Clinical Neuropsychology: Open Access

Review Article Open Access

Types of Seizures and Epilepsy Encountered During Therapies

Lan Xiong*

Department of Psychiatry, University of Montreal, Canada

Abstract

Epilepsy is one of the most common and disabling neurological disorders, but the therapeutic basis for many epilepsy is not understood due to an incomplete understanding of the detailed pathophysiology. This article provides an overview of the clinical aspects of seizures and epilepsy, with the aim of introducing neuroscientists to aspects amenable to scientific investigation. Seizures and epilepsies are defined, diagnostic methods are reviewed, different clinical syndromes are discussed, differential diagnosis, treatment, and prognostic aspects are considered, and neuroscientists can formulate basic and translational research questions increase.

Keywords: Neurological disorder; Seizure; Epilepsy; Diagnosis

Introduction

A "seizure" is a paroxysmal change in neuronal function caused by excessive supersynchronous firing of neurons in the brain. “Epileptic seizure” is used to distinguish between seizures caused by abnormal firing of neurons and non-epileptic events such as psychogenic seizures [1]. Epilepsy is a condition of recurrent seizures without a cause. Epilepsy has many causes, each reflecting an underlying brain dysfunction. Seizures caused by reversible seizures (fever, hypoglycemia, etc.) are not included in the definition of epilepsy as they are transient secondary conditions, not chronic conditions. "Epilepsy syndrome" refers to clinical features with similar seizure type, age of onset, EEG findings, triggers, genetics, natural history, prognosis, and response to antiepileptic drugs (AEDs). “Seizure disorders” should be avoided. Epilepsy is one of the most common neurological disorders, with approximately 50 new cases per 100,000 inhabitants annually [2]. Approximately 1% of the population has epilepsy, and approximately one-third of patients have refractory epilepsy (i.e., seizures uncontrolled by two or more appropriately selected antiepileptic drugs or other treatments). I'm here. About 75% of epilepsy begins in childhood. This reflects the increased susceptibility of the developing brain to seizures. The most recent International League Against Epilepsy (ILAE) Classification of Seizures and Epilepsy (Epilepsy Syndrome), published in 2010, revise the previous classification with now more appropriate terms and concepts. Seizures fall into three categories: generalized seizures, focal (formerly partial) seizures, and epileptic seizures. Focal seizures occur in neural networks confined to a portion of one hemisphere. A generalized seizure starts with a neural network distributed on both sides. A seizure may begin locally and later become generalized [3]. Seizures can occur in cortical or subcortical structures. A detailed medical history, electroencephalographic findings, and supplemental information will often allow your doctor to classify the type of seizure/epilepsy and make an appropriate diagnosis and treatment plan.

Types of Epilepsy

The main subtypes of generalized seizures are absence seizures, generalized tonic-clonic (GTC), myoclonic, and atonic seizures [4, 5]. Absence seizures (previously called minor malformations) are unresponsive to external verbal stimuli and may involve blinking or nodding of the head. GTC seizures (previously known as grand mal) consist of symmetrical jerky movements (twitches followed by rigidity) of all unconscious limbs [6]. Myoclonic seizures consist of sudden, short (“flashes”) movements without apparent disturbance of consciousness. These short, involuntary muscle contractions can affect one or more muscles [7]. Myoclonic seizures can therefore be generalized or focal seizures. In cataplexy, the body loses tone and the head often droops and droops.

The clinical presentation of focal seizures depends on the cortical area affected. For example, focal seizures originating in the occipital lobe may exhibit visual phenomena [8, 9] from the precentral gyrus with rhythmic clonic or tonic motor activity; from the postcentral gyrus with sensory symptoms such as paresthesias. A seizure occurs when consciousness is impaired during a focal seizure, meaning the person cannot respond normally to verbal or tactile stimuli [10].

The origin of the third seizure type category, epileptic seizures, is unknown. Epileptic spasms are manifested by sudden extension or flexion of a limb, held for a few seconds, and then repeated in clusters. Epileptic seizures can occur at any age. It includes a syndrome called infantile spasms (IS) if it occurs in the first year of life [11].

Epilepsy (epileptic syndrome) was classified according to its origin (related to generalization or specific cortical localization) and etiology. H. Is the cause known (syndromic) or unknown (idiopathic)? Here we use the 2010 Revised Guidelines for the Classification of Seizures and Epilepsy [12-14]. The updated system takes into account increased knowledge of structural and genetic causes and includes ictal semiology (type of seizure), syndrome diagnosis (if any), and level of impairment [15]. New classification schemes will continue to evolve as knowledge of epilepsy pathophysiology and genetics develops.

