



AN OVERVIEW OF EPILEPSY DISEASES & PRESENT CONDITION OF EPILEPSY IN BANGLADESH

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ABSTRACT

Even though epilepsy is one of the most prevalent and debilitating neurological disorders, our knowledge of its precise pathogenesis and therapeutic rationale is limited. Sixty-five million individuals throughout the globe are living with epilepsy, which imposes a substantial burden in terms of disability, mortality, comorbidities, stigma, and expenses due to seizures. Significant progress has been made in the last decade toward a better understanding of the pathophysiological underpinnings of the illness and the variables that influence its prognosis. These developments have resulted in updated criteria and terminology for the diagnosis and categorization of epilepsy and new conceptual and operational definitions. However, while there has been a significant rise in antiepileptic medications in the last 20 years, around a third of patients still refuse to respond to conventional therapy and take non-conventional treatment. Furthermore, only a tiny percentage of drug-resistant patients get epilepsy surgery, despite the fact that more than half of those who undergo surgery experience permanent independence from seizures. This article aims to introduce readers to the clinical aspects of epilepsy by covering topics including diagnostic methods, risk factors, and treatment strategies, as well as the current scenario of epilepsy in Bangladesh.

KEYWORDS: Epilepsy, Prevalence, Causes, Diagnosis, Management.

1. INTRODUCTION

Epilepsy is an ailment. When some individuals were ambushed by epilepsy, they trusted they had epilepsy. Transient paroxysms of unnecessary or uncontrolled releases of neurons might be caused by various distinctive etiologies called epileptic seizures [1]. Seizures are reactions of cerebral varieties from the standard. It is a direct result of the abnormal electrical development of the cerebrum.

Furthermore, epilepsy is a blood-borne sickness that is portrayed by effective direction. Term epilepsy is gotten from the Greek word "Epilamvanein or Epilepsia," which signifies "to be seized," "to be grabbed hold of," or "to be assaulted. Around 65 million people on the planet have epilepsy [2]. As of late, there has been an expanding acknowledgment that epilepsy isn't just about repetitive seizures; however, as reflected in the most recent accord definition, it additionally concerns the neurobiology, intellectual, mental, and social outcomes of the condition [3]. The Prevalence of brain

disorders is higher in developing countries than in developed countries [4]. So the incidence frequency is additionally higher in males than females. Epilepsy has an increased risk of premature birth death with mortality risk from 1.2-9.3 of all causes of death and 24% Longevity [5]. Remarkably, the frequency rate can reach 6–9 for every 10³ men per year in a contender for epilepsy surgery [6]. WHO assesses that eight individuals for each 1000 worldwide have this disorder [7]. Rates of occurrence are rising steadily over the globe. That's why it's crucial to understand the condition of epilepsy. It's also essential to realize that a proper diagnosis of drug-safe epilepsy in a male patient is necessary. Here, we report on predominance, occurrence, statistical information, and sorts of epilepsy, etiology, treatment, and forecast to give a complete perspective of epilepsy.



2. METHODOLOGY

We searched the subsequent tabulated databanks, journals, and sites by using the titles of 'prevalence & incidence of epilepsy,' 'cause of epilepsy,' 'pathophysiology,' 'classification,' 'sign & symptoms,' 'risk factors,' & 'management of epilepsy' shown in **Fig 1(See Appendix)** In addition, we used to identify our search by writing 'title & author' in Google and Google Scholar.

3. PREVALENCE AND INCIDENCE

Prevalence is stated per 1000 people in the population, and Incidence is expressed as new cases per 100,000 persons per year. The overall predominance of epilepsy is conflicting and expanded among nations simultaneously. It is assessed that the general pervasiveness is 10/1,000 individuals [8]. The prevalence rate of epilepsy in a different region is summarized in **Fig 2 (See Appendix)** The more significant part of concentrates on epilepsy is directed in Asia, a huge medical issue. The age-balanced predominance of epilepsy is 10.2 for each 1000 in Asia, which is higher than in Europe [9]. Despite the scarcity of data on the Prevalence of epilepsy, a few studies have shown that the median incidence rates in developing and industrialized nations, respectively, are 68.7 and 43.4 per 100,000 people [10].

4. CLASSIFICATION AND SIGNS OF SEIZURES

Classification plays a central role in clinical brain disease and brain disease analysis. The initial international types of brain disease goals are improving communication, providing some organization to the data regarding brain disease at that time, and facilitating research [11]. Classification of epilepsy is moreover frequently used to allude to the rundown of the distinctive types of epilepsy sorted out inside the characterization framework and to an individual analysis itself. Seizures are classified into three categories according to the ILEA classification of epilepsy [12] (**Fig 3(See Appendix)**) A partial seizure influences just a single piece of the cerebrum [13]. Manifestations rely upon which part is influenced. For example, one player in the body, or different body parts restricted to the other side of the body, may begin to jerk wildly [14].

On the other hand, generalized seizures influence both cerebral halves of the sphere (sides of the cerebrum) from the earliest starting point of the episode [15]. They deliver a loss of awareness, either quickly or for a more extended time. However, unclassified seizures have no inadequate data & cause neonatal seizures, e.g., chewing and rhythmic eye movements.

5. CAUSES OF EPILEPSY

There are so many factors that are responsible for epilepsy. Therefore, the epilepsy condition might be followed

by different components, including Birth injuries, birth asphyxia, head damage, cerebrum contaminations, hereditary qualities, a few sicknesses, and so on.

5.1 Inherited genetics

A few kinds of epilepsy are ordered by various sorts of seizures originating from the family history. There is likely a hereditary impact. Pathogenic changes or transformation in qualities and basic variations from the norm in chromosomes (cancellations, inclusions) is in charge of an assortment of epilepsies [10].

5.2 Head injury

Head trauma or damage because of an accident or another traumatic injury can cause epilepsy. Car crashes related to the poor direction of engine vehicle travel, and the absence of safety belts and caps for engine cycle riders may come about head damage [7].

5.3 Brain infections

A portion of the mind contamination that harms the cerebrum, for example, cerebrum tumor or stroke, can cause epilepsy. Stroke is the primary driver of epilepsy in grown-ups more established than age 35 [16]. Neurocysticercosis is most likely an imperative reason for seizures and epilepsy in areas with a high commonness of *Taeniasolium* contamination in people [17].

5.4 Birth trauma

Before birth, babies are susceptible to cerebrum harm that could be happened by different components, for instance, a disease in the mother, poor sustenance, and oxygen lack [18]. In this case, epilepsy may happen.

5.5 Diseases

Some irresistible disorders, for example, meningitis, AIDS, autism, neurofibromatosis, and viral encephalitis, may cause epilepsy [19].

5.6 Drugs

Epilepsy is also connected to withdrawal drugs [20] such as Barbiturates and so forth. Besides, some medicines also induce seizures, for example, Antidepressant: TCA-(Amitriptyline, Imipramine, and Clomipramine), Antipsychotics: Chlorpromazine, Mood Stabilizer: Lithium, Psychostimulant: Amphetamine, etc.

6. RISK FACTORS

A hazard factor makes a man more prone to developing a specific sickness or condition. For example, some particular aspects that may expand the danger of epilepsy are illustrated in **fig 4. (See Appendix)**



7. PATHOPHYSIOLOGY OF EPILEPSY

Epilepsy is related to surrendering an overabundance of mind nerves in the cerebrum that makes the body move [21]. Epileptogenesis is the process through which healthy brain tissue develops epilepsy [22]. The brain neural network develops a greater propensity for seizures [23] which results due to an imbalance between excitatory and inhibitor activity within a neuronal network [24], leading to excessive, aberrant synchronous neuronal firing in a region of the brain or throughout the brain [25] and thereby disrupting standard neuronal processing and is capable of disrupting other neuronal networks [24]. Another possible cause behind this hypersynchronous firing may be the critical construction of the network or any other structural or metabolic issues [25].

As the pathophysiology is unclear, several mechanisms have been proposed to understand the same. It has been suggested that abnormal neural pattern due to chromosomal abnormalities, malformation of cortical development, inflammation and irritation of neural tissue, infiltration of mass, grey matter irritation, enzyme deficiencies, and neurodegeneration contributes the damage to the brain and thereby alters the neuronal circuitry which leads to disturbance of neurotransmitter balance, increased inflammatory cytokines [26], altered neurogenesis and gliosis [26], and abnormal functioning of ion-channel and receptor causing an imbalance of the ion-channel charges. These changes cause the neurons to fire in burst activity mostly hypersynchronously, which further causes abnormal neural movements that form self-reinforcing circuits, ultimately leading to recurrent seizures.

