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Review Article

Paediatric Epilepsy: A Comprehensive Overview of Diagnosis, Treatment and Management

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ABSTRACT

Seizures are characterized as a transient episode of symptoms arising from abnormal, excessive, or synchronized firing of brain neurons that is accompanied by sudden, involuntary skeletal muscle contractions. Among the most common neurological diseases in children, epilepsy is particularly prevalent during the first year of life. Premature birth, neurological comorbidity, positive family history, fever, infections, maternal alcohol misuse, maternal complications and smoking during pregnancy are among the risk factors associated with the beginning of seizures, particularly in children. Children's neurological morbidity is primarily due by epilepsies. About 5 out of every 1,000 children will have epilepsy in any given year, while the average annually rates of new cases of the disorder being around 5-7 cases per 10,000 children from between the ages of one and fifteen. The prevention of epilepsy and seizures requires early diagnosis, treatment and specialized medical care. A comprehensive investigation that includes a detailed medical history, physical examination, and neuroimaging investigations is necessary for the diagnosis of epilepsy in children. Individualized treatment plans are developed for children with epilepsy, taking into account the child's general health, underlying etiology, and seizure type and frequency. Antiepileptic medications (AEDs) are commonly used as first-line therapy based on the type of seizure. In some instances, alternative therapies like the ketogenic diet therapy may be taken into consideration when patients are not responding to medication.

Keywords: Seizures, Premature birth, Epilepsy, Neurological morbidity, Neuroimaging, Antiepileptic drugs, Ketogenic Diet.

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INTRODUCTION

Epilepsy has been the most prevalent neurological diseases globally.[1] It impacts over 50 million individuals globally and is characterized by the transient onset of symptoms brought on by abnormal neuronal discharges in the cerebral cortex.[2] A child suffering epilepsy develops recurrent seizures over time as a result of the brain disorder. The brain is made up of nerve cells; they exchange electrical signals with one another. A seizure develops when abnormal electrical signals from several brain regions collapse, affecting regular neural activity.[3] Up to 0.5% of children in the group of children have epilepsy, therefore among the most prominent neurological illnesses. It is a childhood-predominant syndrome, with one in 150 children diagnosed during the first ten years of life.[2] The International League against Epilepsy (ILAE), epilepsy was redefined in 2017 to mean that it satisfied one of three requirements for inclusion: 1) a minimum of two spontaneous seizures that occur Over 24 hr; 2) An unprovoked seizure and the possibility of more seizures over the course of ten years that follow that is equivalent at least 60% of the overall recurrence risk; 3) A classification of epileptic syndrome.[4]

In whole population, the largest age-specific occurrences (460 per 100,000) are seen among adults over 65 and children under 5 years.[5] As one of the most prevalent neurological conditions affecting children is epilepsy has an incident rate of 33.3 to 82 occurrences per 100,000 annually. The first year after birth marks the peak of incidence, which declines during adolescence.[6] These people have a significant risk of early death as well as cognitive and behavioral comorbidity.[5]

Risk factors

Risk factors are conditions that are associated with a greater chance of developing epilepsy; these conditions are different for later-life epilepsy than for childhood epilepsy. Some of the known risk factors are head injuries, fetal trauma, infections of the Central Nervous System (CNS), and developmental problems, neoplastic diseases and genetic factors. Many occurrences of epilepsy are caused by genetic factors.[7] Preterm delivery (> 37 weeks), smoking throughout pregnancy, mother epilepsy, eclampsia,

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pregnant metrorrhagia and maternal infection regardless of term, were prenatal risk factors for epilepsy. Epilepsy risk is higher in preterm populations due to white matter gliosis, hypoxic-ischemic brain damage, hippocampal sclerosis, decreased brain structure development, and increased infection risk in preterm populations. Eclampsia is a significant condition that can progress to epilepsy in young people via a variety of paths. In reality, women may suffer hypoxia, which can increase the incidence of epilepsy and interfere with the unborn brain's normal growth. Head trauma, cord prolapse, and extended labor (> 6 hr) were correlated with a higher prevalence of epilepsy in newborns. In terms of postnatal variables, low birth weight (less than 2.5 kg), delivery difficulties, male young children are more likely to develop epilepsy due to variations in astrocyte structure and brain connection. Women's cortices, caudate nuclei and hippocampal regions are larger, while men had a larger thalamus and amygdala, influenced by steroid hormones. These were associated with a considerably higher risk of epilepsy. Birth complications like feeding difficulties, crying, respiratory issues, infections, and Apgar<6 significantly increase the risk of developing epilepsy.[8] Additionally, low weight at birth and a short the gestational period at delivery is linked to a higher chance risk of getting epilepsy in childhood and into puberty. Even children whose birth weight falls within the normal range are at heightened risk for epilepsy due to intrauterine growth restriction.[9]

