REVIEW OF LITERATURE
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1) Historical aspects

Epilepsy has the longest medical history (Rock & Knight 1947) as it was recognised in India before the 10th century B.C. (Joshi, 1971). Epilepsy is defined as "Apaśmara" in ancient Indian medical texts. The prefix "Apa" meaning negation or loss & "Smara" meaning recollection of consciousness. Charak described epilepsy as "paroxysmal loss of consciousness due to disturbance of memory and understanding of mind attended with convulsive seizures.

Epilepsy has been described in Greek literature as early as fifth century BC. Famous persons in world like Alexander the great, Julius Caesar, St. Paul Napoleon, Lord Byron & Maupassant had epilepsy.

By careful pathological & clinical correlative studies, Hughlings Jackson was first to interpret various manifestations of psycho-motor seizures on a sound anatomical basis. He was one of the first to stress that psychic phenomena are as much manifestations of epileptic discharge as the more spectacular tonic & clonic contractions.

Nomenclature

Various terms have been used, see has falling disease, fits, convulsions, seizure & epilepsy. The term ‘Epilepsy’ derived
"The Greek words meaning "to seize upon" or "taking hold of" is not a single disease, but a group of diseases manifesting in a common way correct word nowadays is practice in epilepsies or epilepsies and epileptic syndromes.

To define epilepsy precisely is not easy. Epilepsy are said to be a "tendency to recurring epileptic seizures" (Farley & Frickman 1941) it does not seem sufficient. To define epilepsy as paroxysmal cerebral dysrhythmia is also insufficent. Hagling Jackson described epilepsy as recurrent seizures due to intermittent derangement of nervous system as a result of sudden excessive disorderly discharge of cerebral neurons. Best possible definition of epilepsies given by Dichter (1967) in "Epilepsies are a group of disorders characterised by chronic recurrent paroxysmal alterations in neurological function caused by abnormalities in the electrical activity of brain".

Classification

As a basis to any discussion on classification of epileptic phenomena we need to be clear about distinctions between classification of epileptic seizures as opposed to those which apply to epilepsies themselves. With seizures we are primarily concerned with clinical manifestations and with their neurophysiological basis, whereas with epilepsies we are more concerned with grouping of seizures or syndromes, their natural histories and their underlying causes. Failure to observe these distinctions has often led to confusion in the past and still seems to be doing so, therefore at present time both of below
partially classified (classification of epileptic seizures 1981 and classification of epilepsies 1981) are relevant.

CLASSIFICATION OF EPILEPTIC SEIZURES
(Modified from classification of I.L.E.E.1981)

PARTIAL SEIZURES (Focal, Local)
1. Simple partial seizures:
   - With motor signs
   - With somatosensory or special sensory symptoms
   - With autonomic symptoms or signs.
   - With psychic symptoms.

2. Complex partial seizures (with impairment of consciousness)
   - simple partial onset followed by impairment of consciousness
   - with impairment of consciousness at onset.
   (a) — With impairment of consciousness only.
   (b) — With automatism.

3. Partial seizures secondarily generalised.

GENERALISED SEIZURES:

(1) Non-convulsive seizures:

(a) Absence seizures.

(b) Atypical absence seizures.
(c) Myoclonic seizures.
(d) Atonic seizures.

(2) Convulsive seizures -

(a) Tonic–clonic seizures.

(b) Tonic seizures.

(c) Clonic seizures.

UNCLASSIFIED EPILEPTIC SEIZURES.

CLASSIFICATION OF EPILEPSIES AND EPILEPTIC SYNDROMES

(Modification of ILAE 1985 classification proposed in 1989 by commission on classification and terminology of ILAE.)

A. LOCALISATION RELATED (LOCAL, FOCAL, PARTIAL) EPILEPSIES AND SYNDROMES:

(1) Idiopathic (with age related onset)
   - Benign childhood epilepsy with centrotemporal spikes
   - Childhood epilepsy with occipital paroxysms
   - Primary reading epilepsy.

(2) Symptomatic :
   - Chronic progressive epilepsy partialis continua.
   - Syndromes characterized by specific modes of precipitation
   - Temporal lobe epilepsies.
   - Frontal lobe epilepsies.
   - Parietal lobe epilepsies.
   - Occipital lobe epilepsies.
(3) Cryptogenic (cause not known)

B. GENERALISED EPILEPSIES AND SYNDROMES.

(1) Idiopathic

- Benign neonatal familial convulsions.
- Benign neonatal convulsions.
- Benign epilepsy in infancy.
- Childhood absence epilepsy.
- Juvenile absence epilepsy.
- Juvenile myoclonic epilepsy.
- Epilepsy with generalised tonic clonic seizures (GTCS) on awakening.
- Other generalised idiopathic epilepsies.
- Epilepsies with seizures precipitated by specific modes of activation (Reflex epilepsies).

