Psychiatric presentation of childhood epilepsy: Case series and review

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ABSTRACT

Childhood-onset epilepsy has a varied presentation and may have different etiological factors. A multiaxial diagnostic approach should be used before making treatment and management decisions for any individual patient. It is widely accepted that distinction among primary psychiatric disorders, epilepsy, and nonepileptic seizures is a challenge for physicians. This case series demonstrated the identification of three atypical presentations of seizures in children on the basis of detailed history taking and electroencephalogram findings, despite having normal findings in neurological examination and magnetic resonance imaging. We report three rare cases of atypical presentation in epilepsy in patients with symptoms of episodic hallucinations, rage attacks, and secondary enuresis. Clinically, the diagnosis of epilepsy can be strengthened by paying sufficient attention to detailed history and symptom spectrum of partial epilepsy.

Key words: Enuresis, epilepsy, hallucinations, rage attacks

Introduction

Epilepsy has been associated with significant morbidity and mortality and poor quality of life and is also responsible for 10% of the global burden of brain disorders.[1,2] Epilepsy affects approximately 80 million people throughout the world, and among them, about 10.5 million are children <15 years.[3] In the US alone, about 2.9 million adults and children are afflicted by epilepsy, having an economic impact amounting to $15.5 billion.[4] While epilepsy occurs in 0.5%–2% of the general population, the incidence of epilepsy in childhood is more than twice that in the adult population.[5,6] Clinicians and researchers still have a limited understanding regarding the clinical presentation and etiology of epilepsy, despite having such a huge burden on the population. Childhood-onset epilepsy has a varied presentation and may have different etiological factors. Due to the atypical presentation, it is very common for a clinician to miss some of the clinical features of epilepsy.

In this case series, we present three cases of epilepsy who presented to our OPD with atypical symptoms of childhood simple partial seizure, i.e., episodic hallucinations, rage attacks, and secondary enuresis. There can be other atypical presentations also such as episodic nervousness with sensory, motor, and autonomic symptoms in some patients.[5] Detailed history taking is essential in the diagnosis of atypical simple partial seizures.

Case 1 – Epilepsy Presenting as “Episodic Hallucinations”

M, 12-year-old, Hindu male child, studying in Class 6, belonging to a middle socioeconomic status, nuclear family,
rural background, presented in the Child and Adolescent Psychiatry OPD, Department of Psychiatry, PGIMER, Dr. RML Hospital, New Delhi, with chief complaints of episodic hallucinations for last 2 years.

Exploration of history revealed that the boy was apparently well adjusted to his personal and social life 2 years back. Since last 2 years, his father reported that he started complaining of hearing voices, which lasted for 2–3 min. The episodes were sudden in onset, with no precipitating and aggravating factors. In the beginning, the frequency of episodes used to be only 3–4/month, but gradually increasing to 1–2 times a day from last 4 months. Patient’s family members also observed the boy had a vacant stare along with muttering to himself during those episodes. There were also some other episodes when he would suddenly get startled and show fearful behavior. He would start crying suddenly without any apparent reason and would ask his parents to stop the voices. When queried by his father, the boy would report that “some male voice was bothering him.” The patient was not able to give the details of the voices, but used to get distressed during those episodes. He was apparently asymptomatic during the interepisodic period, except for some little apprehension regarding voices. He even stopped going to school from last 3 months due to increased frequency of episodes.