Child outcomes

Approximately 30% of children with epilepsy have cognitive or behavioural problems. When children younger than five exhibit significant delays in two or more aspects of development, involving speech/language, motor skills, daily tasks, cognitive abilities, and social/emotional capabilities this phenomenon is referred to as Global Developmental Delay (GDD). It is suggested for children with epilepsy have frequent GDD screening in order to facilitate early intervention and to optimize their prospects for social, professional, and academic success.[10]

Etiology

In ILAE-2017, the aetiology was divided into four categories: structural, infectious, perhaps genetic, and unknown.[11]

Structural

Certain structural factors may significantly raise the likelihood of developing epilepsy. Acquired (such stroke, trauma, infection, and immune-mediated) and congenital (like cortical dysplasia and tuberous sclerosis) are the two categories of structural etiologies. Seizure types and natural history are significantly connected with structural etiologies like persistent mesial temporal lobe seizures.

Genetic

The term "genetic etiology" refers to epilepsy in which seizures as main symptom that is brought on by a known or suspected genetic defect. Generalized genetic epilepsy syndromes, commonly referred to as idiopathic generalized epilepsies, fall under this category. These include epilepsy with tonic-clonic seizures alone, childhood absence epilepsy, juvenile absence epilepsy, and juvenile myoclonic epilepsy. Heritability is strongly suggested by twin and family studies. CDKL5, ARX mutations, Dravet syndrome, protocadherin 19 female-limited epilepsies, and Down syndrome are other genetic reasons of intellectual impairment and inadequate seizure control.

Metabolic

Metabolic etiology refers to a patient's established metabolic abnormality that increases their likelihood of getting epilepsy significantly. Glucose transporter deficits, creatine deficiency diseases, and mitochondrial cytopathies are a few examples.

Infectious

Infectious CNS infections can induce both acute seizures and epilepsy, making them a leading cause of epilepsy globally. Meningitis and encephalitis are examples of acute CNS illnesses that should not be classified as seizures. Some regions of the world have higher rates of congenital illnesses like Zika virus and CMV, as well as certain infections like neurocysticercosis, TB, HIV, cerebral malaria, and cerebral toxoplasmosis.

Unknown

The word "unknown" is replaced with "cryptogenic," which just indicates that it is yet unknown what the primary cause is exactly. Normal imaging epilepsies without any known immunological, metabolic, genetic or viral cause fall into this category.[12]

Pathophysiology

Deficits in neuronal inhibition, particularly the Aminobutyric acid (GABA) deficit, or changes in GABA function that result in prolonged, high-intensity stimulation can both cause seizures. Alpha-amino-3-hydroxy-5-methyl-4-isoxazole-propionic acid and N-Methyl-D-Aspartate (NMDA), both glutamate receptors, have been implicated in seizure physiopathology in experimental animal models. Teenagers exhibit a higher risk of having febrile seizures because of their increased exposure to respiratory high tract infections and infection by viruses. When febrile seizures start and an increase in neuronal stimulation can be brought on by inflammatory mediators such as IL-1. Serious pathological conditions such as meningitis, encephalitis, and cerebral abscess may be indicated by febrile seizures. According to recent research, viral infections like Rubi virus and HHSV-6 might contribute to the genesis of seizures.[13]

