

## Epilepsy – A Brief Overview

©2005 Alain Koyama; Completed: April 2005; For correspondences, email to: alain.koyama@gmail.com

---

Epilepsy is a neurological condition in which an individual experiences chronic abnormal bursts of electrical discharge in the brain. These seizures can cause a variety of symptoms depending on the areas of the brain affected. Symptoms can vary from mild to severe and can include complete or partial loss of consciousness, loss of speech, uncontrollable motor behavior, and/or unusual sensory experiences. From various studies worldwide<sup>6, 20, 22, 24, 26</sup>, approximately 0.5% of the population is reported to be affected by active epilepsy.

It is also important to dispel a common myth that epilepsy and seizures are the same thing. Although seizures are the primary symptom of epilepsy, they can have other causes, such as high fever, malignant hypertension, or drug abuse. In these cases, the seizures stop when the condition improves, whereas seizures in epilepsy are a chronic long-term condition. Another common myth is that epilepsy occurs only in children. Although much media coverage focuses on epilepsy in children, people of all ages can be affected by epilepsy.

Epileptic seizures are caused by parts of the brain eliciting abnormal electrical activity. The region of seizure-generating tissue, or the *epileptogenic focus*, can be due to structural abnormalities that disrupt normal neural circuitry. These abnormalities may be genetic, caused by head injury, infection, stroke, or tumor. In such cases when the cause is known, it is termed *symptomatic* epilepsy. Other classifications include *idiopathic* epilepsy, when there is no identifiable cause but a genetic basis is presumed, and *cryptogenic* epilepsy, when neither classification fits and the cause is unknown.

Just as there can be multiple causes, individuals can be affected by one or more types of seizures. *Partial* seizures begin in a localized area, while *generalized* seizures develop over a widespread area on the cortex of the brain. Partial seizures can be further subdivided into *simple* and *complex*, where only complex seizures can cause loss of consciousness. Generalized seizures are grouped into six major categories. *Absence* seizures (also known as *petit mal*) are characterized by a partial loss of consciousness when the individual briefly appears vacant and unresponsive. Involuntary muscle twitches, particularly in the face, are often seen. *Myoclonic* seizures consist of very brief and sporadic arrhythmic movements. *Tonic* seizures consist of sudden stiffening movements involving the head, body, or extremities that often occur during sleep. *Clonic* seizures are characterized by repeated, rhythmic motor movements, often involving a large portion of the body as well as causing unconsciousness. *Tonic-clonic* seizures (also called *grand mal*) begin with the tonic phase of sudden stiffening movements when the individual may experience symptoms such as loss of orofacial motor control resulting in tongue biting or clenched teeth and/or urinary incontinence. This is followed by the clonic phase of rhythmic body movements. After the seizure, the individual may be emotionally distraught, feeling confused or sleepy. *Atonic* seizures consist of a sudden loss of muscle tone. A brief atonic seizure may elicit mild symptoms such as drooping of the head, but often the seizure is prolonged and the individual falls down from loss of postural tone. *Status epilepticus* is the term given to describe the life-threatening condition when an individual experiences prolonged or successive seizures with no recovery time. Depending on the medical professional, seizure activity can be considered status epilepticus if it lasts a minimum of 5 minutes up to a more conservative 30 minutes.

Dozens of epileptic syndromes exist, classified based on the symptoms and brain regions affected. One of the more common forms of epilepsy is *temporal lobe epilepsy* (TLE). The most striking symptoms are often not the typical motor behavior seen in partial seizures. Individuals may perceive sounds or smells that are not present, or visual disturbances, such as objects appearing larger or smaller than they are. Psychological symptoms can often be the