There was no history suggestive of any other symptom of epilepsy. His past history, personal history, and premorbid temperament were noncontributory. Detailed child psychiatry assessment using K-SADS did not reveal any significant symptom suggestive of attention deficit hyperactivity disorder or any other childhood psychiatric illness except for mild apprehension regarding the voices. A detailed neurological examination and routine blood and urine investigations did not reveal any abnormality. The patient’s old treatment records reported a diagnosis of psychosis not otherwise specified (NOS), and he was prescribed olanzapine 5 mg once daily in the past. However, there was no response even after 2 months of continuous treatment. Magnetic resonance imaging (MRI) brain scan conducted 3 months back also did not reveal any significant abnormality. His mental status examination revealed no other significant psychopathology except for episodic hallucinations which were nonpervasive in nature. On the basis of history, mental status examination, neurological examination, psychological evaluation, and appropriate laboratory tests, a differential diagnosis of psychosis NOS and complex partial seizures was kept. An electroencephalogram (EEG) was advised to rule out the differential diagnosis. The EEG showed focal spike and wave discharges in left mid temporal leads. On the basis of EEG findings, we confirmed the diagnosis of complex partial seizures. The patient was started on carbamazepine and dose was gradually increased to 400 mg/day over a period of next 4 weeks as per his body weight. Within few weeks of starting the same, the patient showed significant improvement and his hallucinations stopped. He joined the school after 1 month of the start of treatment and is currently well maintained at a dose of 400 mg of carbamazepine since last 6 months.

**Case 2 – Simple Partial Seizure Presenting as “Rage Attack”**

K, 11-year-old, Hindu male child, studying in Class 5, belonging to a middle socioeconomic status, resident of Delhi, living in a joint family, presented in the Child and Adolescent Psychiatry OPD, Department of Psychiatry, PGIMER, Dr. RML Hospital, New Delhi, with chief complaints of episodic “rage attacks” from last 1 year. He was referred by the school authority for detailed psychiatric evaluation.

The patient was apparently asymptomatic 1 year back and was well adjusted to his personal and social life when he showed a “rage attack” lasting for not more than 5 min. The episode was sudden in onset without any precipitating or aggravating factor. During the episodes, he suddenly became very violent and started hitting others and breaking things which were within his reach. He would shout and use abusive languages. The violent behavior was unprovoked and nondirectional. There were no apparent reasons for such behavior as reported by family members. During such episodes, the patient would not respond to any verbal command and would keep on moving around in an abnormal way. It would be difficult to control him and lot of force was needed to control him. He would calm down by himself after around 5 min. He also did not have any memory for any of the episodes. When asked, he would say that he never did any such thing and could not give a reason for such behavior. Following the episodes, he would feel exhausted and would complain about numbness in his limbs. He would prefer to lie down and take rest for 15–20 min following the episodes. In the beginning, the episodes used to occur at a frequency of 2–3/months. As the illness progressed, the number of episodes increased to 2–3/week in last 3 months. Because of such episodes of sudden rage in school, he was suspended from school 1 month back. During the interepisodic period, the patient was totally asymptomatic.

Past history, family history, premorbid temperament, and personal history was noncontributory, and there were no other symptoms suggestive of generalized tonic-clonic seizure. Detailed child psychiatry assessment and neurological examinations did not reveal any abnormality. Detailed routine blood and urine investigations including thyroid and other endocrine functions were within normal limits. Patient’s old treatment records showed that patient was earlier diagnosed as psychosis NOS, bipolar disorder, and dissociative disorder by three different clinicians. The patient was also prescribed olanzapine 10 mg for 2 months, but there was no response. The patient also underwent counseling sessions by psychologists, but the episodes of “rage attacks” increased over a period. No significant psychopathology could be elicited in his mental status examination. On the basis of history, mental status examination, neurological examination, psychological evaluation, and appropriate laboratory tests, a differential diagnosis of intermittent explosive disorder, dissociative disorder, and partial seizures was kept. MRI brain
was within normal limits and EEG showed spike and slow wave discharges in bilateral frontotemporal leads. On the basis of EEG changes, our diagnosis was revised to complex partial seizure. The patient was started on sodium valproate, and dose was gradually increased to 600 mg/day over a period of next 4 weeks as per her body weight. There was a significant reduction in his “rage attacks.” He is currently well adjusted in his personal and social life.

**Case 3 – Simple Partial Seizure Presenting as “Secondary Enuresis”**

L, 7-year-old Hindu female child, studying in Class 1, belonging to a lower socioeconomic status, resident of Delhi, living in a nuclear family, presented in the Child and Adolescent Psychiatry OPD, Department of Psychiatry, PGIMER, Dr. RML Hospital, New Delhi, with chief complaints of urination in clothes from last 1 year. She was referred from the Department of Pediatrics for proper evaluation.