Diagnosis

Laboratory tests, neurologic exams, and EEG imaging methods are all necessary for epilepsy and seizures.[14] EEG is triggered by photic stimulation and hyperventilation and able to identify

aberrant electrical activity, including localized or generalized epilepsy.[15] Photic stimulation can cause paroxysmal epileptiform activity.[16] Concurrent video-EEG monitoring can distinguish enhance the diagnosis' precision by distinguishing between epileptic and non-epileptic episodes.[14] For emergency patients experiencing their first seizures,[17] MRI and CT are advised and are helpful worldwide,[18] but may result in small lesions losing blood.[19] Additional MRIs are required for children who have focal seizures before the age of twelve months.[20] Post-processing methods such as artificial intelligence and computational analysis,[21] can enhance the identification of faint FCDs in negative MRIs.[22] Pre-surgical evaluations for seizure diagnosis use PET and SPECT,[23] which

AED	Paediatric dosing	Mechanism of action	Seizure type
Carbamazepine	10–35 mg/kg/day	Sodium channel blocker targeting voltage gated Na channels.	Focal seizures.
Clobazam	<2yo: 0.5–1 mg/kg/day >2yo: 10–20 mg/day	Activation of $GABA_A$ receptors.	Add on AED for seizures associated with LGS.
Ethosuximide	15–40 mg/kg/day	Targets low voltage Ca channels.	Absence Seizures.
Eslicarbazepine	400–1,200 mg/day based on weight	Sodium channel blocker targeting voltage-gated Na channels.	Focal seizures.
Felbamate	15–45 mg/kg/day	Thought to act on voltage-gated Na channels, high voltage Ca channels, and $GABA_A$ receptors.	Focal seizures, drop attacks in LGS.
Lacosamide	11–29 kg: 6–12 mg/kg/day 30–49 kg: 4–8 mg/kg/day	Sodium channel blocker targeting voltage-gated Na channels.	Focal seizures.
Lamotrigine	Monotherapy: 4.5–7.5 mg/kg/day Add on: 5–15 mg/kg/day	Targets voltage-gated Na channels and possibly acts on high voltage Ca channels.	Focal and most generalised seizures.
Levetiracetam	20–60 mg/kg/day	Targets synaptic vesicle protein 2A, exact mechanism is unknown.	Most seizure types.
Oxcarbazepine	30–40 mg/kg/day	Sodium channel blocker targeting voltage-gated Na channels.	Focal seizures.
Phenytoin	4–8 mg/kg/day, up to 600 mg/day for adolescents.	Sodium channel blocker targeting voltage-gated Na channels.	Focal seizures and generalised seizures.
Phenobarbital	Infants: 5–6 mg/kg/day Children <5: 6–8 mg/kg/day	Activation of $GABA_A$ receptors.	Most seizure types.
Primidone	10–25 mg/kg/day	Activation of $GABA_A$ receptors.	Most seizure types.
Rufinamide	45 mg/kg/day	Sodium channel blocker targeting voltage-gated Na channels.	Focal seizures, drop attacks in LGS.
Tiagabine	<12yo: 4–8 mg/kg/day >12yo: max 32 mg/day	Increases GABA concentration by blocking synaptic GABA reuptake.	Focal seizures.
Topiramate	5–9 mg/kg/day	Thought to act on voltage-gated Na channels, HVA Ca channels, and $GABA_A$ receptors.	Most seizure types.
Valproate	30–60 mg/kg/day	Thought to target Na and Ca channels.	All seizure types.
Vigabitran	Infants: 50–150 mg/day Older children: 500–3,000 mg/day	Increases GABA concentration by inhibition of the mitochondrial enzyme GABA-transaminase.	Infantile spasms, focal seizures.
Zonisamide	5–8 mg/kg/day	Targets voltage-gated Na channels and possibly acts on low voltage Ca channels.	

Figure 1: Commonly used antiepileptic drugs in paediatric patient.[36]

provide additional localizing information and reveal decreased glucose metabolism in the seizure onset zone.[24] SPECT by itself, however, provides less information, particularly when extra temporal lobe epilepsy is present.[25] The spatial distribution of the functional cortex and language-related regions can be ascertained by utilizing functional imaging techniques like fMRI or MEG, which facilitates pre surgical evaluation and resection planning.[26] By improving MRI's ability to detect subtle lesions, functional multimodal imaging helps patients live better lives.[18]