Any procedure that demolishes the mind's cerebrum may bring about the initiation or deactivation of nerves. The primary neural transmission unit is a neurotransmitter, and the particulate channel is the natural part of the neural connection. Hereditary irregularities cause around 33% of epilepsy, for example, an expansion in extracellular k^+ and a decline in cell sugar fixation [27]. Notwithstanding these activity factors, this can make the seizure more compelling. E.g., Ach, aspartate, glutamate, and the inhibitory neurotransmitter GABA are additionally associated with the advancement of episodes. Moreover, when the change happened in GABA, NMDA receptor epilepsy happened. Different pathways to developing epilepsy are schematically represented in **Figures 5 and 6**. (See Appendix)

8. DIAGNOSIS OF EPILEPSY

Different types of diagnosis are available worldwide. However, the diagnosis depends on the patient's conditions, including seizures and epileptic syndrome. Several tests to diagnose epilepsy are:

8.1 Through history taking

The foremost vital things to diagnose epilepsy are to know reliable, valid family history information from patients and from an observer who observed the seizures [28].

8.2 Physical test

The physical test may incorporate neurological examination and blood test. In the neurological exam, the specialist may test the mental capacities, mental brokenness, and different zones to analyze the condition and decide the kind of epilepsy [29]. Again in a blood test, the specialist checks the indication of disease, hereditary conditions, and different variables related to epilepsy [30].

8.3 Laboratory test

Some of the lab tests are performed to analyze epilepsy. These are: S. Electrolytes, S. Prolactin, Blood sugar, CBC, TFT, LFT, RFT, and CSF [31, 32].

8.4 Imaging

Specialists may also suggest some tests to diagnose epilepsy, such as EEG, Video EEG telemetry, CT scan of the brain, MRI of the brain, MRS, PET, and SPECT [33-35].

8.5 Polysomnography

Polysomnography is a non-invasive strategy for break analysis. It records the brain's oxygen levels in the heart, heart breathing, and foot movements [36, 37].

8.6 Electroencephalogram (EEG)

Electroencephalogram (EEG) is the most widely recognized test to utilize as a part of diagnosing epilepsy [38]. Initially, glue is used to attach terminals to your scalp. Taking the test is simple and painless. You could be given a role in which you're expected to complete a specific task. The exam is done amid repose. The terminals will capture the electrical activity of your cerebrum. Whether you're experiencing a seizure or variations in familiar cerebrum wave shapes are normal in epilepsy [38].

9. MANAGEMENT OF EPILEPSY

Patients with epilepsy are managed with an emphasis on three primary goals: seizure control, side effect avoidance, and maintaining or regaining patient satisfaction. The Treatment plan depends on the accurate diagnosis of the seizure types. Epilepsy can be dealt with through numerous techniques, as shown in (Fig 7)(See Appendix) Normally pharmaceutical is expected to control seizures and treat epilepsy; these usually recommended drugs are called anticonvulsants [39]. However, medications alone can't generally stop or diminish attacks [40]. A gadget called a vagus nerve stimulator might help treat epilepsy if you don't get alleviation from the solution [41].



9.1 Conventional treatment

9.1.1 Drug treatment

Epilepsy is a heterogeneous and genuine mind issue. It contains numerous seizure composites and epilepsy disorders. It is traditional to talk about somebody having epilepsy, be that as it may, it may be better, especially in connection to advancing better medical treatment, if we somehow happened to think as far as one of the epilepsies [42]. About 70-80% of grown-ups with new beginning epilepsy will move toward becoming seizure free with current antiepileptic sedation. On the other hand, 20-30% keep on having drug-safe epilepsy with episodes. Different antiepileptic drugs are Diazepam, Carbamazepine, Clonazepam, Clobazam, Vigabatrin, Topiramate, Phenobarbital, etc.

9.1.2 Surgery treatment

When medicines fail to give satisfactory control over seizures, surgery might be an alternative way. With epilepsy surgery, a specialist expels the territory of your mind that is causing seizures [43]. In addition, surgery is needed to remove epileptic focus (e.g., mesial temporal sclerosis), Anterior Temporal Lobectomy, Corpus colostomy, and Subpial transaction [44].

9.1.3 Brain imaging

Cerebrum imaging is a modern innovation for subjective neuroscience, expanding on many years of intellectual brain research, behavioral molding, psychophysics, and mind science [45]. The accessibility of attractive reverberation imaging permits a more continuous etiological finding, diminishing the number of idiopathic cases.

9.1.4 Dietary manipulation

It additionally treats epileptic seizures. A portion of the dietary supplements is - Ketogenic, eating fewer carbs and establishing a ketogenic diet [46]. MCT eats fewer carbs, Atkins consumes fewer calories, and Oligoantigenic abstains from food.

9.1.5 Others

Illustrations incorporate statins, thalidomide, progesterone subordinators, montelukast, Brain cooling, antioxidant and free radical scavengers, immunosuppressive treatment [47], suppression of respiratory alkalosis, etc., are available.

9.2 Alternative therapy for epilepsy

Some elective treatments are likewise used to treat epilepsy. For example, Homeopathic, Yoga, Ayurveda, Herbal Medicine, and so on [48]. 13% of guardians utilized elective prescriptions for epilepsy treatment of their youngsters. Elective treatments, including pressure decrease procedures, can push a few people to control their seizures better.

9.2.1 Homeopathic treatment

Epilepsy can be treated with the homeopathic method of treatment. Regular homeopathic therapy for epilepsy is precious for treating intense fits and additionally in taking out the inclination to have seizures [49]. The consequences of the homeopathic treatment differ for each situation, depending on the length and sort of attack [50]. Some natural homeopathic cures include color, Cicuta [50], Artemisia, Stramonium, Cupremet, Bufo Rana, and so on [51]. These are the best solution for Epilepsy, where the assaults of shaking are set apart by a rough, contorted body shape. Homeopathic prescription Nux Vomica is of tremendous help when outrageous coldness is available with seizures.

9.2.2 Yoga for epilepsy

Epilepsy is a turmoil in which strange electrical releases in the cerebrum cause intermittent seizures. Most seizures can be controlled by antiepileptic drugs (AEDs), yet sometimes attacks create imperviousness to those medications. Individuals may likewise wish to attempt non-tranquilize medicines, for example, yoga [52]. For individuals who have epilepsy and related issues, it is critical to create, assess, and actualize a correlative treatment shown in the standard treatment of epilepsy. The antiquated Indian practice and reasoning of yoga are progressively becoming a point of convergence of therapy and research in epilepsy. Yoga encourages you to pick up control over your body, empowering you to deal with a seizure better without giving it a chance to get excessively extraordinary [43]. Yoga asanas enable you to extend your nerves and oxygenate your cerebrum [53]. They quiet your mind cells and keep them from getting energized. Yoga trains you to make an inward adjustment that backs off excitation [54]. It gives you profound rest, enabling the body to mend and repair itself. There are different sorts of yoga, including postural activities, breath control (pranayama), Uttanasana (Standing Forward Bend), Salamba Sirsasana (Head Stand), and reflection. In an investigation, the act of Sahaja yoga, a basic type of reflection, decreased seizures and EEG changes in individuals with epilepsy.

9.2.3 Ayurvedic treatment

Epilepsy influences more than 1.5-2% of all-inclusive communities worldwide, with higher commonness in creating locales [55]. Antiepileptic drugs, however powerful in symptomatically controlling seizures, do not counteract or reverse the obsessive procedure that underlies epilepsy. Even though antiepileptic drugs are mostly very much endured, there still is a need to look for new medications with fewer reactions and better viability. The ayurvedic solution looks to treat epilepsy by unblocking the heart and mind channels that the abundance of doshas or humor might obstruct. This opening of the tracks is worked on utilizing different inventions and laxatives. Oral hygiene is produced by oil and gaseous (pure oil), and external oil and rain are part of the treatment. The herbs examined for antiepileptic action in human examinations



were distinguished. It was intriguing to note that nearly 60 unique herbs said in Ayurveda writing have been contemplated for antiepileptic action (**Fig 8**) (**See Appendix**). Some of the Herbal formulation used is *Aswagandharishtam*, *Saraswatarishtam*, *Saraswati churna*, *Tiktaka ghruta*, *Apsmarari Rasayana*, *Smritisagara rasa*, *Chaturbuja rasa*, *Yogendra rasa*, *Albizia lebbeck* [52,53], *Aloe vera* [54], *Emblica Officinalis*, etc. The accessible writing focuses on the beneficial impact of herbs utilized as a part of Ayurveda therapeutics for epilepsy.

