Original Article

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Cognitive changes in stable epileptic patients attending a tertiary care hospital

Ambuj kr Pandey[1], Taniya Majhi[2], Surjya Roy[3], Asim kr Mallick[4]

[1] Bolpur SD Hospital

[2, 3] Diamond Harbour Medical College & Hospital

[4] ICARE Institute of Medical Science and Research Haldia

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Abstract

Background: Epilepsy is a complex neurological condition in which cognitive impairments in unstable patients are extensively documented. there is a lack of research concerning cognitive outcomes in patients who are clinically stable while on medication.
Objectives: This study aimed to delineate the cognitive profile of stable epilepsy patients and examine the relationship between cognitive domains and clinical variables such as seizure frequency, illness duration, and age.
Methods: A cross-sectional study was conducted at Burdwan Medical College (January–December 2017) involving 50 epilepsy patients stable on medication for at least three months. Cognitive function was assessed using the Post Graduate Institute Battery of Brain Dysfunction (PGI-BBD). Statistical analysis utilized Pearson correlation to identify associations between clinical factors and cognitive domains.
Results: Despite clinical stability, significant cognitive variations were observed. Remote memory demonstrated a constant, highly significant negative correlation with seizure frequency ($r = -0.360$, $p = 0.010$), illness duration ($r = -0.511$, $p = 0.000$), and age ($r = -0.465$, $p = 0.001$). Conversely, visual retention and attention/concentration showed significant positive correlations with illness duration and age, suggesting potential compensatory neural mechanisms. Other domains, including performance quotient, remained relatively stable.
Conclusion: Clinical stability in epilepsy with anti-epileptic's drugs or without does not avert the gradual decline of remote memory function. These observations indicate a "cognitive fingerprint" reflecting selective susceptibility in long-term memory, even when attentional skills remain intact. Upcoming therapeutic approaches should include cognitive rehabilitation and ongoing assessment to maintain long-term quality of life.

Keywords: epilepsy, cognitive impairment, PGI –BBD

INTRODUCTION

The term epilepsy comes from the Greek word epilepsia, which means "to seize" or "to attack." In accordance with International League Against Epilepsy guidelines (ILAE), epilepsy is a brain disorder defined by having at least two unprovoked seizures that occur more than 24 hours apart, or by experiencing a single unprovoked seizure with a 60% chance of recurrence within the next ten years, or by having a specific diagnosis of an epileptic syndrome. A seizure is an abnormal, brief, excessive discharge of electrical activity and can be classified as — partial (focal), generalized and unknown onset[1,2]. Focal-onset seizures originate from circumscribed brain regions within a single cerebral hemisphere or quickly progress to bilateral tonic-clonic affecting all cortical regions. Generalized seizures involve simultaneous onset of abnormal electrical activity in both cerebral hemispheres, with subsequent spread to other brain networks, evident clinically or on EEG[3,4].

Various comorbidities are associated with epilepsy, the most severe and burdensome being the cognitive dysfunction. Cognitive deficits may sometimes be present even in new-onset epilepsy, which is suggestive of a bidirectional relationship between cognitive impairment and epilepsy[5]. There may be partial or complete deterioration in cognitive skills involving difficulty in attention, concentration, memory impairment, executive dysfunction, psychomotor slowing, impaired naming abilities and visuo-spatial abilities adversely affecting the quality of life[6-8]. Cognitive functions may be affected during the ictal, postictal and occasionally the normal interictal period.

It is also found that all commonly used anti-epileptic drugs(AEDs) have both positive and negative impact on cognitive function. While seizure reduction due to AED intake may improve cognition, AEDs can also produce dose-dependent

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Address for correspondence:

Dr. Surjya Roy, Assistant Professor
Department of Psychiatry, Diamond Harbour Medical College and Hospital, Diamond harbour, West Bengal, India
Email:drroysurjya@gmail.com

impairments, particularly with polytherapy, through modulation of neuronal excitability and inhibitory neuro transmission[9-12]. As a result, assessment and correction of these cognitive effects is often deemed necessary to ensure long term compliance to AEDs. Although cognition in unstable epilepsy has been widely studied, there is a paucity of studies regarding the cognitive outcome in stable patients of epilepsy. Hence, this current study was conceptualized to assess the cognitive decline in patients stable on anti-epileptic medications.