Exploration of history revealed that patient was apparently well 1 year back and was well involved in her daily routine. As per family members, patient’s toilet training was complete by 5 years of age. She would go to toilet by herself or would inform her parents, whenever she would feel the urge for urination. Since last 1 year, she lost her bladder control and would urinate in her clothes. In the beginning, parents noticed that she would urinate in clothes during sleep only; however, as the illness progressed, she started urinating during daytime also. In the beginning, patient’s parents tried conservative measures such as clearing the bladder before sleep, waking the child in the midnight to urinate to avoid bedwetting, but with no relief in symptoms. Earlier, the episodes used to occur 4–5 times in a month only during the sleep, but from last 2 months, the patient started urinating in clothes 3–4 times any time in a day. Many a times, the patient would cry for 2–3 min following urination in clothes. It would be difficult to console the patient during the crying spells, as the patient would be indifferent to her surroundings during crying. Parent would think that patient was crying because of embarrassment, but the patient would not give any reason for it. Parents even made a video recording of the crying episodes during night and even during daytime. The video recordings showed crying spells with inappropriate affect and unconcerned to surroundings. When the patient was asked about the reason for urinating in clothes and crying, she would respond by saying that she was not aware of any urge and it happened suddenly. There were no precipitating or aggravating factors, and no stressors could be elicited. Due to increase in the frequency of episodes, the patient stopped going to school since last 1 month due to embarrassment.

There were no other symptoms suggestive of convulsive seizures. Past history, personal history, and premorbid temperament were noncontributory. Detailed child psychiatry assessment using K-SADS and detailed neurological examinations did not reveal any abnormality. Detailed routine blood and urine investigations including thyroid and other endocrine functions were within normal limits. Patient’s mother consulted two pediatricians in private and was diagnosed as secondary enuresis. The patient was also prescribed imipramine 25 mg, but there was no improvement in her symptoms despite 2 months treatment. On the basis of history, mental status examination, neurological examination, psychological evaluation, and appropriate laboratory tests, a differential diagnosis of secondary enuresis and partial seizure was kept. MRI brain and EEG were advised to rule out the differential diagnosis. The EEG showed generalized epileptiform discharges. On the basis of EEG changes, our diagnosis was revised to generalized epilepsy. The patient was started on sodium valproate, whose dose was gradually increased to 400 mg/day over a period of next 4 weeks as per her body weight. She stopped urinating in clothes and is now well adjusted to her daily routine.

**Discussion**

Due to the rare and atypical presentation of childhood epilepsy, there is often a higher chance of misdiagnosis even by the most experienced clinicians. Moreover, since psychiatrists and neurologists are better acquainted with neurotic symptoms such as anxiety, depression, and dissociative symptoms, there is more likelihood of patient with the atypical presentation being diagnosed as psychiatric disorders as happened in the above cases. The illnesses of Case 1, Case 2, and Case 3 represent a diagnostic challenge of distinguishing partial epilepsy from other neuropsychiatric disorders such as episodic hallucinations, rage attacks, and secondary enuresis. We will give a detailed diagnostic approach in all the three cases.