Treatment

Non Pharmacological Treatment

First Aid

Helping the patient manage the situation during an epileptic seizure is known as first aid treatment. Because of the common misconception, people appear afraid when they witness someone having an epileptic seizure. Since studies have shown that patients and their families can easily control or manage epileptic seizures with the correct guidance and training, it is more important than ever to stay calm and help the sufferer (Cross *et al*., 2022). Since epileptic seizures are not emergencies, there is no need to call for an ambulance. Consequently, the best way to help patients manage their seizures on their own and boost their confidence is through self-management training (Wiles *et al*., 2023).[27]

Ketogenic Diet

Throughout history, diets have been used to manage seizures. However, it was the first documented in the early 1920s, the nutritional diet was used as a therapeutic treatment for epilepsy. Dietary treatments for DRE include Low Glycaemic Index Therapy (LGIT), Modified Atkins diet (MAD), and the traditional Ketogenic Diet (KD). KD and MAD diets are high in fat, but LGIT focuses mostly on consuming low-glycaemic foods that are low in carbohydrates. These diets' compositions are explained below.[28] A diet consisting of 90% fat, 7% protein, and 3% carbohydrates is the traditional ketogenic diet,[29] that produces metabolic changes associated with the starvation state. A ketogenic diet, sometimes known as the "keto diet," may be an option for certain kids to manage their seizures if medicine isn't enough.[28]

Neurostimulation

This treatment involves the nervous system receiving tiny electric currents via a gadget. Three forms of neurostimulation are now used to treat epilepsy they are; Vagus nerve stimulation, Responsive neurostimulation, Deep brain stimulation.[30] The FDA, approved left VNS in 1997 as an adjuvant therapy for adults and children 12 years of age and older who have refractory partial onset seizures.[31]

Pharmacological Treatment

There are currently over 20 Antiepileptic Drugs (AED) available, and selecting the right medicine primarily relies on age and seizure type. The first-line accepted treatment is AED therapy,[32] both for symptomatic myoclonic seizures and generalized tonic-clonic seizures were best administrated with sodium valproate. Primary first-line therapies for complex partial seizures were carbamazepine and oxcarbazepine, with valproate also being recommended.[33] Although using AEDs alone or in combination, approximately 20% of children with epilepsy will still have seizures.[34] Patients on their first AED, about half of the patient's experience seizure-free status, and roughly two-thirds will experience seizure-free status. This leaves about 30% of people with epilepsy who are not responding to medicines.[35] Some of the Antiepileptic drugs are summarised in Figure 1 with their mechanism of actions and dosing in paediatrics.

CONCLUSION

Seizures in children are a serious medical issue that has to be diagnosed and treated quickly. Finding the underlying cause of the seizures requires an understanding of the several etiologies, which range from neurological problems to metabolic issues. Children's risk of seizures is further influenced by risk variables such age, genetic predisposition, and prior neurological history. Treatment choices depend on an accurate diagnosis, which frequently requires imaging, laboratory testing, and clinical examination. The main goals of treatment are to manage seizures and stop them from happening again by using medicine, changing one's lifestyle, and occasionally having surgery. Children with seizures require a comprehensive approach to treat because early identification and intervention can greatly enhance results.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

AEDs: Anti Epileptic Drugs; **CNS:** Central nervous system; **CT:** Computed Tomography; **CMV:** Cytomegalovirus; **DRE:** Drug related Epilepsy; **EEG:** Electroencephalography; **FDA:** Food and Drug Administration; **FCD:** Focal Cortical Dysplasia; **FMRI:**

Functional Magnetic Resonance Imaging; **GABA:** Gamma aminobutyric acid; **GDD:** Global developmental delay; **HIV:** Human influenza virus; **ILAE:** International League Against epilepsy; **IL:** Interleukins; **KD:** Ketogenic Diet; **LGIT:** Low glycaemic index therapy; **MAD:** Modified Atkins diet; **NMDA:** N-Methyl-D-aspartate; **PET:** Positron Emission Tomography; **SPECT:** Single Photon Emission Computed Tomography; **TB:** Tuberculosis; **VNS:** Vagus Nerve Stimulation.