METHODS

Study settings and participants

50 patients of epilepsy (coming for follow up who were previously diagnosed by signs and symptoms of epilepsy as per International League Against Epilepsy classification) and they are now stable on medication (for last 3 months seizure free) and gave valid and informed consent were recruited. They were assessed by Liverpool Seizure Severity Scale for severity. The research conducted was a cross-sectional study with a defined timeframe of one year, extending from January 2017 to December 2017, held at the Psychiatry Outpatient Department of Burdwan Medical College and Hospital, and was approved by the Institutional Ethics Committee. Individuals with any form of traumatic brain injury, patients with mental disabilities, any structural brain lesions identified through imaging, and all secondary epilepsy cases were excluded.

Study tools

Semi-structured socio-demographic proforma

A semi-structured interview schedule was specifically created and approved by the ethical committee of Burdwan Medical College and Hospital, incorporating parameters such as age, sex, religion, education, occupation, monthly family income in rupees, and family type. Socioeconomic status was classified using the BG Prasad Socio Economic Scale[13].

Liverpool Seizure Severity Scale

It is a patient rated scale. The scale comprises 20 items, which patients rate according to their perceived severity of seizures. The possible scoring ranges are between 7 and 32 for the percept scale and between 10 and 48 for the ictal/postictal scale. The higher the score the more severe the seizures[14].

Post graduate Institute Battery of Brain Dysfunction(PGI-BBD)

The PGI-BBD was developed by Dr Dwaraka Prasad and Dr Santosh K Verma of Post Graduate Institute of Medical Education and Research, Chandigarh in 1990. The components include: (1) PGI Memory Scale (PGIMS) assessing memory, attention and concentration, recall, retention and mental balance; (2) Revised Bhatia's Short Battery of Performance Tests of Intelligence (BSR-R); (3) Verbal Adult Intelligence Scale (VAIS); (4) Nahor–Benson test; (5) Bender visual motor Gestalt test (Bender–Gestalt test) for evaluation of perceptual and visual motor functioning. Raw scores are converted into adjusted scores using PGI norms, and cumulative scores classify impairment as no impairment [0–19], slight impairment [20–38], or severe impairment [39–57][15].

Statistical analysis

Statistical analysis was done after completion of data collection using standard statistical methods and analysed using IBM SPSS version 20. Descriptive statistics was done to measure mean, median, mode and standard deviation. Chi square test was used for categorical variable comparison. Analysis of Variance(ANOVA) test was done to compare means of quantitative data between epileptic patients and Healthy Control groups and appropriate Post hoc test were followed for multiple comparison between the groups, independent sample t was used for comparing means between two groups. Data was presented as percentage, means and standard deviation. All tests were 2 tailed. p value less than 0.05 was considered statistically significant (95% confidence interval).

RESULTS

The participants in the study comprised of 50 diagnosed patients of epilepsy who have remained stable for the last 3 months prior to the commencement of the study.

Table 1: Sociodemographic and Clinical Characteristics (n=50)

Variable	Category	n	%
Age (mean ± SD, years)	-	29.36 ± 5.48	-
Gender	Male	23	46
	Female	27	54
Residence	Rural	39	78
	Urban	11	22
Family Type	Nuclear	29	58
	Joint	21	42
Education	Illiterate	7	14
	Primary School	15	30
	Middle School	8	16
	High School	7	14
	Higher Secondary	7	14
	Graduation & Above	6	12
Socioeconomic Status (BG Prasad)	Upper	2	4
	Upper Middle	10	20
	Middle	12	24
	Lower Middle	12	24
	Lower	14	28
Age of Onset (mean ± SD, years)	-	21.4 ± 6.2	-
Duration of Illness (mean ± SD, years)	-	7.9 ± 4.1	-

Among 50 stable epileptic patients (mean age 29.36 ± 5.48 years), 46% were male and 54% female, with 78% rural residents, 58% from nuclear families, and 30% primary school educated. Socioeconomic status showed 28% lower class as per BG Prasad scale. Clinical characteristics included mean age of onset at 21.4 ± 6.2 years and illness duration of 7.9 ± 4.1 years [Table 1].