**Misdiagnosis in epilepsy**

Traditionally, there have been high rates of misdiagnosis in epilepsy. In a study done on 233 children in Denmark, the expert assessment concluded that 87 (39%) did not have epilepsy, and of these, 35 (40%) were already on antiepileptic drugs.[7] The most frequently observed differential diagnoses were nonepileptic staring spells (52.8%), psychogenic nonepileptic seizures (10.3%), syncope (3.4%), dystonia (3.4%), and parasomnias (3.4%). Misdiagnosis of epilepsy as nonepileptic event appears to be less common. In a Dutch study including 888 children with paroxysmal events, 19/124 (5.6%) children were diagnosed as epilepsy.[8] There was a significant reduction in misdiagnosis rates when an experienced clinician with expertise in epilepsy did the initial assessment. The diagnosis of an epileptic or nonepileptic event can often be accurately made by a detailed description of the episodes by the patient and witnesses. If the events were recurrent and there is doubt about the diagnosis, use of smartphones can greatly facilitate the diagnosis and parents should be strongly encouraged to capture them on video. The video recording of the child crying episode during the episode of enuresis actually contributed significantly in finalizing the diagnosis in Case 3.
It should be reemphasized that for a patient with presumed epilepsy, detailed history taking and exploration of individual symptoms are the primary ways to achieve an accurate diagnosis in up to 90% of cases.\(^9,10\) The basic requirement in the evaluation of epilepsy in psychiatric patients is to establish whether the nonspecific somatic manifestations possess the core features of epilepsy, i.e., unprovoked, sudden-onset, paroxysmal, or episodic, and of short duration. Even prolonged duration (such as 2–4 h, or even most of the daytime) of symptoms should not be ruled out for the possibility of epilepsy, since they may be the presenting feature of a subclinical nonepileptic status epilepticus. Whatever the presentation, a clinician should never shy away from advising an EEG or an MRI or doing a detailed neurological examination to rule out any focal neurological cause.

**Description of seizure types**

The clinician should try to classify the seizure type if the clear description of the event(s) is thought to be epileptic. An EEG is helpful particularly in focal epilepsies as focal epileptiform activity may help localize a lobar origin.

Case 1 presented with only auditory hallucinations with no other associated symptom. The history of hallucinations, muttering to self, inappropriate crying, and sudden onset fearfulness associated with significant distress in patients life will lead any clinician to diagnose the patient as psychosis as happened in Case 1. We also kept our first differential as psychosis NOS, but the only symptom which was atypical was the episodic nature of the illness as these symptoms lasted for only few minutes. The auditory hallucinations can be seen in >70% of schizophrenia patients; however, hearing voices is not specific for schizophrenia, despite being the core symptom in schizophrenia as defined in the Diagnostic and Statistical Manual of Mental Disorders-IV diagnostic system.\(^11\) Auditory hallucinations can be frequently seen in the general population as evident in a population-based study by Krabbendam and van Os.\(^12\) Auditory hallucinations are also reported in nonpsychotic patient groups, for example, Alzheimer’s disease,\(^13\) major depression,\(^14\) and in epilepsy.\(^15\) Some studies have shown that epileptic patients fail to show a right ear advantage on dichotic listening tasks.\(^16,17\) Therefore, it could be hypothesized that auditory hallucinations may have a neuronal origin in the speech perception areas in the left temporal lobe and not unique for schizophrenia. Evidence of reduced gray matter density in temporal lobe areas in patients with temporal lobe epilepsy also suggests the neuronal origin of auditory hallucinations in epilepsy.\(^18\) Gray matter density or volume reduction may be an independent predisposing factor leading to auditory hallucinations in both patients with schizophrenia and epilepsy. In the indexed case 1, the reason for suspicion for seizure was atypical psychotic features, episodic hallucinations, nonpervasive in nature, no associated change in affect, nonresponsive to any treatment, and stereotyped presentation of hallucinations. The atypical clinical presentation guided us to keep a differential of partial seizure and to advice for an EEG investigation. Moreover, nonresponse to antipsychotics and very early presentation of psychotic symptoms also raised serious doubt in our mind regarding the diagnosis of psychosis. The significant response to antiepileptic further confirmed our diagnosis of partial seizure.

Case 2 presented with episodes of rage attacks. The reason for suspicion for seizure was stereotyped presentation, episodic nature, memory loss of the episodes, unprovoked aggression, nongoal-directed abusiveness, nonresponsive to verbal commands, abnormal movements, nonresponse to treatment, absence of precipitating factors, and early age of presentation of dissociative symptoms, which is very rare in the absence of any kind of stressor. Reports of violence and aggression during the ictal phase of epilepsy have been well documented, but criminality associated with seizure activity is not very common. Aggressive behavior in epilepsy patients can occur in either of the interictal, ictal, or postictal phases.\(^19\) Ictal aggressive behavior is usually resistant, not targeted, stereotyped, and more commonly originates from the frontal or temporal regions.\(^20,21\) Postictal aggression usually occurs during the confusional state and is more likely to be resistive violence.\(^20\) Temporal or frontal lobe seizures have been associated with aggressive and violent behaviors.\(^22\) It is generally believed that well-organized, purposeful, complex, goal-directed behavior is highly unlikely during a seizure.\(^23\) Interactive behavior with the ability to respond to visual and verbal stimuli is also rare in complex partial seizures. The unusual presentations in the Case 2 differentiated it from normal aggression and led us to review our diagnosis to epilepsy, which was corroborated by the EEG findings. The abusiveness and aggressive behavior could localize to the prefrontal cortex as well as limbic system due to the presence of emotional symptoms such as anger outburst. The patient also reported amnesia for the ictal episodes, which further points toward a simple partial seizure.\(^24,25\)

