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A Neuroscientist's Guide to Seizures and Epilepsy

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OPINION

The excessive hypersynchronous discharge of neurons in the brain causes seizures which is a paroxysmal disruption of neurologic function. The term epileptic seizure is used to distinguish a seizure caused by abnormal neuronal firing from a seizure that is not caused by abnormal neuronal firing such as a psychogenic seizure. Recurrent spontaneous seizures are referred to as epilepsy. Epilepsy can be caused by a variety of factors each of which reflects underlying brain dysfunction because it is a short-term secondary disease, not chronic. A seizure triggered by a reversible insult like fever, hypoglycemia does not qualify as epilepsy. The term epilepsy syndrome refers to a group of clinical characteristics that appear to be related such as seizure types, age of onset, Electrocardiographic (EEG) findings, triggering factors, genetics, natural history, prognosis, and Antiepileptic Treatment Response (AEDs). The unspecified term seizure disorder should be avoided. Epilepsy is one of the most common neurologic cases with 50 new cases per year per 100,000 people. About 1% of people have epilepsy and about one-third of patients have epilepsy that is unconsciousness that can be controlled by two or more well-chosen antiepileptic drugs or other therapies. About 75% of epilepsy begins in childhood indicating that the developing brain can scan. The most recent edition of the International League against Epilepsy (ILAE), published in 2010, reviews the past divisions using terms and concepts appropriate for the modern era. Fainting is divided into three categories: generalized, concentrated (formerly partial), and epileptic seizures. Concentrated fainting occurs in neuronal networks limited to one part of the cerebral hemisphere. Typical captures start on distributed networks that are distributed in two countries. Fainting can start with concentration and later produce. Fainting can occur in the cortex or subcutaneous structures. Using detailed history, EEG detection, and helpful information, the physician may classify the type of seizures/epilepsy, after which a proper diagnostic and diagnostic procedure is performed. The main subtypes of common seizures are absent, Generalized Tonic-clonic (GTC), myoclonic, and atonic. Absent fainting (formerly called petit mal) involves looking unconsciously at the outer objects of the mouth, sometimes blinking an eye or nodding one's head. The GTC grip (formerly known as grand mal) contains a concomitant movement (stiffness followed by shaking) of all body parts with cognitive impairment. Myoclonic fainting consists of sudden, short movements (lightning-fast) that are not related to obvious visual disturbances. These short unavoidable muscle changes can affect one or more muscles hence myoclonic seizures may be normalized or concentrated. Atonic seizures include loss of body tone which often leads to headaches or falls. Clinical manifestations of focused capture depend on the location of the affected cortex. For example, focused grip from the occipital lobe can also produce tangible objects from the precentral gyrus by the work of a clonic or tonic rhythm and from the postcentral gyrus with sensory symptoms such as paresthesias. When consciousness is impaired during focused seizures as the patient is unable to respond normally to a stimulus or stimuli fainting is considered dyscognitive (formerly called partial complication) fainting from the temporal lobe is often misdiagnosed. Another seizure is preceded by an aura which is the focus of attention in which the patient maintains awareness and describes motor, sensory, autonomic, or psychological symptoms. The aura precedes seizures of abnormal or normal focus in seconds or minutes and is often experienced by patients with temporal lobe epilepsy. The origin of the third stage of the seizure, epilepsy spasms, is uncertain. Epilepsy spasms are characterized by sudden enlargement or extension of the edges which are maintained for several seconds and then recur. Epilepsy outbreaks can occur at any time when they begin in the first year of life they contain a disease called infantile spasms. Epilepsies (epilepsy syndromes) were previously classified according to their original location (generalized or related

cortical area) and etiology that is whether the cause was known (symptomatic) or unknown (idiopathic). Fainting can be thought of as occurring when there is a distortion of the normal range between stimulation (E) and inhibition (I) in the brain. This E/I imbalance can result in changes in many levels of brain function, from genetic and subcutaneous signaling to distributed neuronal circuits. Elements that change the E/I balance can be genetic or inherited. Genetic pathologies leading to an attack can occur anywhere from the regional level (abnormal synaptic connectivity in cortical dysplasia) to the receptor level (potassium channel modification in the fight against childhood epilepsy). Similarly, acquired cerebral circulation may alter circuit function (modification of the hippocampal circulatory structure following chronic seizures or head trauma). The developing brain is prone to fainting for a variety of physical reasons. Even in the normal growing brain, pleasurable synaptic activity increases before inhibitory synaptic activation, reaping improved development and production of excretion. Moreover, early in life, the neurotransmitter Gamma-Aminobutyric acid (GABA) causes more attraction than inhibition. This comment explains why the very young brain is at risk of an attack. However, fainting causes less structural damage to the developing brain than to the old brain with a recent explosion of new information about the genetic basis of epilepsy. Both monogenic and polygenic modifications can lead to seizures. Many types of epilepsy have a complex genetic basis with many genetic defects that contribute to a state of altered cellular zeal, causing epilepsy. As genetic information grows it is hoped that treatment-specific interventions can be designed.