(2) Cryptogenic

- West syndrome.
- Lennox Gastaut syndrome.
- Epilepsy with myoclonic astatic seizures.

(3) Symptomatic

(i) Non-specific aetiology:
- Early myoclonic encephalopathy.
- Early infantile encephalopathy with suppression bursts.
- Other symptomatic generalised epilepsies.
(ii) Specific syndromes
   - Epileptic seizures complicating other disease states.

(C) Epilepsies and syndromes undetermined whether focal or generalised.

(1) With both generalised and focal seizures.
   - Neonatal seizures.
   - Severe myoclonic epilepsy of infancy.
   - Epilepsy with continuous spike and waves during slow wave sleep.
   - Acquired epileptic aphasia.
   - Other undetermined epilepsies.

(2) Without unequivocal generalised or focal features.

(D) Special syndromes
   - Situation related seizures
   - Febrile convulsions.
   - Isolated seizures or isolated status epilepticus.
   - Seizures occurring only with acute metabolic or toxic events.

The important modifications in 1989 classification of epilepsies and epileptic syndromes proposed by ILAE are

1- Differentiation between the term "idiopathic" and "Cryptogenic" and inclusion of latter subtype. Idiopathic epilepsy has no underlying cause and is defined by age related onset, clinical and electroencephalographic characteristics and a presumed genetic etiology. The term cryptogenic refers to a disorder whose cause is hidden or occult.
2. Inclusion of primary reading epilepsy as an additional entity in Localisation related Idiopathic epilepsies and syndromes.

3. Inclusion of chronic progressive epilepsy partialis continua in the Localisation related symptomatic epilepsies and syndromes.

4. Specific mention of anatomical Cortical seizure Localisation in subgroup Localisation related symptomatic epilepsies and syndromes.

5. Another important inclusion is the category of epilepsies with seizures precipitated by specific mode of activation. The classic example of this group is reflex epilepsies for example—Hotwater epilepsy, eating epilepsy, Startle epilepsy.

**Epidemiology**

The overall prevalence rate of epilepsy in general population varies from as low 3-4 per 1000 to as high as 10.5 per thousand. Large variations in prevalence rates are attributed mainly to different criteria for selection of cases. A higher prevalence rate in developing countries is expected due to increase in birth trauma and infections.

In vast majority of studies males tend to predominate presumably due to more frequent head injuries. The commonest age of onset of epilepsies is 0-4 years. The incidence rates are higher within first decade, somewhat lower in second decade and
then become low. The lowest prevalence rates which usually occur in first decade, increase in second decade and show a decline after 50 years.

**AETIOPATHOGENESIS**

Li and Jasper (1961) have clearly demonstrated that the epileptic process consists fundamentally of hyperactive and hypersynchronous neuronal discharges. In chronic epilepsies the recurrent neuronal paroxysms that underlie ictal events are transient expression of a more permanently physiologically disordered cortex.

The typical interictal EEG spike and wave complex reflects the summation of synchronised abnormal neuronal membrane potentials consisting of large paroxysmal depolarization shifts followed by prolonged after hyperpolarization. The depolarization shift results in enhanced neuronal excitation, while after hyperpolarization represents inhibition that may prevent ictal episode. Neurones in the cortical areas surrounding an epileptic focus may demonstrate paroxysmal hyperpolarization only forming an inhibitory zone, which appears to prevent epileptic spread during interictal period.

Despite knowledge of neuronal defects that can destabilize the membrane or interfere with balance of excitatory and inhibitory synaptic activity, fundamental mechanisms underlying spontaneous recurrent seizures in epilepsies remain to be unknown. The neuronal basis of seizure termination is mainly due
to self activating inhibitory mechanisms rather than neuronal 
extinction.

Site of origin of generalised tonic-clonic seizures is 
accepted as thalamic intralaminar system and mid brain spreading 
to cerebral cortex in bilaterally symmetrical and synchronous 
manner manifested in EEG as generalised spike and wave 
pattern. This condition has been termed "Corticoreticular 
epilepsy".