most striking, when derealization or strong spiritual sensations may be experienced. One of the most common epileptic syndromes in childhood is *benign childhood epilepsy with centrotemporal spikes*. It is considered benign as seizures are infrequent, often responsive to treatment, and typically subside in adolescence. Despite the optimistic prognosis, neuropsychiatric testing shows that cognitive difficulties can exist in areas such as language and memory<sup>5, 13</sup>. Another common syndrome is *childhood absence epilepsy*. As the name implies, it consists of absence seizures usually starting around or slightly after the preschool years, and has a similar prognosis to benign childhood epilepsy. Less frequent and more severe is *Lennox-Gastaut Syndrome* (LGS), which can consist of multiple types of seizures, developmental delay, and a high prevalence of status epilepticus. Prognosis is often poor, especially those with earlier onset<sup>4, 16</sup>, although some can have near complete remission of symptoms. Another more rare form of epilepsy, with only around 200 reported cases since 1957<sup>2</sup>, is *Landau-Kleffner Syndrome* (LKS). Initial symptoms consist of the abrupt onset of seizures and regression of language skills. Medication may alleviate seizure activity, but language impairment is difficult to treat and may continue even if the patient recovers.

*Antiepileptic drugs* (AED) are the main form of treatment for epilepsy. Although many AED's have been developed, approximately one-third of epilepsy patients are not responsive to pharmaceutical treatments<sup>12</sup>. If non-responsive to medication, surgical options can be considered. One common method is the removal or *resection* of epileptogenic tissue. Before resection, the patient undergoes extensive electrophysiological, neuroimaging and neuropsychiatric testing to strictly localize the epileptogenic tissue. Brain tissue can also be lesioned such as in a corpus callosotomy, where the main tract of fibers connecting the two hemispheres of the brain is severed to disrupt the pathways responsible for propagating generalized seizures.

A newer surgical method is *multiple subpial transections* (MST), where multiple parallel incisions are made in a restricted region of cortex in order to disrupt synchronous neural activity responsible for seizure generation. MST can be a viable alternative to resection when the epileptogenic tissue transcends critical areas in the brain, and removal of the tissue may result in serious cognitive impairment. MST is sometimes combined with resection to improve seizure activity slightly more effectively than MST alone<sup>19, 27</sup>.

A less invasive surgical treatment is *vagus nerve stimulation* (VNS), where an electrical stimulator implanted in the neck directs intermittent pulses to the vagus nerve. The patient can also activate the stimulator magnetically if they feel a seizure about to begin in order to prevent the seizure or reduce its severity. Common side effects of this treatment, such as voice alterations and tingling sensations, tend to be mild to moderate, and subside with time<sup>3</sup>.

One increasingly used noninvasive treatment for children is the *ketogenic diet*. It consists of low carbohydrate, high protein, and high fat consumption, similar to the popularized Atkins diet but more strict. Although it can be an effective alternative<sup>11, 18</sup>, the child's growth should be closely monitored as it may be negatively affected by the restrictive diet<sup>14, 17</sup>.

*Neurofeedback* is a method in which a patient attempts to regulate the abnormal brain activity responsible for seizures. Scalp electrodes relay the brain's electrical activity usually in visual form on a screen, providing feedback for the patient's progress. Neurofeedback can be a low-cost noninvasive solution with long term benefits, shown in repeated studies to have consistent positive results<sup>21, 23, 25</sup>.

In addition to physiological symptoms, epilepsy can also adversely affect quality of life by incurring mood disorders. Of particular concern is the high prevalence of depression<sup>7, 10</sup> and suicide<sup>8</sup>. Depression can result from the location of the epileptic foci, with left TLE patients most affected<sup>1, 15</sup>, as a side effect from medication, or as a reaction from the personal, social, and professional difficulties from seizure activity. It has also been theorized that as several studies have reported, high prevalence rates of depression preceding epilepsy suggest that rather than a side effect, depression can be a contributing factor to the development of epilepsy<sup>9</sup>.