REFERENCES

1. Pratibha Singhi and Ajay Gupta;Epilepsy in Children-Important Facets https://doi.org/10.1007/s12098-021-03682-z. The Indian Journal of Paediatrics (October 2021) 88(10):991-2
2. Ester Maria de Almeida Costa, Brenda Martins da Silva, Iris Maria Thomazini, Isabella Otoni Porto, Mariana Santos Furtado, Carlos Umberto Pereira, Nicollas Nunes Rabelo Epilepsy in childhood: an update on management. DOI: 10.46900/apn. v5i1.158; 08 January 2023.
3. Seizures and Epilepsy in Children [Internet]. Johns Hopkins Medicine Health Library. 2019. Available from: https://www.hopkinsmedicine.org/health/conditions-and-diseases/epilepsy/seizures-and-epilepsy-in-children
4. Patel P, Moshé SL. The evolution of the concepts of seizures and epilepsy: What's in a name? Epilepsia Open. 2020;5(1):22-35. doi: 10.1002/epi4.12375. PMID: 32140641; PMCID: PMC7049807.
5. Symonds JD, Elliott KS, *et al*, Early childhood epilepsies: epidemiology, classification, aetiology, and socio-economic determinants. Brain. 2021;144(9):2879-91. Doi: 10.1093/brain/awab162. PMID: 34687210; PMCID: PMC8557326.
6. Fine A, Wirrell EC. Seizures in Children. Paediatrics in Review [Internet]. 2020;41(7):321-47.
7. Asadi-Pooya AA, Hojabri K. Risk factors for childhood epilepsy: a case-control study. Epilepsy and Behavior. 2005;6(2):203-6.
8. Imen Ketata, Emna Ellouz, Rahil Mizouri. Impact of prenatal, neonatal, and postnatal factors on epilepsy risk in children and adolescents: a systematic review and meta-analysis. Acta Epileptologie. 2024;6(1).
9. Sun Y, Vestergaard M, Pedersen CB, Christensen J, Basso O, Olsen J. Gestational Age, Birth Weight, Intrauterine Growth, and the Risk of Epilepsy. American Journal of Epidemiology [Internet]. 2007 Nov 27 [cited 2022 Oct 18];167(3): 262-70. Available from: https://academic.oup.com/aje/article/167/3/262/132284 10
10. Albaradie R, Habibullah H, Mir A, Alshammari AK, Alajmi MS, Alsubaie FA, *et al.* The prevalence of seizures in children with developmental delay. Neurosciences. 2021;26(2):186-91.
11. Egesa IJ, Newton CRJC, Kariuki SM. Evaluation of the International League Against Epilepsy 1981, 1989, and 2017 classifications of seizure semiology and etiology in a population-based cohort of children and adults with epilepsy. Epilepsia Open. 2021.
12. Nickels KC, Noe K. Etiology and Pathology of Epilepsy. Epilepsy. 2021;23-35.
13. Minardi C, Minacapelli R, Valastro P, Vasile F, Pitino S, Pavone P, Astuto M, Murabito P. Epilepsy in Children: From Diagnosis to Treatment with Focus on Emergency. J Clin Med. 2019;8(1):39. Doi: 10.3390/jcm8010039. PMID: 30609770; PMCID: PMC6352402.
14. Stafstrom CE, Carmant L. Seizures and Epilepsy: An Overview for Neuroscientists. Cold Spring Harbor Perspectives in Medicine. 2015;5(6):a022426-6.
15. Schuchmann S, Schmitz D, Rivera C, Vanhatalo S, Salmen B, Mackie K, *et al.* Experimental febrile seizures are precipitated by a hyperthermia-induced respiratory alkalosis. Nature Medicine. 2006;12(7):817-23.
16. Verrotti A, Beccaria F, Fiori F, Montagnini A, Capovilla G. Photosensitivity: epidemiology, genetics, clinical manifestations, assessment, and management. Epileptic Disorders. 2012;14(4):349-62.
17. Harden CL, Huff JS, Schwartz TH, Dubinsky RM, Zimmerman RD, Weinstein S, *et al.* Reassessment: Neuroimaging in the emergency patient presenting with seizure (an evidence-based review): Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology. 2007;69(18):1772-80.
18. Cendes F, Theodore WH, Brinkmann BH, Sulc V, Cascino GD. Neuroimaging of epilepsy. Handbook of clinical neurology. 2016;136:985-1014.