Table 2: Correlation Between Cognition and Seizure Episodes

Cognitive Domain	Pearson Correlation (r)	P-value
Remote Memory	-0.360	0.010*
Recent Memory	-0.279	0.049*
Mental Balance	-0.029	0.844
Attention and Concentration	-0.035	0.812
Delayed Recall	-0.018	0.903
Immediate Recall	-0.019	0.894
Retention for Similar Pairs	0.137	0.343
Retention for Dissimilar Pairs	-0.264	0.064
Visual Retention	-0.120	0.407
Recognition	-0.115	0.428

In the stable epilepsy cohort, Pearson correlation analysis indicates a statistically significant negative association between the frequency of seizure episodes and memory performance, particularly in Remote Memory (r = -0.360, p = 0.010) and Recent Memory (r = -0.279, p = 0.049).Although several other areas including mental balance, attention, and recognition showed negative correlation tendencies, these did not achieve statistical significance (p > 0.05).These findings support the conclusion that frequent seizure activity may contribute to cumulative, localized cognitive decline due to secondary neuronal, metabolic, and structural deterioration. Clinically, this underscores the vital necessity of attaining early and comprehensive seizure control to reduce the risk of potentially irreversible memory impairment [Table 2].

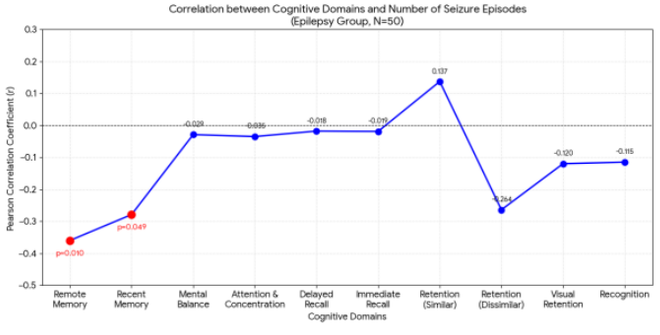

Figure 1: Correlation between Number of Seizure Episodes and Cognitive Domains

The above trend confirmed that memory functions are uniquely sensitive to the frequency of seizures. Unlike other cognitive processes that may remain relatively stable, both recent and remote memory show a clear and statistically significant decline as the number of seizure episodes increases, reinforcing the need for aggressive seizure management to preserve memory integrity [Figure 1].

Table 3: Correlation between Cognitive Domains and Duration of epilepsy

Cognitive Domain	Pearson Correlation (r)	Significance (p-value)
Attention and Concentration	0.318	0.025*
Visual Retention	0.472	0.001**
Remote Memory	-0.511	0.000**
Delayed Recall	0.258	0.070
Nahor and Benson Test	0.232	0.105
Performance Quotient	0.034	0.814

*statistically significant(p<0.05)

Based on the findings presented in the table, the study demonstrated a variable association between duration of epilepsy and cognitive performance. A statistically significant positive correlation is observed for Attention and Concentration (r = 0.318, p = 0.025) as well as Visual Retention (r = 0.472, p = 0.001). In contrast, Remote Memory exhibits a highly significant negative correlation (r = −0.511, p = 0.000), indicating that longer duration of illness is linked to increasing difficulty in long-term memory retrieval. No significant associations are noted for Delayed Recall (p = 0.070), the Nahor and Benson Test (p = 0.105), or Performance Quotient (p = 0.814). Overall, these results imply that while certain attentional and visual retention abilities may show relative preservation or domain-specific changes over time, prolonged epilepsy constitutes an important risk factor for progressive deterioration of remote memory [Table 3].

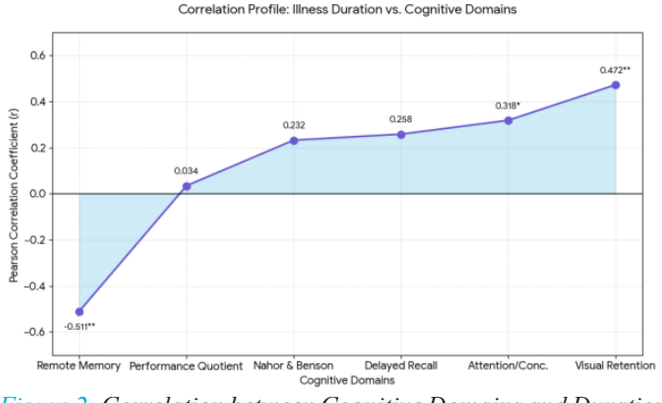

Figure 2: Correlation between Cognitive Domains and Duration of epilepsy

The area–line graph depicts a clear divergence in cognitive outcomes in relation to the duration of epilepsy. A pronounced negative trough is evident for Remote Memory (r = −0.511, p < 0.001), reflecting a highly significant decline in long-term memory with increasing illness duration. In contrast, notable positive elevations are seen for Visual Retention (r = 0.472) and Attention and Concentration (r = 0.318). These