In partial epilepsies an epileptic discharge can be assumed 
to be originating from a particular part of cerebral cortex. In 
some of the partial epilepsies, epileptic discharges though 
originating in one hemisphere is reflected as mirror image focus 
in the contralateral homologous area due to spread through 
commissural connections. The amplitude of the mirror image focus 
is usually smaller. So in cases of partial epilepsies, EEG findings 
are confirmatory in localisation of epileptic foci when findings 
are limited to specific lobe which is unusual.

Secondary generalization of partial epilepsies occurs 
through reticulothalamic cortical pathway of spread or through 
wide spread of discharges to same hemisphere from epileptic foci 
and spread via commisural connections to contralateral 
hemisphere.

In cases of complex partial seizures 90% of seizures 
originate from temporal lobe, usually medial aspect of temporal 
lobe. In remaining cases frontal lobe is commonly involved. 
Disturbance in consciousness favours involvement of
centrocephalic region.

Inhibitory neurotransmitter gamma amino butyric acid (GABA) may be a natural anticonvulsant found in the brain. GABA and acetyl choline have opposite effects upon neuronal excitability and an imbalance between these two substances could be predisposing factor for seizure activity (Jurgelsky and Thomas, 1966).

AETIOLOGY

Commonly combination of cerebral insult and a genetic predisposition determines the appearance of epileptic seizures.

Genetic factors may contribute in three ways:

(i) An individual may inherit a low threshold for seizures as in reactive seizures i.e. benign febrile convulsions of infancy and childhood.

(ii) Specific primary epileptic conditions (Primary epilepsies) autosomal dominant genetic traits has been identified i.e. childhood and juvenile absence epilepsies, partial sylvian epilepsy.

(iii) Secondary epilepsies i.e Myoclonic epilepsies, phenyl ketonuria.
ACQUIRED FACTORS

These include congenital lesion, head trauma, infection of brain and its coverings, brain tumours, systemic toxic and metabolic disorders, mesial temporal sclerosis, birth trauma.

The diagnostic role of inter-ictal EEG in epilepsies has been questioned by many authors (Goodin, 1984). Diagnosis of epilepsy is mainly clinical and role of EEG is restricted to confirmation of provisional diagnosis, to classify the seizure disorder, localisation of epileptic foci and guiding prognosis (in some cases) (Githhchley, 1978). In a study by Goodin (1964) 60% of epileptic patients in general population had positive epileptiform activity on EEG. While only 4% of non epileptic has positive EEG.

LOCALISATION RELATED EPILEPSIES AND SYNDROMES

1. Benign childhood epilepsy with centro-temporal spikes.

This type occurs in 15-20% of epileptics in the age group of 3-15 year, more commonly in males (60%). Seizure pattern consists of brief spells of simple partial hemifacial motor seizures, frequently during sleep (70%), with somatosensory onset and tendency to tonic, tonic-clonic seizures of facial and neck muscles, manifesting as speech arrest and drooling of saliva, seizure, occurs usually once in 2-12 months. EEG changes consists of blunt high voltage centrotemporal spikes often followed by slow waves usually precipitated by sleep.
It is an autosomal dominant gene with age dependent penetrance. Monotherapy with phenytoin or carbamazepine is successful. Usually good recovery occurs before 15-16 years of age.

2. Childhood epilepsy with occipital paroxysms.

Onset is usually between 1½ years to 8 years, usual symptoms (amaurosis in 65%, Scotoma in 55%, hallucinations in 25%, illusions in 10% cases) predominate, nocturnal partial seizures (usually hemi-convulsive) and post ictal headache (30%) are fairly common.

Paroxysmal high voltage spike wave and/or sharp waves localised in the occipital or posterior temporal region over one or both hemispheres occur rhythmically on EEG.

These finding tend to disappear after 9 years of age. Prognosis is favourable regardless of age, seizures are well controlled in 60% cases. Drugs useful are phenytoin, carbamazepine and primidone.

3. Benign partial epilepsy with frontal foci:

Partial seizures usually begin between 4-8 years of age. The seizure pattern is of adiversive type with head deviation, simple absence seizures may precede or coexist. EEG findings consists of unilateral or bilateral frontal foci. In about 50% cases 3 Hz spike and wave complexes are observed, EEG abnormalities normalize before the age of 13 years.
4. Primary Reading Epilepsy

Symptomatic:

Chronic progressive epilepsy partials continua:

It is seen in young children with unilateral chronic cerebral inflammatory disorders or in adults after severe anoxia and major cerebrovascular accident. The continual partial motor seizures usually involving face reflect multiple rather than single lesion. This form is often unresponsive to medications. Seizures may disappear after about 1-2 years in about one third of cases.