Seizures

Seizures can be thought of as occurring when the normal balance between excitation (E) and inhibition (I) in the brain is disturbed. This E/I imbalance can result from alterations at various levels of brain function, from genes and intracellular signaling cascades to large-scale neural circuits. Factors that alter the E/I balance are genetic or acquired [16]. Genetic pathologies leading to epilepsy range from the circuit

***Corresponding author:** Lan Xiong, Department of Psychiatry, University of Montreal, Canada, E-mail: xionglan@rediff.com

Received: 1-Aug-2022, Manuscript No: cnoa-22-72964; **Editor assigned:** 5-Aug-2022, Pre-QC No: cnoa-22-72964 (PQ); **Reviewed:** 19-Aug -2022, QC No: cnoa-22-72964; **Revised:** 23-Aug-2022, Manuscript No: cnoa-22-72964 (R); **Published:** 30-Aug-2022, DOI: 10.4172/cnoa.1000147

Citation: Xiong L (2022) Types of Seizures and Epilepsy Encountered During Therapies. Clin Neuropsycho, 5: 147.

level (such as abnormal synaptic connections in cortical dysplasia) to the receptor level (such as abnormal γ-aminobutyric acid [GABA] receptor subunits in Angelman syndrome) to abnormal ion channels. It extends to Function (eg, potassium channel mutations in benign familial neonatal epilepsy [BFNE]). Similarly, acquired brain injury can alter circuit function (eg, structural changes in hippocampal circuits following prolonged febrile seizures or head trauma). The developing brain is particularly prone to seizures for a variety of physiological reasons [17]. Even in a normally developing brain, excitatory synaptic functions develop before inhibitory synaptic functions, facilitating excitatory and seizure development. Moreover, the neurotransmitter GABA causes excitation rather than inhibition at a young age. These observations help explain why very young brains are particularly prone to seizures.However, epileptic seizures cause less structural damage to the developing brain than the adult brain. Recently, there has been an explosion of new information on the genetic basis of epilepsy syndromes. Both single- and polygenic mutations can cause epilepsy. Many types of epilepsy have a complex genetic basis, and multiple genetic defects contribute to the altered state of cell excitability that underlies epilepsy [18]. For example, copy number variants that are de novo or inherited >1 kb deletions or duplications are increasingly recognized as causes of genetic alterations in patients with epilepsy. As genetic knowledge expands, there is hope that syndrome-specific therapeutic interventions can be developed [19].

Formerly termed 'syndromic localization-associated' epilepsy syndromes are those in which seizures occur in focal brain regions caused by acquired or congenital lesions. Etiologies include tumors, scarring (eg, hippocampal sclerosis), cortical dysplasia, foramen cysts, and vascular malformations. Seizure semiotics refers to the affected areas of the brain. Seizures often begin locally and then become generalized. Intermittent EEG shows focal spikes, sharp waves, or delays associated with affected areas of the brain. Surgery is considered when imaging findings, EEG evidence of seizure onset, and supporting data (eg, neurobehavioral findings) are consistent. Temporal lobe epilepsy Midtemporal sclerosis syndrome is a related case of structural lesions (hippocampal scarring), where seizures are often uncontrolled and surgery is a viable option. Seizures occur in the medial temporal region with symptoms such as postural changes, reactivity and memory/behavior. Spreading of ictal discharges beyond the hippocampus is common. Seizures often get out of control, and emotional comorbidities are common. If two agents fail, surgical examination should be performed [20]. Large-scale laboratory studies have been conducted to understand the mechanisms of seizure initiation and propagation. Impaired GABAergic inhibition, increased synaptic excitation by axonal sprouting, and altered ion channel distribution and function are all involved in the pathophysiology of temporal lobe epilepsy, and genetic factors may also play a role [21].

Hemispheric childhood epilepsy syndromes

Hemispheric childhood epilepsy syndromes which includes several important childhood epilepsy syndromes affect the entire hemisphere. Rasmussen's encephalitis is a focal encephalitis that affects only one hemisphere and is characterized by progressive hemiplegia, intractable epilepsy (focal seizures that can progress to a persistent state called persistent partial epilepsy), and cognitive decline. cause Rasmussen's encephalitis may have an autoimmune role, but the exact etiology has not been identified. Unilateral lesions may be the result of local disruption of the blood-brain barrier. Neuroimaging shows progressive unilateral cortical atrophy. Another hemispheric syndrome, Sturge-Weber syndrome (SWS; cerebral trigeminal angiomatosis) [22], consists of hemispheric vascular malformations and causes intractable epilepsy and hemiplegia. A mutation in her GNAQ, a gene that controls angiogenesis, was recently identified in SWS. Some experts believe that early surgery (hemispherectomy) improves prognosis for hemispheric epilepsy syndrome. Metabolic, Mitochondrial, and Autoimmune Epilepsy Epilepsy caused by metabolic, mitochondrial, or autoimmune etiologies is increasingly recognized. Alterations in neuronal energy metabolism or expenditure can lead to E/I imbalances and seizures [23]. The role of autoantibodies against various cellular proteins in patients with previously undiagnosed neurological deterioration sheds new light on how epilepsy manifests.