patterns indicate that although prolonged epilepsy substantially compromises remote memory, other domains such as visual retention and attentional abilities may be relatively preserved or exhibit distinct adaptive relationships in this clinical sample. Meanwhile, Delayed Recall and Performance Quotient display only minor, non-significant variations around the baseline, reinforcing that illness duration does not uniformly influence all aspects of cognitive functioning [Figure 2].

Table 4: Correlation between Cognitive Domains and Age of onset of epilepsy

Cognitive Domain	Pearson Correlation (r)	Significance (p-value)
Remote Memory	-0.465	0.001**
Visual Retention	0.472	0.001**
Attention and Concentration	0.318	0.025*
Delayed Recall	0.258	0.070
Nahor and Benson Test	0.232	0.105
Performance Quotient	0.034	0.814

*statistically significant(p<0.05)
**statistically significant(p<0.01)

Among patients with epilepsy, correlation analysis demonstrates a highly significant negative association between age and Remote Memory ($r = -0.465$, $p = 0.001$), indicating that increasing age is linked to a substantial reduction in long-term memory retrieval. In contrast, Visual Retention ($r = 0.472$, $p = 0.001$) and Attention and Concentration ($r = 0.318$, $p = 0.025$) exhibit significant positive correlations. Other cognitive domains, including Delayed Recall, the Nahor and Benson Test, and Performance Quotient, do not show statistically significant relationships ($p > 0.05$). These patterns mirror duration-of-illness correlations, suggesting confounding effects of aging and disease chronicity. The selective vulnerability of remote memory to aging in epilepsy patients reflects the cumulative neurological burden of both natural aging processes and chronic seizure activity on medial temporal lobe structures, particularly the hippocampus. The preservation of visual retention and attention suggests compensatory neural mechanisms or treatment-induced cognitive benefits, possibly indicating neural plasticity and adaptive reorganization in response to ongoing epilepsy [Table 4].

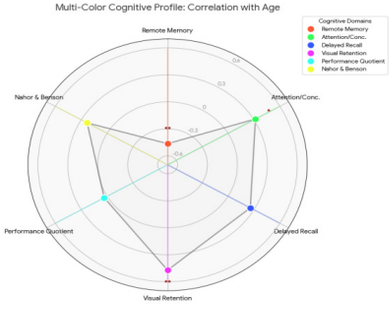

Figure 3: Correlation between Cognitive Domains and Age of epilepsy

The above radar chart visually summarizes the multi-dimensional impact of age on cognition in epilepsy. Each cognitive domain is represented by a unique color-coded marker and spoke, allowing for easy identification of the "cognitive fingerprint." Red asterisks denote statistical significance levels (*$p < 0.05$, **$p < 0.01$).

The "pull" toward the centre for Remote Memory ($r = -0.465$, $p < 0.01$) highlights the highly significant negative correlation, while the outward expansion for Visual Retention and Attention represents significant positive correlations. The symmetrical distribution of non-significant domains near the baseline confirms that global intellect (Performance Quotient) remains relatively centered despite chronological ageing in this patient group [Figure 3].

DISCUSSION

The aim of the present study was to delineate the cognitive profile of patients with epilepsy who are clinically stable on treatment, with particular emphasis on examining the relationship between key clinical variables such as seizure frequency, duration of illness and age across specific cognitive domains.

A major finding of the current research is that cognitive impairments continue to be evident even among patients who have been clinically stable on medication for at least three months. This observation is consistent with growing evidence suggesting that merely controlling seizures does not ensure the maintenance of optimal cognitive performance[16].