10. RECENT RESEARCH AND ACHIEVEMENT

Some current advances and accomplishments for epilepsy treatment for most, if not all, conventional AEDs act by decreasing over-the-top cortical sensitivity through the instrument of epileptogenesis [56]. In the previous ten years, there has been a surge in epilepsy sedate improvement, with no less than ten new antiepileptic drugs (AED) presented, such as felbamate, carbamazepine, lamotrigine, topiramate, and vigabatrin [57], etc. Nevertheless, numerous patients encounter symptoms or try not to have finished seizure control [58]. Some new bearings for treatment incorporate novel pharmacotherapies, biosensor-coupled transportation frameworks, quality and cell treatment & surgical approaches.

10.1 In drug treatment

Sodium valproate or lamotrigine is picked to start with first-line medications for absence seizures and incomplete episodes (54), and Valproate is essentially superior to Topiramate and Lamotrigine in the treatment of idiopathic generalized seizures [59].

10.2 Surgery

In creating nations, patients with Mesial TLE are attainable by a proficient group comprising epileptologist, neurosurgeon, and experts utilizing MRI and EEG [60].

10.3 Seizure after stroke

The general rate of seizures within 24 hrs when the stroke was 3.1%. A higher rate was seen in bleeding (8.4%). Seizures when stroke had higher mortality at 30 days when stroke [61].

10.4 Antiepileptic drug & pregnancy

As of late, proof regarding major congenital malformation (MCM) rates has been related to epilepsy and pregnancy registries. The latest information from the UK epilepsy and pregnancy enlist demonstrates a reasonable dosage-related impact with valproate chance 5% with 600 mg every day expanding to 11% at more than 1000 mg [62].

11. THE PERSPECTIVE OF EPILEPSY IN BANGLADESH

Bangladesh is a hugely populated nation. There is a considerable measure of issues; for example, irresistible

ailments, unhealthiness, and neurological confusion are most common in this nation. However, it is estimated that at least 1.5–2.0 million people in Bangladesh are living with epilepsy, despite the fact that there are no official measures available concerning the Prevalence of epilepsy [63]. According to reports, the incidence rate is about 5 per 1,000 people in affluent countries; however, it is more significant in poorer nations. Men are far more impacted than women, and rural residents are more afflicted than city dwellers [64]. As demonstrated by the normality of 10 out of 1,000 people, the amount of patients with epilepsy in Bangladesh is around 1.3 million. About 10-12 per 1000 people in Bangladesh are affected by epilepsy, and the most common ages are mostly 16 to 31 for epileptic patients [65]. According to the reports of Bangabandhu Sheikh Mujib Medical University, the prevalence rate of epilepsy from January 2008 to June 2010 is shown in **Fig. 9** [66] (**See Appendix**). The causes of epilepsy vary with age. In Bangladesh, the dominant part of individuals have superstitious beliefs, and thus, it functions as a solid hindrance for add up to the care of patients with epilepsy. A misjudging and negative state of mind of the guardians, relatives, and society towards epilepsy is common. In this manner, numerous patients with epilepsy are yet disregarded in analysis, treatment, training, restoration, and other social needs. Besides, childhood epilepsies are likewise not known in Bangladesh [67].

Epilepsy patients are frequently hesitant to seek doctors' counsel [68], or maybe they trust epilepsy has no cure and seek guidance from indigenous prescription professional 'Kabiraj,' wind charmer 'Ojha' and profound healers [69]. However, antiepileptic drugs [70] & some physiotherapy [71] are available in this country to treat epilepsy. Besides, epileptical surgery is finally given to the patient according to their need [72].

12. CONCLUSION

There have been noteworthy advances in comprehending the study of disease transmission of epilepsy in recent years [73]. The middle lifetime commonness rate is evaluated as six for every 1000 individuals, which is lower than in other creating areas. Nevertheless, today, the etiology of seizures often stays idiopathic [74]. As a rule, seizures can be controlled with medicines with minor symptoms. The correct finding in managing treatment is fundamental. Because of the expanding number of epileptic seizures worldwide, researchers are looking for a superior method to fix illnesses with fewer symptoms. As of late, researchers have imagined that it would be imperative to test if embryonic stem cell transplant may be compelling to repair harms caused by epilepsy. Furthermore, governments should identify centers equipped with physical offices like video-electroencephalography and MRI and prioritize the development of an epileptic medical process for the treatment of medication-safe epilepsy.



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13. REFERENCES

1. Milton, J. and P. Jung, *Epilepsy as a dynamic disease*. 2013: Springer Science & Business Media.
2. Mazumder, A.G., et al., *Ginkgo biloba L. attenuates spontaneous recurrent seizures and associated neurological conditions in lithium-pilocarpine rat model of temporal lobe epilepsy through inhibition of mammalian target of rapamycin pathway hyperactivation*. *Journal of ethnopharmacology*, 2017. 204: p. 8-17.
3. Niemiec, R.M., *Movies and mental illness: Using films to understand psychopathology*. 2014: Hogrefe Publishing.
4. Bell, G.S., A. Neligan, and J.W. Sander, *An unknown quantity—the worldwide Prevalence of epilepsy*. *Epilepsia*, 2014. 55(7): p. 958-962.
5. Valerie Jewells, H.W.S., *Review of Epilepsy - Etiology, Diagnostic Evaluation and Treatment*. *International Journal of Neurorehabilitation*, 2014. 01(03).
6. Surges, R., et al., *Sudden unexpected death in epilepsy: risk factors and potential pathomechanisms*. *Nat Rev Neurol*, 2009. 5(9): p. 492-504.
7. Mac, T.L., et al., *Epidemiology, aetiology, and clinical management of epilepsy in Asia: a systematic review*. *The Lancet Neurology*, 2007. 6(6): p. 533-543.
8. Yemadje, L.P., et al., *Understanding the differences in Prevalence of epilepsy in tropical regions*. *Epilepsia*, 2011. 52(8): p. 1376-81.
9. Ullah, S., et al., *Shakir, Niaz ali, aslam khan and M.Nabi. The Prevalence, Incidence and Etiology of Epilepsy*. *International Journal of Clinical and Experimental Neurology*, 2014, Vol. 2, No. 2, 29-39. 2014.
10. Ngugi, A.K., et al., *Incidence of epilepsy A systematic review and meta-analysis*. *Neurology*, 2011. 77(10): p. 1005-1012.
11. Berg, A.T. and J.J. Millichap, *The 2010 revised classification of seizures and epilepsy*. *CONTINUUM: Lifelong Learning in Neurology*, 2013. 19(3, Epilepsy): p. 571-597.
12. Ucar, H., et al., *Comparison of 2017 ILAE and Semiological seizure classifications before and after video-EEG monitoring in childhood epilepsy*. 2022. 26(7): p. 2343-2352.
13. Lesser, R.P., et al., *Simple partial seizures, in Epilepsy: electroclinical syndromes*. 1987, Springer. p. 223-278.
14. Devinsky, O., et al., *Clinical and electroencephalographic features of simple partial seizures*. *Neurology*, 1988. 38(9): p. 1347-1347.
15. O'leary, D.S., et al., *Effects of age of onset of partial and generalized seizures on neuropsychological performance in children*. *The Journal of nervous and mental disease*, 1983. 171(10): p. 624-629.
16. Shih-Hui, L., *Epidemiology and etiology of seizures and epilepsy in the elderly in Asia*. *Neurology Asia*, 2004. 9(Supplement 1): p. 31-32.
17. Tran, D.-S., et al., *Prevalence of epilepsy in a rural district of central Lao PDR*. *Neuroepidemiology*, 2006. 26(4): p. 199-206.
18. Wilson, J.G., *Environmental effects on development—teratology, in Fetal-Placental Disorders*. 1972, Elsevier. p. 269-320.
19. Senanayake, N. and G.C. Román, *Epidemiology of epilepsy in developing countries*. *Bulletin of the world health organization*, 1993. 71(2): p. 247.
20. Gillberg, C., *The treatment of epilepsy in autism*. *Journal of Autism and Developmental Disorders*, 1991. 21(1): p. 61-77.
21. Kotulak, R., *Inside the brain: Revolutionary discoveries of how the mind works*. 1997: Andrews McMeel Publishing.
22. Dudek, F.E. and K.J.J.J.S.B.M.o.t.E.t.e. Staley, *The time course and circuit mechanisms of acquired epileptogenesis*. 2012.
23. Pitkänen, A., et al., *Epileptogenesis*. 2015. 5(10): p. a022822.
24. Falco-Walter, J. *Epilepsy—definition, classification, pathophysiology, and epidemiology*. in *Seminars in Neurology*. 2020. Thieme Medical Publishers, Inc.
25. Vezzani, A., et al., *The role of cytokines in the pathophysiology of epilepsy*. 2008. 22(6): p. 797-803.
26. Patel, D.C., et al., *Neuron–glia interactions in the pathophysiology of epilepsy*. 2019. 20(5): p. 282-297.
27. Browne, T.R. and G.L. Holmes, *Handbook of epilepsy*. 2008: Jones & Bartlett Learning.
28. Gilliam, F. and A.M. Kanner, *Treatment of depressive disorders in epilepsy patients*. *Epilepsy & Behavior*, 2002. 3(5): p. 2-9.
29. Myers, M.F. and G.O. Gabbard, *The physician as patient: a clinical handbook for mental health professionals*. 2009: American Psychiatric Pub.
30. Doose, H. and W. Baier, *Benign partial epilepsy and related conditions: multifactorial pathogenesis with hereditary impairment of brain maturation*. *European journal of pediatrics*, 1989. 149(3): p. 152-158.
31. Thakkar, K., R. Mariappan, and B. Nair, *Detection and management of intraoperative seizure with bispectral index monitoring in a paralyzed patient*. *Neurology India*, 2017. 65.
32. Ben-Menachem, E., *Is prolactin a clinically useful measure of epilepsy?* *Epilepsy currents*, 2006. 6(3): p. 78.
33. Brodbeck, V., et al., *Electrical source imaging for presurgical focus localization in epilepsy patients with normal MRI*. *Epilepsia*, 2010. 51(4): p. 583-591.
34. Hyde, T.M. and D.R. Weinberger, *Seizures and schizophrenia*. *Schizophrenia bulletin*, 1997. 23(4): p. 611-622.
35. Duncan, J.S., et al., *Brain imaging in the assessment for epilepsy surgery*. *The Lancet Neurology*, 2016. 15(4): p. 420-433.
36. Krishna, J., *Polysomnography and MSLT 49. Principles and Practice of Pediatric Sleep Medicine E-Book*, 2014. 35(11): p. 399.