Neonatal seizures

Neonatal seizures, occurring in preterm infants within 30 days of age or <44 weeks of conception, fall into a special class because of their age-specific features, wide range of etiologies, and unique pathophysiology. Its detection is very important as it is a sign of central nervous system dysfunction in newborns. Based on behavioral observations, four types of semiotics of neonatal seizures have been described. Subtle seizures may include repetitive mouth-cheek-tongue movements such as sucking, leg or arm kicking, or eye deviation. Subtle seizures are often associated with severe her CNS damage. Neonatal tonic seizures include postures with intermittent tonic stretches of the arms and legs. They are usually associated with severe brain lesions and are most common in preterm infants. Clonic seizures consist of rhythmic spasms of muscle groups in a focal or multifocal pattern. In multifocal clonic seizures, movements are transferred from one part of the body to another. Partial seizures can result from focal brain abnormalities or injuries, such as: [24] such as perinatal stroke and disorders that diffusely affect the brain. B. Suffocation, metabolic disturbances or infections occur. As a result of immature myelination and cortical tissue, the neonatal brain is unable to sustain generalized epileptiform discharges, thus GTC and absence seizures do not occur. Simultaneous video and EEG monitoring can help distinguish behaviors with EEG correlation ("seizures") from behaviors without associated EEG changes [25]. Focal-clonic seizures are most correlated with EEG ictal abnormalities. Many behaviors that are considered subtle seizures on clinical grounds (e.g., chewing and kicking movements) have no associated EEG abnormalities, suggesting that these behaviors are not epileptic in nature. Subtle or tonic seizures may represent epileptic seizures due to brainstem dysfunction or deep subcortical structures that cannot be recorded on surface EEG.

Neonatal EEG is usually not specific to any particular etiology, but can provide clues to the severity and time course of CNS damage. Epileptic discharge often occurs without overt clinical symptoms ("disconnection" of electrocardiogram and clinical seizure). EEG background patterns and sleep-wake cycles are particularly important for prognosis. Amplitude integrated EEG (aEEG) is a new technique that allows continuous sampling of a limited number of EEG channels at the bedside. aEEG has proven to be very reliable in documenting likely seizure events. Determining the etiology of neonatal seizures is important because cause drives treatment and is highly correlated with outcome. The main causes of neonatal seizures are hypoxic-ischemia (H-I), hypocalcemia, hypoglycemia, hyponatremia, intracranial hemorrhage, infections, congenital malformations, genetic factors, hereditary metabolic disorders, and It's drug withdrawal. H-I, which usually occurs before birth, is the most common cause of neonatal seizures. Decisions to treat infants with recurrent seizures are based on seizure duration and frequency, associated autonomic dysfunction, etiology, and EEG abnormalities [26]. If attacks are brief and not accompanied by autonomic instability, treatment may be delayed or treated with short-acting benzodiazepines. Conversely, infants with

frequent seizures, especially those with obstructed ventilation, require prompt and vigorous treatment. Phenobarbital, the primary drug used to treat neonatal seizures, is either effective for more than 50 minutes but less than 15 minutes or has a focal component (e.g., begins on one side of the body) with lateral displacement of the eyeball), or recur within 24 hours. Simple febrile seizures require no treatment, and most complex febrile seizures do not. A child who has had one simple febrile seizure has a 33% chance of having another febrile seizure followed by a fever. If a child has had two simple febrile seizures her second, there is a 50% chance that she will have a third febrile seizure.

The risk of additional febrile seizure recurrence is highest if the child had the first febrile seizure before 12 months of age or had a family history of febrile seizures. The risk of developing febrile seizures (epilepsy) is even more worrisome. A significant proportion of adults with temporal lobe epilepsy (caused by medial temporal lobe sclerosis) had persistent febrile seizures during childhood [27]. After a simple febrile seizure, the risk of developing epilepsy later in life is slightly higher than in the general population, about 2%. The risk of developing epilepsy after a febrile seizure varies as much as 9% and is greatest if the child has pre-existing neurological deficits). An ongoing multicenter study (FEBSTAT) is investigating the consequences of febrile status epilepticus in a large cohort using longitudinal clinical and MRI data. A full understanding of febrile seizures will also require animal models to gain better insight into the effects of hyperthermia and febrile immunological changes in the developing brain.