19. Bernasconi A, Cendes F, Theodore WH, Gill RS, Koepp MJ, Hogan RE, *et al.* Recommendations for the use of structural magnetic resonance imaging in the care of patients with epilepsy: A consensus report from the International League Against Epilepsy Neuroimaging Task Force. Epilepsia. 2019;60(6).
20. Nascimento FA, Friedman D, Peters JM, Bensalem-Owen MK, Cendes F, Rampp S, *et al.* Focal epilepsies: Update on diagnosis and classification. 2023;25(1):1-17.
21. Wang I, Alexopoulos A. MRI post processing in presurgical evaluation. Current Opinion in Neurology. 2016 [cited 2020 Feb 23];29(2): 168-74.
22. Ganji Z, Hakak MA, Zamanpour SA, Zare H. Automatic Detection of Focal Cortical Dysplasia Type II in MRI: Is the Application of Surface-Based Morphometry and Machine Learning Promising? Frontiers in Human Neuroscience. 2021;15.
23. Niu N, Xing H, Wu M, Ma Y, Liu Y, Ba J, *et al.* Performance of PET imaging for the localization of epileptogenic zone in patients with epilepsy: a meta-analysis. European Radiology. 2021;31(8):6353-66.
24. Stamoulis C, Verma N, Himanshu Kaulas, Halford JJ, Duffy FH, Pearl PL, *et al.* The promise of subtraction ictal SPECT co-registered to MRI for improved seizure localization in paediatrics epilepsies: Affecting factors and relationship to the surgical outcome. Epilepsy Research. 2016 [cited 2025 Jan 5];129: 59-66.
25. Kim S, Mountz JM. SPECT Imaging of Epilepsy: An Overview and Comparison with F18 FDG PET. International Journal of Molecular Imaging. 2011; 2011:1-9.
26. Schmid E, Thomschewski A, Taylor A, Zimmermann G, Kirschner M, Kobulashvili T, *et al.* Diagnostic accuracy of functional magnetic resonance imaging, Wada test, magnetoencephalography, and functional transcranial Doppler sonography for memory and language outcome after epilepsy surgery: A systematic review. Epilepsia. 2018;59(12):2305-17.
27. Elsadek Hel. Paediatric Epilepsy and its Health Conditions in UAE: A Review Article. American Journal of Medical Science and Innovation. 2024;3(1):35-42.
28. Wilder RM. The effects of ketonemia on the course of epilepsy. Mayo Clin Proc. 1921;2:307-8.
29. Koss off, *et al.* Optimal clinical Management of children receiving Dietary therapies for epilepsy: Updated recommendations of the International ketogenic diet study Group. Epilepsia Open. 2018;3(2)175-92.
30. Epilepsy in children: Types, symptoms, diagnosis, and treatment [Internet]. www.medicalnewstoday.com.2019.Availablefrom:https://www.medicalnewstoday.com/articles/327288#outlook
31. Morris GL 3rd, Gloss D, Buchhalter J, Mack KJ, Nickels K, Harden C. Evidence-based guideline update: vagus nerve stimulation for the treatment of epilepsy: report of the Guideline Development Subcommittee of the American Academy of Neurology. Neurology. 2013;81:1453-9.
32. Sarah Rubenstein1, Andrew Levy2; Seizures in Childhood: Aetiology, Diagnosis, Treatment, and What the Future May Hold. EMJ Neurol. 2019;7[1]:62-70.
33. Wheless JW, Clarke DF, Arzimanoglou A, Carpenter D. Treatment of pediatric epilepsy: European expert opinion, 2007. Epileptic Disorders: International Epilepsy Journal with Videotape [Internet]. 2007 [cited 2022 Nov 1];9(4): 353-412. https://doi.org/10.1684/epd.2007.0144;Availablefrom:https://pubmed.ncbi.nlm.nih.gov/18077226/
34. Wirrell EC. Predicting pharmacoresistance in paediatric epilepsy. Epilepsia. 2013;54 (Supply 2):19-22.
35. Perucca P, *et al.* The management of Epilepsy in children and adults. Med J Aust. 2018;208(5):226-33.
36. Sara Rubenstein1, Andrew Levy2 Seizures in Childhood: Aetiology, Diagnosis, Treatment, and What the Future May Hold,2019.

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