The most salient result of the present study was the constant negative association between Remote Memory and key clinical variables, including seizure frequency ($r = -0.360$), duration of illness ($r = -0.511$), and age ($r = -0.465$). This selective susceptibility is consistent with the “network abnormalities” hypothesis, which proposes that recurrent seizures and persistent subclinical epileptiform activity lead to progressive hippocampal atrophy[17,18]. Butler et al. demonstrated that impairment of remote memory is a defining feature of temporal lobe epilepsy and may persist despite intact short-term learning. This phenomenon, termed “accelerated long-term forgetting” (ALF), provides a plausible explanation for the marked long-term recall deficits reported by our clinically stable, seizure-free patients[19]. An interesting observation in this study was the positive relationship of Visual Retention and Attention with increasing duration of illness and advancing age in our sample. Despite its apparent paradox, evidence suggests that stable patients can exhibit compensatory neural mechanisms[20]. Longitudinal investigations of tract integrity report white matter reorganization and functional connectivity changes following medication stabilization, which may underlie enhancements in specific cognitive functions, including spatial memory and selective attention[21,22].

Recurrent brief seizure episodes resulted in cumulative, irreversible functional and structural alterations of the hippocampus, characterized by deficits in spatial memory and progressive pattern of neuronal loss in a manner analogous to human hippocampal sclerosis[23].

Hermann et al. identified diverse cognitive profiles in temporal lobe epilepsy, notably a subgroup demonstrating minimal impairment despite extended illness duration, suggesting

marked inter-individual differences in neural compensation[24]. A significant association was observed between age of onset and cognitive performance, with outcome patterns resembling those linked to illness duration. The pronounced negative correlation between age and remote memory ($r = -0.465$, $p = 0.001$) suggests a complex interplay between age-related processes, chronicity of epilepsy, and underlying neuropathological changes. Bhise et al. reported that the aetiology of memory impairments in focal epilepsy varies according to the temporal onset of the condition, with neurobiological determinants significantly influencing diminished cognitive performance in individuals diagnosed with early-onset epilepsy[25]. Early-life seizure onset during key neurodevelopmental periods is associated with atypical hippocampal development, diminished neurogenesis, and altered receptor expression, with ongoing seizures contributing to the cumulative progression of these pathological changes. The confounding influences of ageing and the enduring nature of disease chronicity identified in our study correspond with the premise of accelerated cognitive decline associated with epilepsy.

CONCLUSION

Our findings highlight that stability in epilepsy is a multidimensional concept. While seizure control represents an essential initial milestone, it does not constitute the ultimate therapeutic endpoint. The "cognitive fingerprint" of stable patients is characterized by progressive remote memory impairment in contrast to preserved or relatively maintained attention and concentration. Future therapeutic interventions should move "beyond seizures" to include cognitive rehabilitation and subclinical discharge monitoring to preserve the long-term quality of life for these patients.

Study Limitations and Future Directions

This cross-sectional study is subject to certain limitations. The methodological framework inhibits the establishment of causal inferences; longitudinal research following patients from the onset of symptoms would yield more robust evidence. We were deficient in a matched control group and did not conduct a systematic evaluation of specific pharmacological agents or their dosages. The PGI Battery may fail to identify nuanced deficits that could be elucidated through specialized neuropsychological assessments.

Subsequent research ought to integrate neuroimaging methodologies to evaluate hippocampal volumetric measures and functional connectivity patterns, explore biomarkers indicative of neuroplasticity and neurogenesis, and assess cognitive rehabilitation interventions tailored specifically for individuals diagnosed with epilepsy.

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Conflict of interest

There are no conflict of interest.

REFERENCES

1. Fisher RS, Acevedo C, Arzimanoglou A, Bogacz A, Cross JH, Elger CE, et al. ILAE official report: a practical clinical definition of epilepsy. Epilepsia. 2014 Apr;55(4):475–82.

2. Milligan TA. Epilepsy: A Clinical Overview. Am J Med. 2021 Jul;134(7):840–7.

3. Fisher RS, Cross JH, D'Souza C, French JA, Haut SR, Higurashi N, et al. Instruction manual for the ILAE 2017 operational classification of seizure types. Epilepsia. 2017 Apr;58(4):531–42.

4. Sarmast ST, Abdullahi AM, Jahan N. Current Classification of Seizures and Epilepsies: Scope, Limitations and Recommendations for Future Action. Cureus. 12(9):e10549.

5. Sayed NM, Aldin MTK, Ali SE, Hendi AE. Cognitive functions and epilepsy-related characteristics in patients with generalized tonic–clonic epilepsy: a cross-sectional study. Middle East Curr Psychiatry. 2023 Feb 3;30(1):15.