Diagnosis in Case 3 was the most difficult one as the onset of symptoms was after 5 years of age and secondary enuresis being the most commonly made diagnosis at this age based on the patient’s presentation. The reason for suspicion for seizure was repeated episodes of unexplained enuresis associated with inappropriate crying spells as shown in videos made by the parents, indifference to surroundings, stereotyped presentation, loss of memory of the episodes, absence of stressors, complete toilet training before the onset of illness, and no associated medical illness. The atypical clinical presentation raised our concern to think in a different line and advice for EEG. The EEG finding of generalized epileptic discharges in our case 3 helped us confirm our diagnosis. The diagnosis of ictal enuresis is very difficult to be made as there has been a very limited scientific literature describing true simple ictal enuresis except few reports.\(^16-29\) Penfield and Kristiansen in 1951 were the first to describe the phenomenon of ictal enuresis as a sole manifestation in simple seizures.\(^26\) Micturition is a highly complex process involving coordination of central and peripheral nervous system through multiple centers. Although there is little knowledge about the exact
brain areas involved in the control of micturition, it is widely accepted fact that the basic micturition reflex is under spinal control and beginning of micturition is under the control of higher brain areas.\(^{[30]}\) We were unsuccessful in the localization of the seizure foci in Case 3, as MRI findings were normal, but simple autonomic seizures with enuresis and their ictal EEG presentation could be helpful in providing an insight into the localization of the cortical centers of micturition. In general, enuresis during seizure occurs at the end of the clonic phase of a tonic–clonic episode as the sphincter muscle relaxes in a patient with a full bladder. Sometimes, it may also complicate absence and focal seizures, possibly resulting from an impaired cortical inhibition of the micturition reflex due to the involvement of cortical centers by seizure foci and increased intravesical pressure.\(^{[30]−32]}\)

**The role of the electroencephalogram**

Although EEG is a helpful guide to the clinicians in the diagnosis of seizure types and epilepsy syndromes, it has both poor specificity and sensitivity. One should always keep in mind that EEG should not be used as a diagnostic test for epilepsy as about one-third of patients with epilepsy will have a completely normal interictal EEG and 5% of nonepileptic children may present with frank epileptiform discharges.\(^{[33]}\) A routine EEG performed in a patient with a presumed first seizure will show epileptiform abnormalities about 7%–34% of the time. An EEG performed in the first 24 h after an attack increases the EEG yield to 51%.\(^{[34]}\) Repeated EEGs, sleep deprivation before the EEG, and sleep recorded during the EEG all increase the likelihood of recording epileptiform discharges.\(^{[35]}\)

The EEG will almost always show some paroxysmal change in seizures with altered consciousness and most of the time an evolving rhythmic ictal discharge will be apparent if the seizures are epileptic.\(^{[36]}\) When there is clinical doubt regarding the diagnosis of epilepsy, video-telemetry or ambulatory EEG can be very useful, since most epileptic events will have an EEG correlate. Simple partial seizures usually present with just nonspecific or unremarkable EEG findings even during attacks because the foci of partial seizure may involve only subcortical regions.\(^{[37]}\) Although EEG is only supplementary to the diagnosis, EEG findings helped us in confirming our suspicion and supported our clinical findings in all the three cases. Therefore, EEG should be strongly suggested whenever there is clinical doubt regarding the diagnosis as we did in all the three cases. The aim is to detect the pathogenic mechanism underlying these episodes, which is not always possible by simply history taking and following known guidelines.