## References:

1. Altshuler, L.L., et al. (1990). *Depression, anxiety, and temporal lobe epilepsy. Laterality of focus and symptoms.* Arch Neurol, 47(3): p. 284-8.
2. Beaumanoir, A., *About continuous or subcontinuous spikewave activity during wakefulness: electroclinical correlations.* Continuous spikes and waves during slow sleep electrical status epilepticus during slow sleep., ed. A. Beaumanoir, et al. 1995, London: John Libbey.
3. Ben-Menachem, E. (2002). *Vagus-nerve stimulation for the treatment of epilepsy.* Lancet Neurol, 1(8): p. 477-82.
4. Chevrie, J.J. and J. Aicardi (1972). *Childhood epileptic encephalopathy with slow spike-wave. A statistical study of 80 cases.* Epilepsia, 13(2): p. 259-71.
5. Croona, C., et al. (1999). *Neuropsychological findings in children with benign childhood epilepsy with centrotemporal spikes.* Dev Med Child Neurol, 41(12): p. 813-8.
6. Forsgren, L., et al. (2005). *The epidemiology of epilepsy in Europe - a systematic review.* Eur J Neurol, 12(4): p. 245-53.
7. Hermann, B.P., M. Seidenberg, and B. Bell (2000). *Psychiatric comorbidity in chronic epilepsy: identification, consequences, and treatment of major depression.* Epilepsia, 41 Suppl 2: p. S31-41.
8. Jones, J.E., et al. (2003). *Rates and risk factors for suicide, suicidal ideation, and suicide attempts in chronic epilepsy.* Epilepsy Behav, 4 Suppl 3: p. S31-8.
9. Kanner, A.M. (2003). *Depression in Epilepsy Is Much More Than a Reactive Process.* Epilepsy Curr, 3(6): p. 202-203.
10. Kanner, A.M. and S. Palac (2000). *Depression in Epilepsy: A Common but Often Unrecognized Comorbid Malady.* Epilepsy Behav, 1(1): p. 37-51.
11. Kossoff, E.H., et al. (2003). *Efficacy of the Atkins diet as therapy for intractable epilepsy.* Neurology, 61(12): p. 1789-91.
12. Kwan, P. and M.J. Brodie (2000). *Early identification of refractory epilepsy.* N Engl J Med, 342(5): p. 314-9.
13. Monjauze, C., et al. (2005). *Language in benign childhood epilepsy with centro-temporal spikes abbreviated form: rolandic epilepsy and language.* Brain Lang, 92(3): p. 300-8.
14. Peterson, S.J., et al. (2005). *Changes in growth and seizure reduction in children on the ketogenic diet as a treatment for intractable epilepsy.* J Am Diet Assoc, 105(5): p. 718-25.
15. Piazzini, A., et al. (2001). *Depression and Anxiety in Patients with Epilepsy.* Epilepsy Behav, 2(5): p. 481-489.
16. Roger, J., et al. (1987). *[Lennox-Gastaut syndrome in the adult].* Rev Neurol (Paris), 143(5): p. 401-5.
17. Santoro, K.B. and T. O'Flaherty (2005). *Children and the ketogenic diet.* J Am Diet Assoc, 105(5): p. 725-6.
18. Sinha, S.R. and E.H. Kossoff (2005). *The ketogenic diet.* Neurologist, 11(3): p. 161-70.
19. Spencer, S.S., et al. (2002). *Multiple subpial transection for intractable partial epilepsy: an international meta-analysis.* Epilepsia, 43(2): p. 141-5.
20. Sridharan, R. and B.N. Murthy (1999). *Prevalence and pattern of epilepsy in India.* Epilepsia, 40(5): p. 631-6.
21. Stermann, M.B. (2000). *Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning.* Clin Electroencephalogr, 31(1): p. 45-55.
22. Tellez-Zenteno, J.F., et al. (2004). *National and regional prevalence of self-reported epilepsy in Canada.* Epilepsia, 45(12): p. 1623-9.
23. Uhlmann, C. and W. Froscher (2001). *Biofeedback treatment in patients with refractory epilepsy: changes in depression and control orientation.* Seizure, 10(1): p. 34-8.
24. (unknown) (1994). *Current Trends Prevalence of Self-Reported Epilepsy -- United States, 1986-1990.* CDC Morbidity and Mortality Weekly Report, 43(44): p. 810-811,817-818.

25. Walker, J.E. and G.P. Kozlowski (2005). *Neurofeedback treatment of epilepsy*. Child Adolesc Psychiatr Clin N Am, 14(1): p. 163-76, viii.
26. Wang, W.Z., et al. (2003). *The prevalence and treatment gap in epilepsy in China: an ILAE/IBE/WHO study*. Neurology, 60(9): p. 1544-5.
27. Zhao, Q., et al. (2003). *Evaluation of the combination of multiple subpial transection and other techniques for treatment of intractable epilepsy*. Chin Med J (Engl), 116(7): p. 1004-7.