37. Oldani, A., et al., Autosomal dominant nocturnal frontal lobe epilepsy. A video-polysomnographic and genetic appraisal of 40 patients and delineation of the epileptic syndrome. *Brain: a journal of neurology*, 1998. 121(2): p. 205-223.
38. Flink, R., et al., Guidelines for the use of EEG methodology in the diagnosis of epilepsy: International League Against Epilepsy: Commission Report Commission on European Affairs: Subcommission on European Guidelines. *Acta Neurologica Scandinavica*, 2002. 106(1): p. 1-7.
39. Patsalos, P.N., et al., Antiepileptic drugs—best practice guidelines for therapeutic drug monitoring: a position paper by the subcommission on therapeutic drug monitoring, ILAE Commission on Therapeutic Strategies. *Epilepsia*, 2008. 49(7): p. 1239-1276.
40. Conrad, P., The meaning of medications: another look at compliance. *Social science & medicine*, 1985. 20(1): p. 29-37.
41. Meneses, M.S., et al., Vagus nerve stimulation may be a sound therapeutic option in the treatment of refractory epilepsy. *Arquivos de neuro-psiquiatria*, 2013. 71(1): p. 25-30.
42. Feely, M., Fortnightly review: drug treatment of epilepsy. *BMJ: British Medical Journal*, 1999. 318(7176): p. 106.
43. Téllez-Zenteno, J.F., R. Dhar, and S. Wiebe, Long-term seizure outcomes following epilepsy surgery: a systematic review and meta-analysis. *Brain*, 2005. 128(5): p. 1188-1198.
44. Daniel, R. and M. Chandy, Epilepsy surgery: overview Of forty years experience. *Neurology India*, 1999. 47(2): p. 98.
45. Binder, J.R., Brain Imaging in Epilepsy. *The Lancet Neurology*, 2004. 3(4): p. 255.
46. Dixon, M., Ketogenic diet for epilepsy. *Clinical paediatric dietetics*, 2001: p. 222-232.
47. Jung, S., et al., The immunosuppressant cyclosporin A inhibits recurrent seizures in an experimental model of temporal lobe epilepsy. *Neuroscience letters*, 2012. 529(2): p. 133-138.
48. Saxena, V.S. and V.V. Nadkarni, Nonpharmacological treatment of epilepsy. *Ann Indian Acad Neurol*, 2011. 14(3): p. 148-52.
49. Panayiotopoulos, C., Epileptic syndromes and their treatment. *Neonatal Seizures*, 2007: p. 185-206.
50. Ricotti, V. and N. Delanty, Use of complementary and alternative medicine in epilepsy. *Current neurology and neuroscience reports*, 2006. 6(4): p. 347-353.
51. Bum, E.N., et al., Antiepileptic medicinal plants used in traditional medicine to treat epilepsy, in *Clinical and genetic aspects of epilepsy*. 2011, InTech.
52. Panebianco, M., K. Sridharan, and S. Ramaratnam, Yoga for epilepsy. *Cochrane Database Syst Rev*, 2015(5): p. CD001524.
53. Power, A.Y.P.T.I.Y.B., Yoga for brain improvement. 2017.
54. Lundgren, T., et al., Acceptance and commitment therapy and yoga for drug-refractory epilepsy: a randomized controlled trial. *Epilepsy & Behavior*, 2008. 13(1): p. 102-108.
55. Wilkinson-Meyers, L., et al., Met and unmet need for personal assistance among community-dwelling New Zealanders 75 years and over. *Health & social care in the community*, 2014. 22(3): p. 317-327.
56. Wyllie, E., et al., Wyllie's treatment of epilepsy: principles and practice. 2012: Lippincott Williams & Wilkins.
57. Goldenberg, M.M.J.P. and Therapeutics, Overview of drugs used for epilepsy and seizures: etiology, diagnosis, and treatment. 2010. 35(7): p. 392.
58. Semah, F., et al., Is the underlying cause of epilepsy a major prognostic factor for recurrence? *Neurology*, 1998. 51(5): p. 1256-1262.
59. Brigo, F., S.C. Igwe, and S.J.C.D.o.S.R. Lattanzi, Ethosuximide, sodium valproate or lamotrigine for absence seizures in children and adolescents. 2021(1).
60. Singh, V.J.J.o.P.N., Epilepsy surgery in India. 2011. 6(Suppl1): p. S130.
61. Szaflarski, J.P., et al., Incidence of seizures in the acute phase of stroke: a population-based study. 2008. 49(6): p. 974-981.
62. Wlodarczyk, B.J., et al., Antiepileptic drugs and pregnancy outcomes. 2012. 158(8): p. 2071-2090.
63. foundation, B.e. epilepsy. Available from: <https://www.ilae.org/regions-and-countries/national-chapters/bangladesh>.
64. Mannan, M., Epilepsy in Bangladesh. *Neurol Asia*, 2004. 9(1): p. 18.
65. Mannan, M.J.N.A., Epilepsy in Bangladesh. 2004. 9(1): p. 18.
66. Rahman, M.M. and K.J.J.o.e.r. Fatema, Epilepsy in children with tuberous sclerosis complex: a prospective observational study in Bangladesh. 2020. 10(1): p. 18.
67. Banu, S.H., et al., Profile of childhood epilepsy in Bangladesh. *Developmental medicine and child neurology*, 2003. 45(7): p. 477-482.
68. Anwarullah, A., et al., Attitude of family and community towards epilepsy. *Neuron*, 2004. 4: p. 14-20.
69. Epilepsy, I.B.F., Epilepsy in Bangladesh. 2016.
70. Habib, M., et al., Antiepileptic drug utilization in Bangladesh: experience from Dhaka Medical College Hospital. *BMC research notes*, 2013. 6(1): p. 473.
71. Mamin, F.A. and R. Hayes, Physiotherapy in Bangladesh: inequality Begets inequality. *Frontiers in public health*, 2018. 6: p. 80.
72. Chowdhury, F., et al., Microneurosurgical management of temporal lobe epilepsy by amygdalohippocampectomy (AH) plus standard anterior temporal lobectomy (ATL): a report of our initial five cases in Bangladesh. *Asian journal of neurosurgery*, 2010. 5(2): p. 10.
73. Epilepsy, G.C.A., Epilepsy in the Western Pacific Region. A call to action. Geneva: WHO, 2004.
74. Jallon, P. and P. Latour, Epidemiology of idiopathic generalized epilepsies. *Epilepsia*, 2005. 46: p. 10-14.