**Conclusion**

Since the diagnosis of epilepsy can have significant implications for patients, their families, and therapeutic management, one should always take extra caution in making a diagnosis of epilepsy. Moreover, due to lack of definitive diagnostic investigation, diagnosis can sometimes be made by detailed chronological history and EEG findings only, as demonstrated in the above three rare cases of episodic hallucinations, rage attacks, and enuresis. The diagnosis of seizures should always be considered in cases of episodic stereotyped behavior with atypical presentations. As understanding of this disease continues to evolve, it is important for clinicians to stay up-to-date with the latest advances to provide the best care for patients.

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**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Newton CR, Garcia HH. Epilepsy in poor regions of the world. Lancet 2012;380:1193-201.
2. Sillanpää M, Shinnar S. SUDEP and other causes of mortality in childhood-onset epilepsy. Epilepsy Behav 2013;28:249-55.
3. Carpio A, Bharucha NE, Jallon P, Beghi E, Campostrini R, Zorzetto S, et al. Mortality of epilepsy in developing countries. Epilepsia 2005;46 Suppl 11:28-32.
4. Centers for Disease Control and Prevention. Epilepsy Fast Facts; 2015. Available from: http://www.cdc.gov/epilepsy/basics/fast‑facts.htm. [Last accessed on 2015 Oct 08].
5. Kanner AM, Soto A, Gross-Kanner H. Prevalence and clinical characteristics of postictal psychiatric symptoms in partial epilepsy. Neurology 2004;62:708-13.
6. Kotsopoulos IA, van Merode T, Kessels FG, de Krom MC, Knothanrus JA. Systematic review and meta-analysis of incidence studies of epilepsy and unprovoked seizures. Epilepsia 2002;43:1402-9.
7. Uldall P, Alving J, Hansen LK, Kibaek M, Buchholt J. The misdiagnosis of epilepsy in children admitted to a tertiary epilepsy centre with paroxysmal events. Arch Dis Child 2006;91:219-21.
8. Stroink H, van Donselaar CA, Geerts AT, Peters AC, Brouwer OE, Arts WF. The accuracy of the diagnosis of paroxysmal events in children. Neurology 2003;60:979-82.
9. Bazil CW, Morrell MJ, Pedley TA. Epilepsy. In: Rowland LP, Editor. Merritt’s Neurology. 11th ed. Philadelphia. Lippincott Williams and Wilkins; 2005. p. 990-1016.
10. Schapiro AH, Byrne E, DiMauro S. Epilepsy. In: Editors. Neurology and Clinical Neuroscience. 3rd ed. Philadelphia: Mosby Elsevier; 2007. p. 662-719.
11. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders (DSM). Washington, D.C.: American Psychiatric Press; 1994.
12. Krabbendam L, van Os J. Schizophrenia and urbanicity: A major environmental influence – Conditional on genetic risk. Schizophr Bull 2005;31:795-9.
13. Perez-Madriñan G, Cook SE, Saxton JA, Miyahara S, Lopez OL, Kauer DI, et al. Alzheimer disease with psychosis: Excess cognitive impairment is restricted to the misidentification subtype. Am J Geriatr Psychiatry 2004;12:449-56.
14. Skaf CR, Yamada A, Garrido GE, Buchpiguel CA, Akamine S, Castro CC, et al. Psychotic symptoms in major depressive disorder
are associated with reduced regional cerebral blood flow in the subgenual anterior cingulate cortex: A voxel-based single photon emission computed tomography (SPECT) study. J Affect Disord 2002;68:295-305.

15. Brasic JR, Perry R. Unilateral auditory hallucinations in a boy with ipsilateral conductive hearing loss. J Neurol Neurosurg Psychiatry 1997;62:302.

16. Gramstad A, Engelsen BA, Hugdahl K. Left hemisphere dysfunction affects dichotic listening in patients with temporal lobe epilepsy. Int J Neurosci 2003;113:1177-96.