6. Hamed SA. The Aspects and Mechanisms of Cognitive Alterations in Epilepsy: The Role of Antiepileptic Medications. CNS Neuroscience & Therapeutics. 2009 Jun;15(2):134–56.

7. Hoxhaj P, Habiya SK, Sayabugari R, Balaji R, Xavier R, Ahmad A, et al. Investigating the Impact of Epilepsy on Cognitive Function: A Narrative Review. Cureus. 2023 Jun;15(6):e41223.

8. Wang L, Chen S, Liu C, Lin W, Huang H. Factors for cognitive impairment in adult epileptic patients. Brain Behav. 2020 Jan;10(1):e01475.

9. Ortinski P, Meador KJ. Cognitive side effects of antiepileptic drugs. Epilepsy Behav. 2004 Feb;5 Suppl 1:S60-65.

10. Ijff DM, Aldenkamp AP. Cognitive side-effects of antiepileptic drugs in children. Handb Clin Neurol. 2013;111:707–18.

11. Fisher JE, Vorhees CV. Developmental toxicity of antiepileptic drugs: relationship to postnatal dysfunction. Pharmacol Res. 1992;26(3):207–21.

12. Park SP, Kwon SH. Cognitive effects of antiepileptic drugs. J Clin Neurol. 2008 Sep;4(3):99–106.

13. Mangal A, Kumar V, Panesar S, Talwar R, Raut D, Singh S. Updated BG Prasad socioeconomic classification, 2014: a commentary. Indian J Public Health. 2015;59(1):42–4.

14. Baker GA, Smith DF, Jacoby A, Hayes JA, Chadwick DW. Liverpool Seizure Severity Scale revisited. Seizure - European Journal of Epilepsy. 1998 Jun 1;7(3):201–5.

15. Pershad D, Verma SK. Handbook Of P G I Battery Of Brain Dysfunction (pgi-Bbd). National Psychological Corpora; 1990. 172 p.

16. Manuel RGR, Raúl MR, Rodriguez-Leyva I, Cristina MT, Mario MCL, Luis RCJ, et al. Cognitive and Neuropsychiatric Outcomes in Economically Active Young Patients with Epilepsy: A Case-Control Study from a Secondary Medical Center in Mexico [Internet]. SSRN; 2024 [cited 2026 Jan 12]. Available from: https://www.ssrn.com/abstract=4998354

17. Novak A, Vizjak K, Rakusa M. Cognitive Impairment in People with Epilepsy. JCM. 2022 Jan 5;11(1):267.

18. Mukaino T, Uehara T, Yokohama J, Okadome T, Arakawa T, Yokoyama S, et al. Atrophy of the hippocampal CA1 subfield relates to long-term forgetting in focal epilepsy. Epilepsia. 2022 Oct;63(10):2623–36.

19. Butler CR, Zeman AZ. Recent insights into the impairment of memory in epilepsy: transient epileptic amnesia, accelerated long-term forgetting and remote memory impairment. Brain. 2008 Sep 1;131(9):2243–63.

20. Sharma S, Nehra A, Tripathi M. Applicability of Compensatory Cognitive Training in Epilepsy to Low Resource and Literacy Settings: A Focused Review.

21. Bell B, Lin JJ, Seidenberg M, Hermann B. The neurobiology of cognitive disorders in temporal lobe epilepsy. Nat Rev Neurol. 2011 Mar;7(3):154–64.

22. Liu H, Stover KR, Sivanenthiran N, Chow J, Cheng C, Liu Y, et al. Impaired Spatial Learning and Memory in Middle-Aged Mice with Kindling-Induced Spontaneous Recurrent Seizures. Front Pharmacol [Internet]. 2019 Sep 24 [cited 2026 Jan 15];10. Available from: https://www.frontiersin.org/journals/pharmacology/articles/10.3389/fphar.2019.01077/full

23. Desai JD. Epilepsy and cognition. Journal of Pediatric Neurosciences. 2008 Jun;3(1):16.

24. Hermann B, Seidenberg M, Lee EJ, Chan F, Rutecki P. Cognitive phenotypes in temporal lobe epilepsy. J Int Neuropsychol Soc. 2007 Jan;13(1):12–20.

25. Bhise VV, Burack GD, Mandelbaum DE. Baseline cognition, behavior, and motor skills in children with new-onset, idiopathic epilepsy. Developmental Medicine & Child Neurology. 2010;52(1):22–6.