APPENDIX

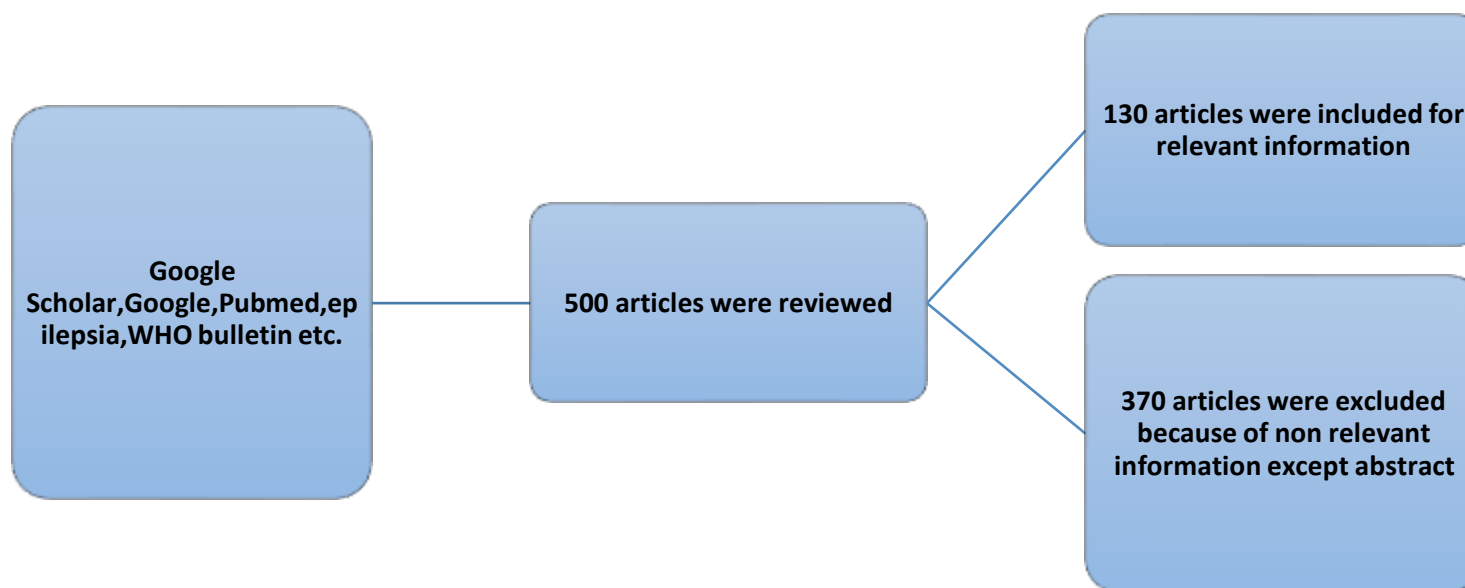


Fig 1. Article search protocol

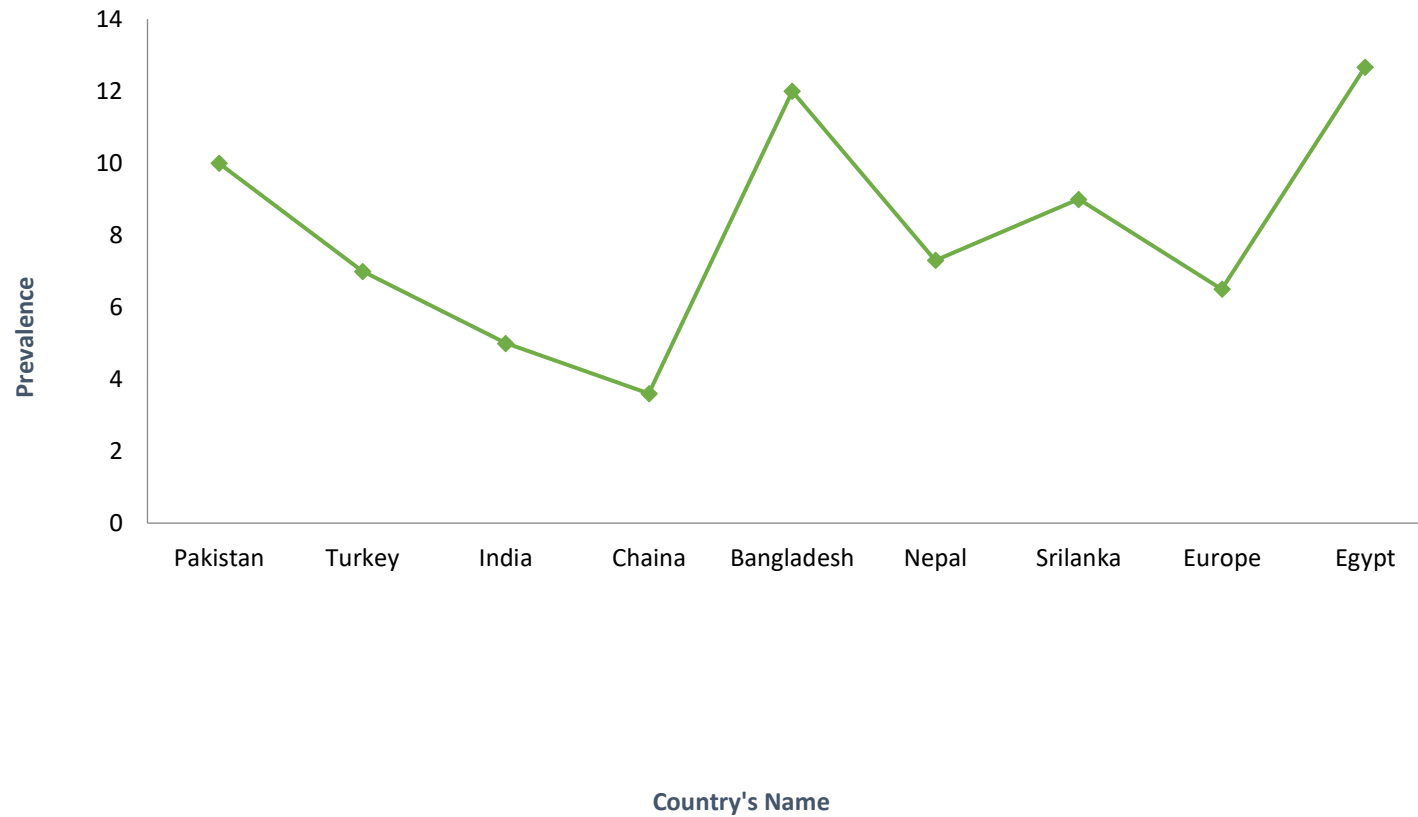


Fig 2. Prevalence of epilepsy in a different region

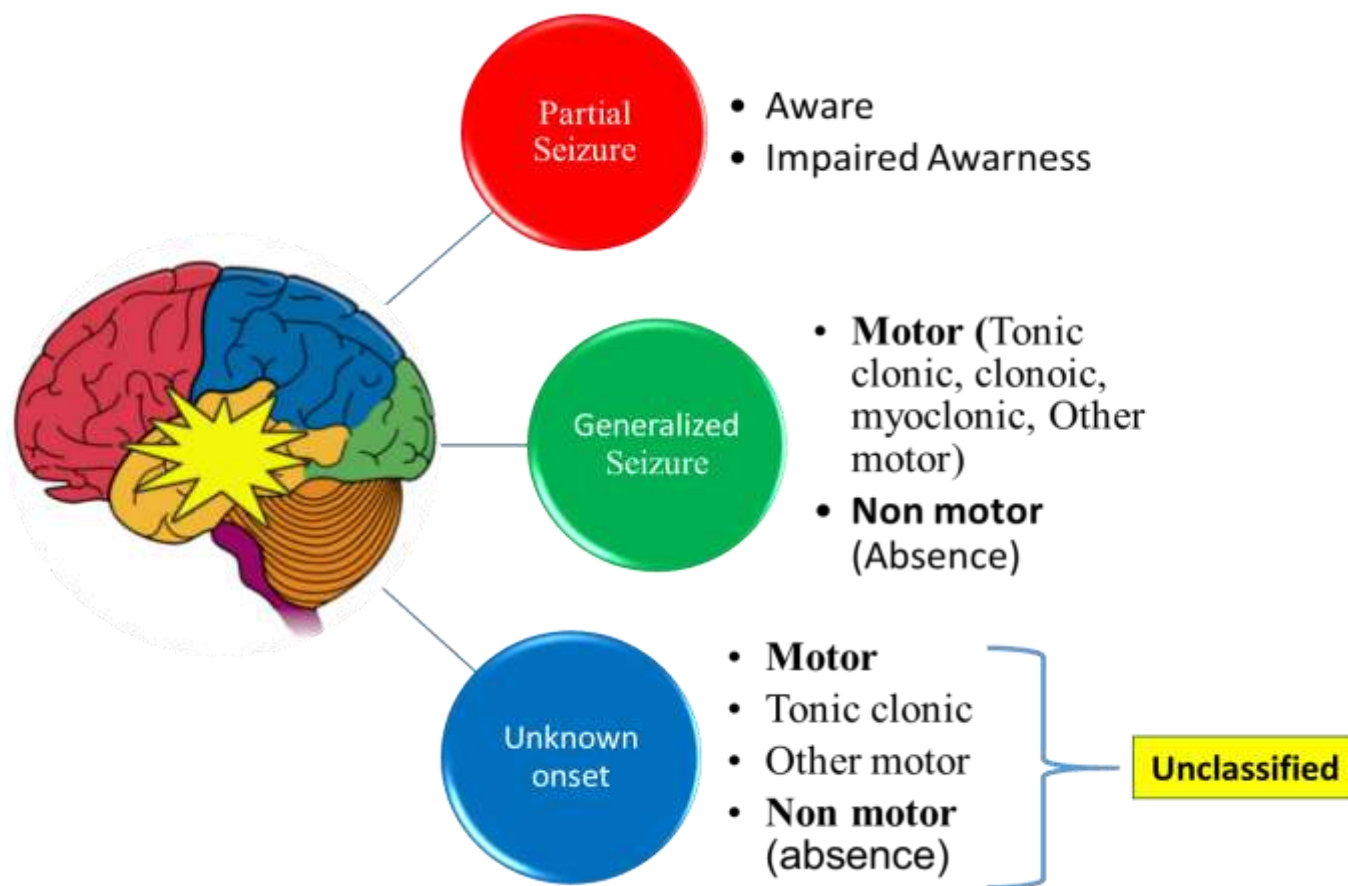