Conclusion

This review introduced the concept of clinical epilepsy to enable neuroscientists to assess conditions in the field and formulate relevant research questions. A number of clinical mysteries for researchers to consider were discussed in a recent opinion paper. Among them, the following issues were deemed ripe for neuroscientific investigation. Role of genes and acquired factors in seizure predisposition. To know how epilepsy develops in an otherwise normal brain and how the consequences of seizures can be prevented (e.g. after brain injury (epileptogenesis, neuroprotection) how to properly identify and identify circuits at risk for surgical intervention, how to predict (and thereby prevent) the occurrence of seizures. Optimization of medication for specific age groups and epileptic syndromes; novel methods of drug delivery to vulnerable circuits. Interventions to reduce postictal and interictal comorbidities that dominate a patient's daily functioning but disproportionately affect quality of life.

References

1. Arciniegas BD (2015) Psychosis. Continuum (Minneap Minn) 21: 715–736.
2. https://www.psychiatry.org/psychiatrists/practice/dsm
3. https://apps.who.int/iris/handle/10665/37958
4. Poole R, Brabbins C (1996) Drug induced psychosis. Br J Psychiatry 168: 135-138.
5. Liz P, Martina K, Sarah N, Mary W, Anthony PM (2007) Researching recovery from psychosis: A user-led project. Psychol Bull 31: 55-60.
6. https://psycnet.apa.org/record/2008-05769-022
7. British Psychological Society (2017) Understanding psychosis and schizophrenia. Leicester: British Psychological Society.
8. Sue D (2010) Microaggressions in everyday life: Race, gender, and sexual orientation. New Jersey: John Wiley and Sons.
9. Warren K (2003) Exploring the Concept of Recovery from the Perspective of People with Mental Health Problems. Br J Soc Work 34: 1055-1070.
10. https://www.worldcat.org/title/roads-to-recovery-how-people-with-mental-health-problems-recover-and-find-ways-of-coping/oclc/173238361
11. Johnstone L, Boyle M (2018) The Power Threat Meaning Framework: An Alternative Non-diagnostic Conceptual System. J Humanist Psychol.
12. Higgins L, Zheng M (2002) An introduction to Chinese psychology—Its historical roots until the present day. J Psychol 136: 225-239.
13. Bürgy M (2012) The origin of the concept of psychosis: Canstatt 1841. Psychopathology 45: 133–134.
14. Read J, Bentall PR, Fosse R (2009) Time to abandon the bio-bio-bio model of psychosis: Exploring the epigenetic and psychological mechanisms by which adverse life events lead to psychotic symptoms. Epidemiol Psychiatr Sci 18: 299-310.
15. Lim A, Hoek WH, Blom JD (2015) The attribution of psychotic symptoms to jinn in Islamic patients. Transcult Psychiatry 52: 18-32.
16. Al-Krenawi A, Graham RJ (1997) Spirit possession and exorcism in the treatment of a Bedouin Psychiatric Patient. Clin Soc Work J 25: 211–222.
17. https://webarchive.nationalarchives.gov.uk/20180328130852tf_/http://content.digital.nhs.uk/catalogue/PUB21748/apms-2014-full-rpt.pdf/
18. Brown S, Kim M, Mitchell C, Inskip H (2010) Twenty-five year mortality of a community cohort with schizophrenia. Br J Psychiatry 196: 116–121.
19. Hayes J, Marston L, Walters K, King M, Osborn D (2017) Mortality gap for people with bipolar disorder and schizophrenia: UK-based cohort study 2000–2014. Br J Psychiatry 211: 175-181.
20. Hayes JF, Miles J, Walters K, King M, Osborn DPJ (2015) A systematic review and meta□analysis of premature mortality in bipolar affective disorder. Acta Psychiatrica Scandinavica 131: 417-425.
21. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/774680/Psychosis_data_report.pdf
22. Kane J (1996) Treatment-resistant schizophrenic patients. J Clin Psychiatry 57: 35-40.
23. Iversen TSJ, Steen N E, Dieset I, Hope S, Mørch R, et al . (2018) Side effect burden of antipsychotic drugs in real life–Impact of gender and polypharmacy. Prog Neuro-Psychopharmacol Biol Psychiatry 82: 263-271.
24. Lawrence C, Jones J, Cooper M (2010) Hearing voices in a non-psychiatric population. Behav Cogn Psychother 38: 363.
25. Maarten B , Myin GI, Manon H, Rob B, Wilma V, et al. (2003) When does experience of psychosis result in a need for care? A prospective general population study. Schizophrenia Bulletin 29: 349-358.
26. Garety P (2003) The future of psychological therapies for psychosis. World Psychiatry 2: 147–152.
27. https://www.nice.org.uk/guidance/cg178