50% reduction in the frequency of their seizures. The responses to VNS range from complete control of seizures (less than 10% of patients) to no noticeable improvement. The device is not a substitute for epilepsy surgery and should be considered only after there is an evaluation for epilepsy surgery. The implantation of the device requires relatively minor surgery with two incisions, one in the neck and the other in the left upper chest area.

The battery in the device lasts up to eight to ten years, after which the device can be replaced. Side effects of VNS therapy include voice hoarseness that typically does not impair communication. Like any surgery, there is an initial risk of infection, bleeding, and **pain**. Recovery takes a few weeks. Individuals can return to their usual activities once the incisions have healed.

Clinical trials

The National Institute of Neurological Disorders and Stroke list a number of **clinical trials**. There are also a number of studies being conducted at a more basic science stage evaluating the role of the following in seizures and epilepsy: **neurotransmitters**, non-neuronal cells, and genetic factors. Treatment strategies including **deep brain stimulation** and intracranial early seizure detection devices are being studied at different stages.

Prognosis

The prognosis of epilepsy varies widely depending on the cause, severity, and patient's age. Even individuals with a similar diagnosis may have different experiences with treatment. For example, in benign epilepsy of childhood with centrotemporal spikes (also called benign rolandic epilepsy), the prognosis is excellent with nearly all children experiencing remission by their teens. With childhood absence epilepsy, the prognosis is variable. In this case, the absence seizures become less frequent with time, but almost half of patients may eventually develop generalized tonic-clonic seizures. Overall, the seizures are responsive to an appropriate anticonvulsant. On the other hand, the seizures in Lennox-Gastaut syndrome are very difficult to control. In this case, however, the ketogenic diet can help. In seizures that begin in adulthood, one can

expect that medications will control seizures in up to 60–70% of cases. However, in some of the more than 30% of medically intractable cases, epilepsy surgery can improve or even cure the problem.

Overall, most patients have a good chance of controlling seizures with the available options of treatment. The goal of treatment is complete cessation of seizures since a mere reduction in seizure frequency and/or severity may continue to limit patients' quality of life: they may not be able to drive, sustain employment, or be productive in school.

Resources

BOOKS

- Browne, T. R., and G. L. Holmes. *Handbook of Epilepsy*, 2nd edition. Philadelphia: Lippincott Williams & Wilkins, 2000.
- Devinski, O. *A Guide to Understanding and Living with Epilepsy*. Philadelphia: F.A. Davis Company, 1994.
- Engel, J., Jr., and T. A. Pedley. *Epilepsy: A Comprehensive Textbook*. Philadelphia: Lippincott-Raven, 1998.
- Freeman, M. J., et al. *The Ketogenic Diet: A Treatment for Epilepsy*, 3rd Edition. New York: Demos Medical Publishing, 2000.
- Hauser, W. A., and D. Hesdorffer. *Epilepsy: Frequency, Causes, and Consequences*. New York: Demos Medical Publishing, 1990.
- Pellock, J. M., W. E. Dodson, and B. F. D. Bourgeois. *Pediatric Epilepsy Diagnosis and Therapy*, 2nd Edition. New York: Demos Medical Publishing, 2001.
- Santilli, N. *Managing Seizure Disorders: A Handbook for Health Care Professionals*. Philadelphia: Lippincott-Raven, 1996.
- Schachter, S. C., and D. Schmidt. *Vagus Nerve Stimulation*, 2nd Edition. Oxford, England: Martin Dunitz, 2003.
- Wyllie, E. *The Treatment of Epilepsy: Principles and Practice*, 3rd Edition. Philadelphia: Lippincott Williams & Wilkins, 2001.

PERIODICALS

- Kwan, P., and M. J. Brodie. "Early Identification of Refractory Epilepsy." *New England Journal of Medicine* no. 342 (2000): 314–319.

ORGANIZATIONS

- American Epilepsy Society. 342 North Main Street, West Hartford, CT 06117-2507. 860.586.7505. <www.aesnet.org>.
- Epilepsy Foundation of America. 4351 Garden City Drive, Landover, MD 20785-7223. (800) 332-1000. <www.epilepsyfoundation.org>.
- International League Against Epilepsy. Avenue Marcel Thiry 204, B-1200, Brussels, Belgium. + 32 (0) 2 774 9547; Fax: + 32 (0) 2 774 9690. <www.epilepsy.org>.

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Erb's palsy see **Brachial plexus injuries**