Fig 3. ILEA Classification of Epilepsy

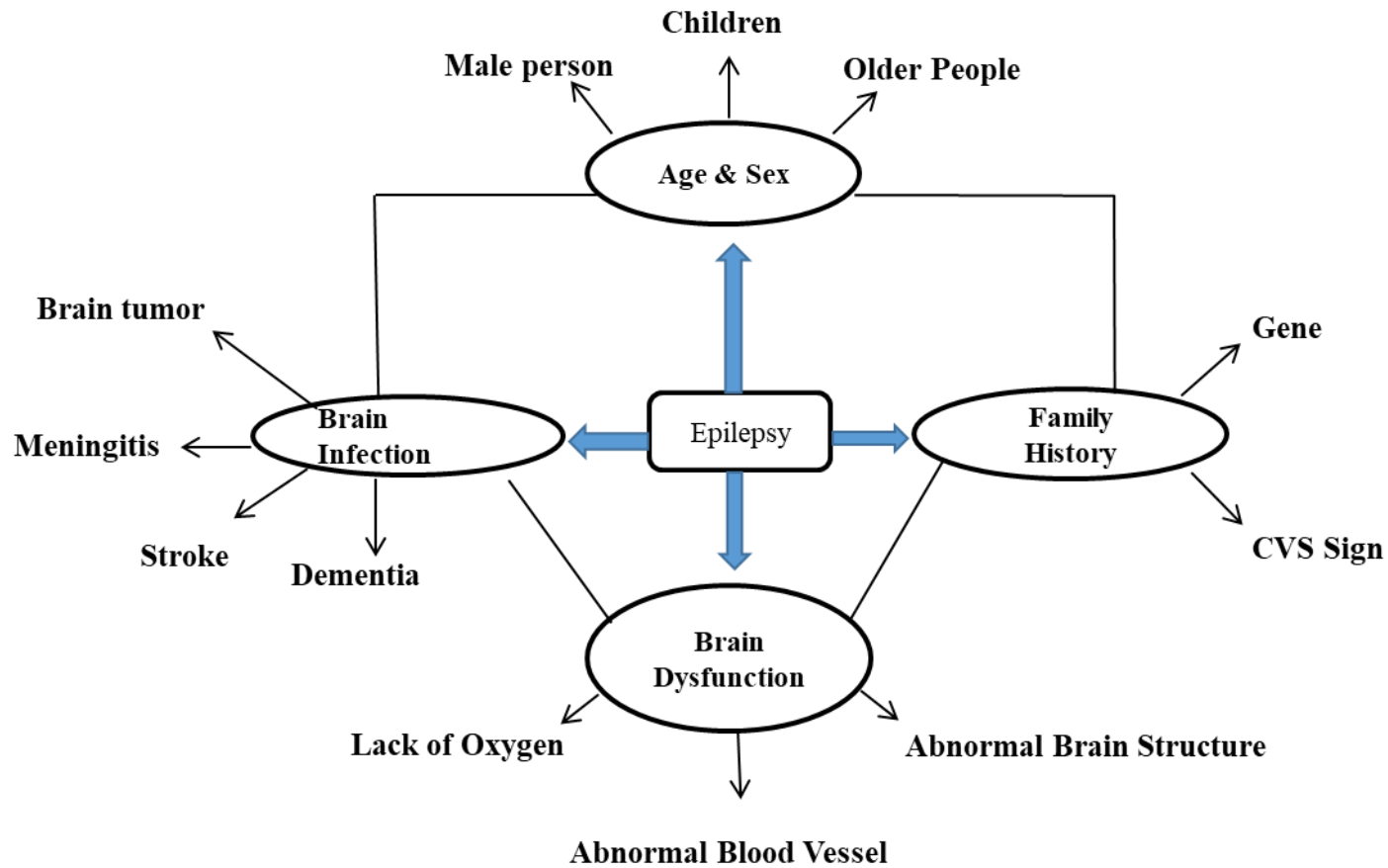


Fig 4. Schematic diagram of certain risk factors which influence epilepsy

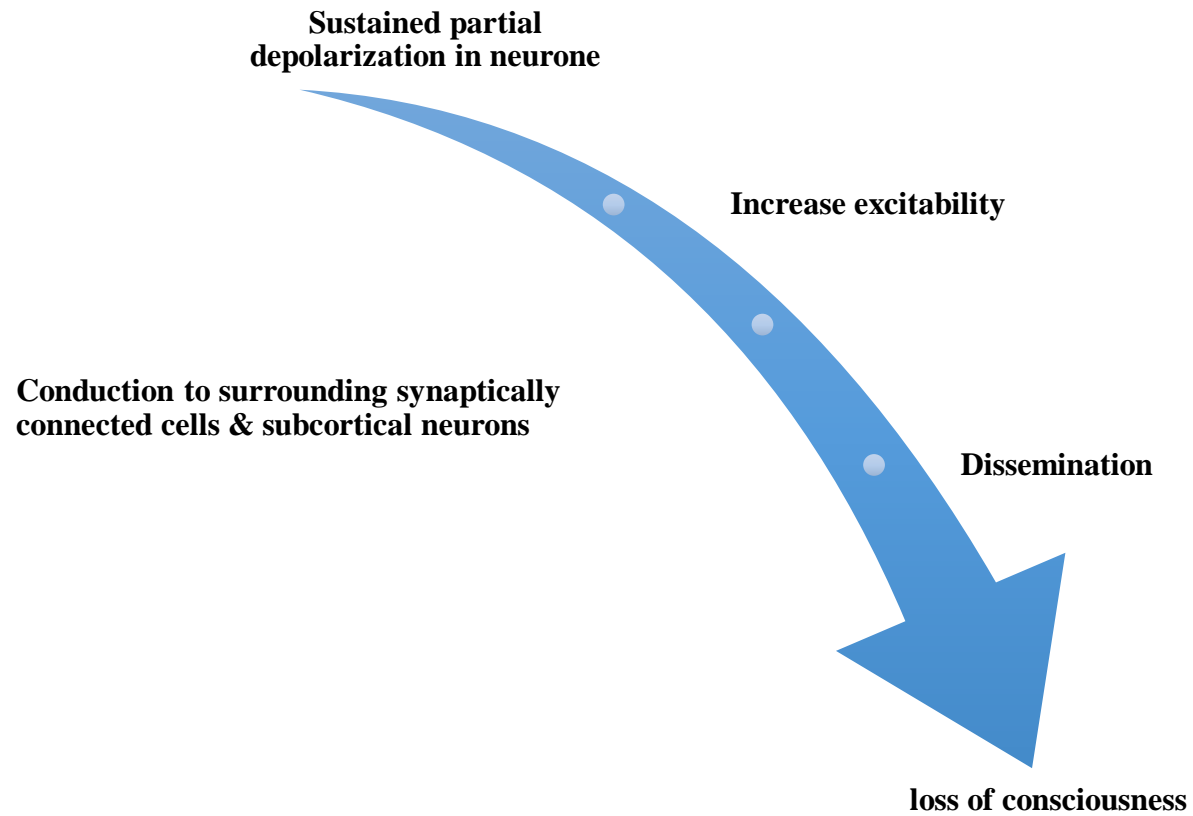