17. Fontoura DR, Branco Dde M, Anés M, Costa JC, Portuguez MW. Language brain dominance in patients with refractory temporal lobe epilepsy: A comparative study between functional magnetic resonance imaging and dichotic listening test. Arq Neuropsiquiatr 2008;66:34-9.

18. Bell B, Hermann B, Seidenberg M, Davies K, Cariski D, Rosenbek J, et al. Ipsilateral reorganization of language in early-onset left temporal lobe epilepsy. Epilepsy Behav 2002;3:158-64.

19. Kim JM, Chu K, Jung KH, Lee ST, Choi SS, Lee SK. Characteristics of epilepsy patients who committed violent crimes: Report from the national forensic hospital. J Epilepsy Res 2011;1:13-8.

20. Marsh L, Krauss GL. Aggression and violence in patients with epilepsy. Epilepsy Behav 2000;1:160-8.

21. Ito M, Okazaki M, Takahashi S, Muramatsu R, Kato M, Onuma T. Subacute postictal aggression in patients with epilepsy. Epilepsy Behav 2002;7:106-114.

22. Jobst BC, Siegel AM, Thadani VM, Roberts DW, Rhodes HC, Williamson PD. Intractable seizures of frontal lobe origin: Clinical characteristics, localizing signs, and results of surgery. Epilepsia 2000;41:1139-52.

23. Mendez MF. Postictal violence and epilepsy. Psychosomatics 1998;39:478-80.

24. Rüdigerkof KR, van den Wildenberg WP, Segalowitz SJ, Carter CS. Neurocognitive mechanisms of cognitive control: The role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. Brain Cogn 2004;56:129-40.

25. Hasselmo ME. A model of prefrontal cortical mechanisms for goal-directed behavior. J Cogn Neurosci 2005;17:1115-29.

26. Penfield W, Kristensen K. Epileptic Seizure Patterns. Springfield, IL: Charles C. Thomas; 1951.

27. Lesser R, Lüders H, Dinner D, Morris H. Simple partial seizures. In: Lüders H, Lesser RP, editors. Epilepsy: Electroclinical Syndromes. London, Berlin-Heidelberg, New York, Paris, Tokyo: Springer-Verlag; 1987. p. 223-78.

28. Mulder DW, Daly D, Bailey AA. Visceral epilepsy. AMA Arch Intern Med 1954;93:481-93.

29. Baumgartner C, Lurger S, Leutmezer F. Autonomic symptoms during epileptic seizures. Epileptic Disord 2001;3:103-16.

30. Holstege G, Mouton LJ. Central nervous system control of micturition. Int Rev Neurobiol 2003;56:123-45.

31. Blok BE, Willemsen AT, Holstege G. A PET study on brain control of micturition in humans. Brain 1997;120(Pt 1):111-21.

32. Nour S, Svarer C, Kristensen JK, Paulson OB, Law I. Cerebral activation during micturition in normal men. Brain 2000;123(Pt 4):781-9.

33. Okubo Y, Matsuura M, Asai T, Asai K, Kato M, Kojima T, et al. Epileptiform EEG discharges in healthy children: Prevalence, emotional and behavioral correlates, and genetic influences. Epilepsia 1994;35:832-41.

34. King MA, Newton MR, Jackson GD, Fitt GJ, Mitchell LA, Silvapulle MJ, et al. Epileptology of the first-seizure presentation: A clinical, electroencephalographic, and magnetic resonance imaging study of 300 consecutive patients. Lancet 1998;352:1007-11.

35. Ellingson RJ, Wilken K, Bennett DR. Efficacy of sleep deprivation as an activation procedure in epilepsy patients. J Clin Neurophysiol 1984;1:83-101.

36. Devinsky O, Sato S, Kafta CV, Ito B, Rose DF, Theodore WH, et al. Electroencephalographic studies of simple partial seizures with subdural electrode recordings. Neurology 1989;39:527-33.

37. Talbot JD, Marrett S, Evans AC, Meyer E, Bushnell MC, Duncan GH. Multiple representations of pain in human cerebral cortex. Science 1991;251:1355-8.