Fig 5. Pathogenesis of Epilepsy

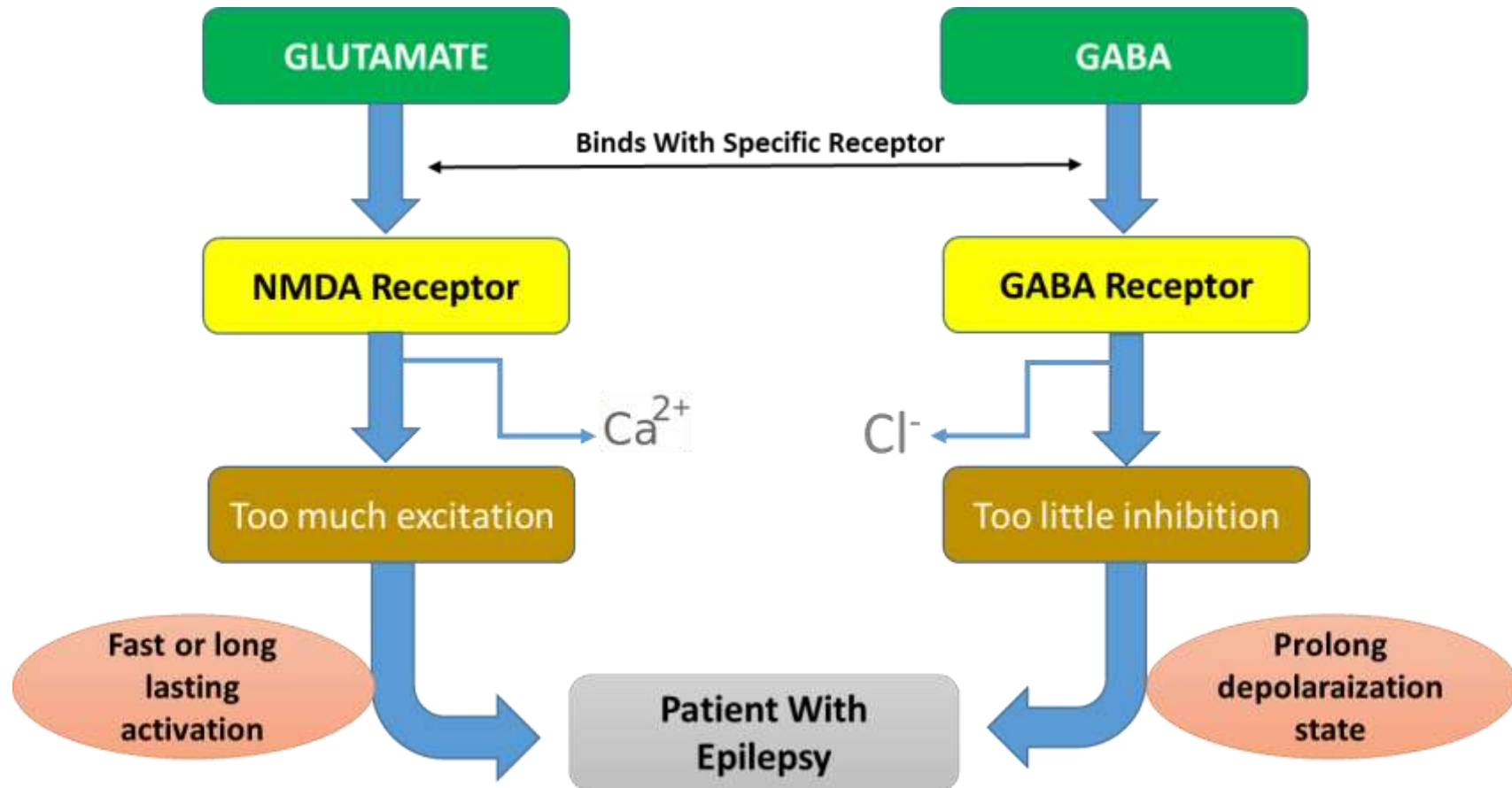


Fig 6. Role of glutamate & GABA to influence epilepsy

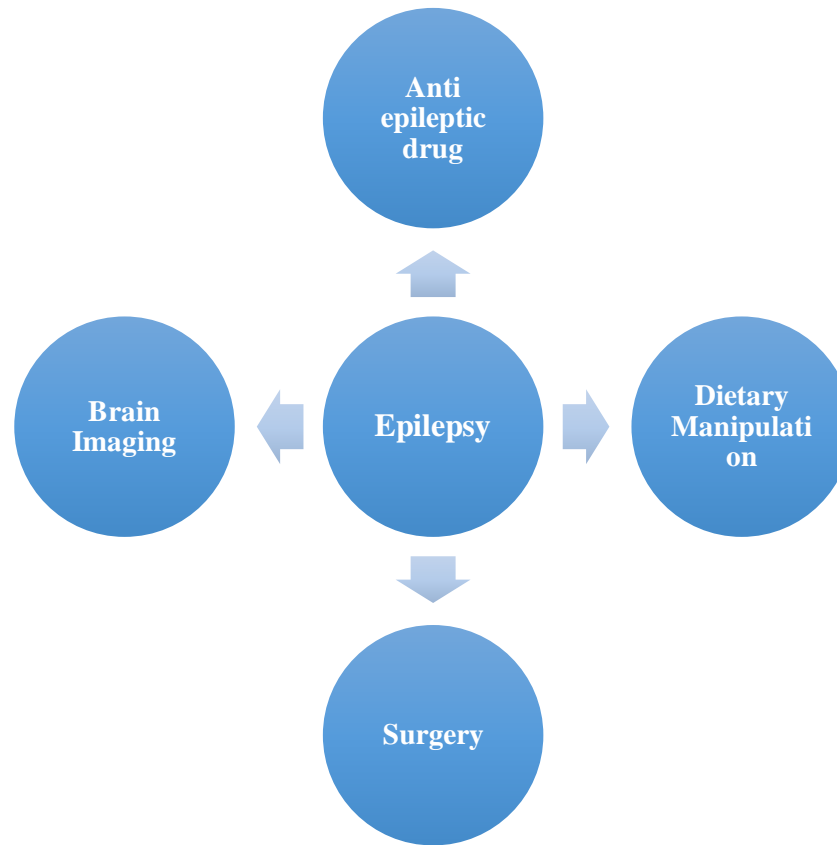


Fig 7. Treatment option for epilepsy

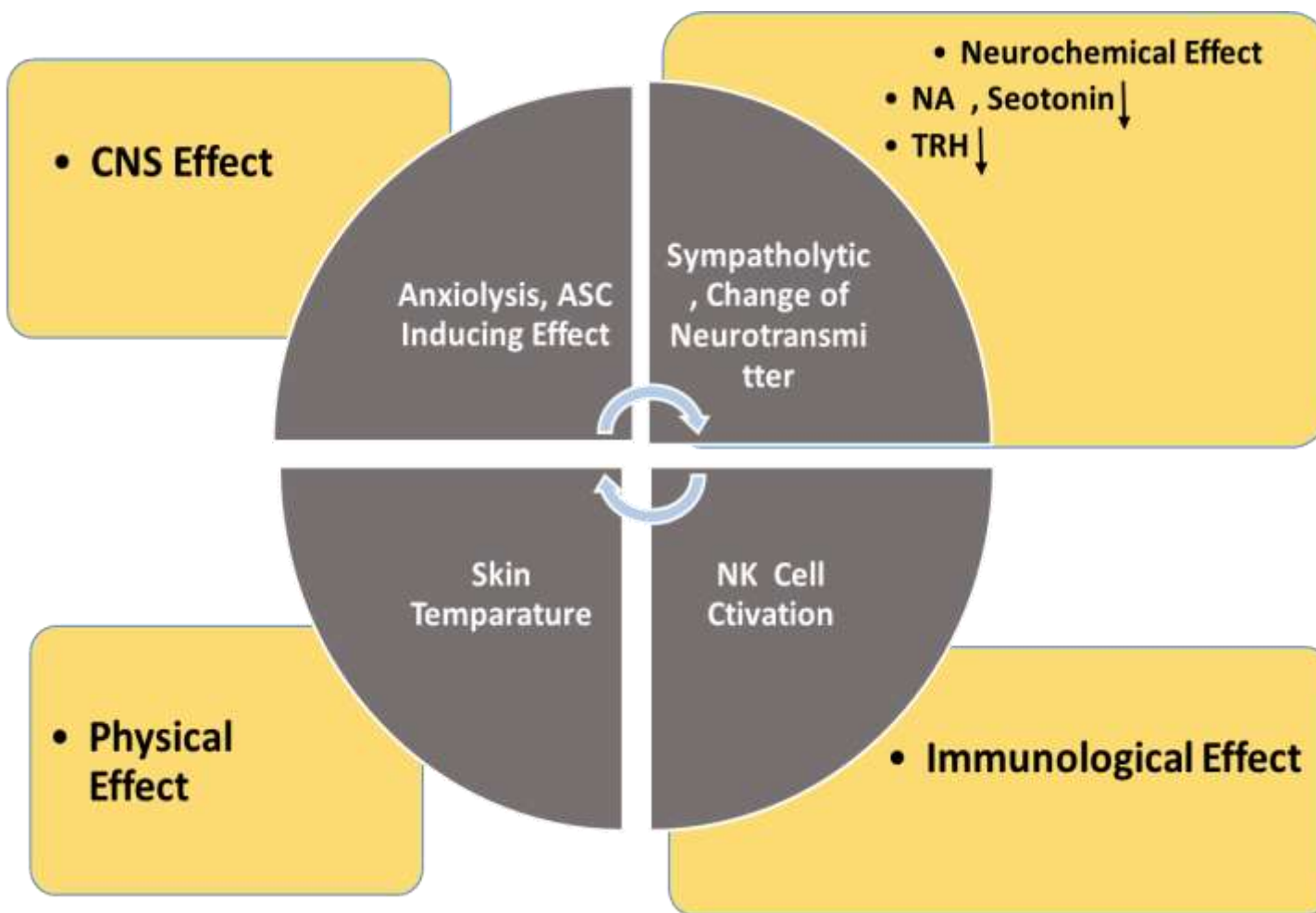


Fig 8. Shirodhara induced CNS effects, immunological & physical Change

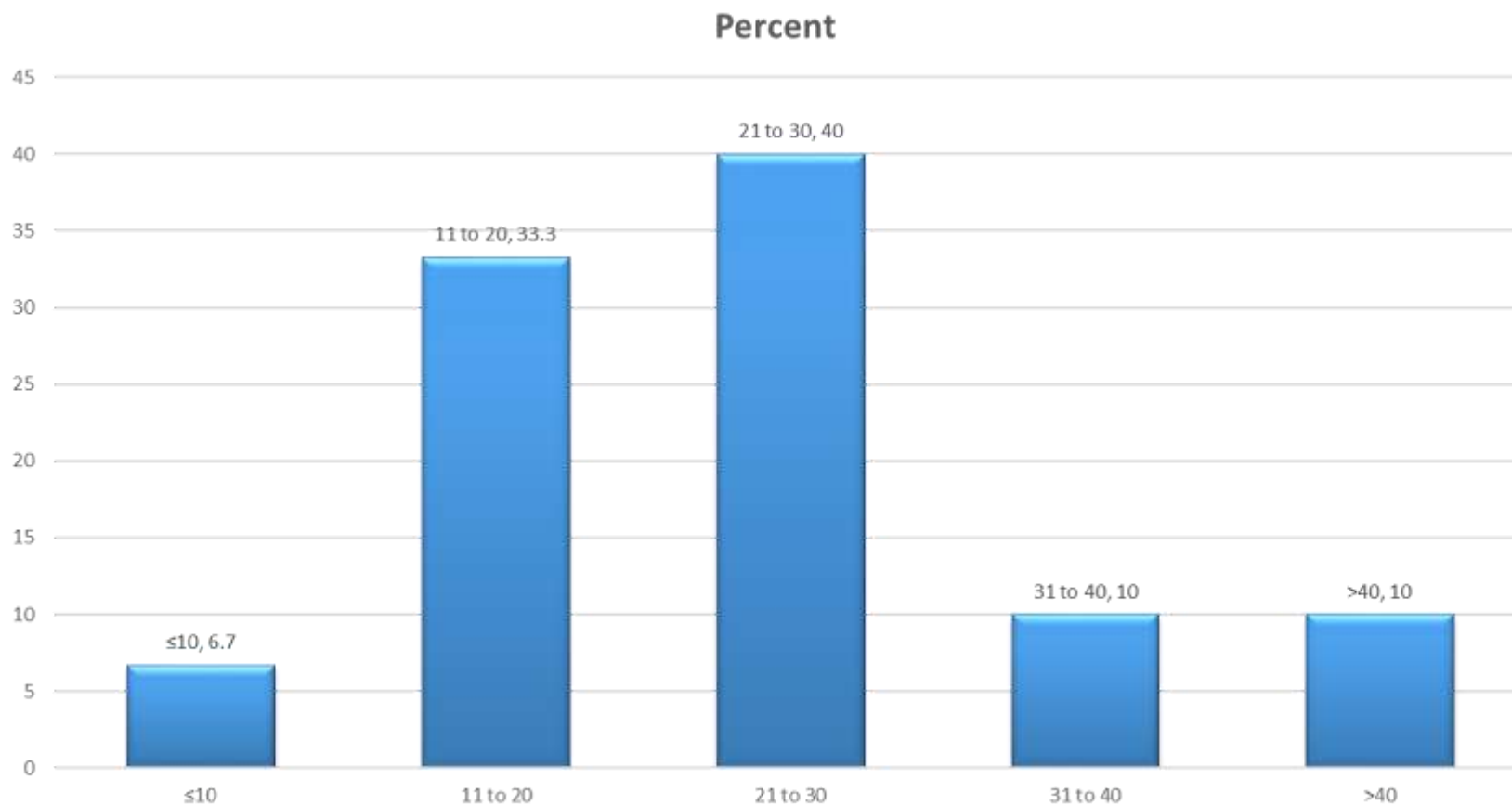


Fig 9. The age-specific prevalence rate of epilepsy in Bangladesh