Temporal lobe epilepsies:

This term is not synonymous with complex partial seizures, psychomotor and limbic seizure. The term temporal lobe epilepsies means that a temporal lobe focus in EEG is demonstrable. Symptomatology usually consists of an aura, psychic symptoms, alteration of consciousness, automatisms and commonly secondary spread to manifest as tonic-clonig seizures.

Auras consists of experiential phenomena, which patient perceives as warning of impending seizure episode. Psychic symptoms consist of:

a. dysmnesic symptoms i.e. feeling of familiarity (de ja vu) and unfamiliarity (Jamais vu)

b. cognitive disturbances such as dreamy state, depersonalization and time distortion.
c. Affective symptoms, fear and rage, depression and elation and sometimes sexual excitement (organic epilepsy).

d. Delusions and hallucinations.

Alteration of consciousness unusually reflects involvement of limbic structures and their connections. Autonomic auras such as epigastric distress or vomiting most commonly precede temporal lobe epilepsies which are associated with altered consciousness. Those seizures which are preceded by olfactory aura's are called uncinate seizures. EEG findings consist of unilateral or bilateral spikes.

**Frontal lobe epilepsies.**

Seizures pattern may consist of:

a. Tonic-clonic movements starting from discrete parts such as feet, hands and facial muscles and progressing leading to unilateral or bilateral involvement. When spread occurs in an orderly fashion along the precentral gyrus jacksonian march occurs. These seizures may be followed by post ictal weakness (Todd's paresis).

b. Complex iversive movements in form of, turning of head, eyes and body towards one side and posturing of one or more extremities.

c. Adversive seizures: Turning of head and eyes away from epileptic focus and elevation of contralateral arm. It occurs with involment of supplementary motor cortex.
d. Inappropriate laughter associated with humour (Gelastic epilepsy).

e. Sometimes they may manifest as psychomotor seizures and atypical absence seizures.

EEG usually shows unilateral or bilateral spike wave complexes and sharp waves.

Parietal lobe epilepsies:

They usually manifest as localised paresthesia or numbness from onset of a seizure. Spread occurs readily to frontal cortex leading to sensory motor seizures. Post ictal phenomena such as anaesthesia may follow after simple partial seizures involving parietal lobe.

Occipital lobe epilepsies:

The seizures pattern usually manifest as unformed luminous vision and amaurosis.

GENERALISED EPILEPSIES AND SYNDROMES

1. Benign Neonatal familial convulsions:

They usually occur on 2nd or 3rd day. Seizures pattern consists of clonic or apneic seizures. No specific EEG changes are observed. About 14% of these develop some form of epilepsy.

2. Neonatal convulsions.

They occur on 5th day of birth. Seizure pattern is of clonic or apneic type. Alternate sharp theta waves are observed on EEG.
3. Benign myoclonic epilepsy in infancy:

Age group involved is 1-2 years. Seizures pattern consists of brief spells of generalised myoclonus and brief bursts of spike wave complexes are observed on EEG.

4. Childhood absence epilepsy (Pyknolepsy)

It affects children 6-7 years of age, manifests as many absence seizures during a day. EEG findings consist of bilateral synchronous spikes and waves. Some of these children may develop Generalised tonic clonic seizures (GTCS) later on.

5. Juvenile Absence: (Petit mal).

It manifests as absence with retropulsive movements, seizure frequency is less than childhood absence. EEG findings show 3 Hz spike-wave bursts. This type of epilepsy has excellent response to therapy.

6. Juvenile myoclonic epilepsy: (Impulsive petit mal)

It manifests as bilateral single or repetitive arrhythmic irregular myoclonic jerks in arms and shoulders. It may progress to generalised Tonic clonic Seizures associated with infrequent absences. EEG findings consist of precipitation of episode by photic stimulation, rapid generalised 4 to 6 Hz spike waves/polyspike-wave. It affects about 7% of adolescent epileptic patients. Sodium valproate is the drug of choice.

7. Epilepsy with Generalised Tonic-Clonic Seizures on awakening:

It usually affects young adults. Familial occurrence is
frequent. Seizure type consist of Generalised Tonic Clonic Seizures shortly after awakening (90%) or in the evening (10%). EEG findings consist generalised high voltage sharp waves following by slow waves.

8. Epilepsies with seizures precipitated by specific modes of activation:

Classic example of this group are reflex epilepsies which are generalised i.e. Hotwater epilepsy, eating epilepsy, tactile epilepsy, paroxysmal dystonic seizures.

Cryptogenic

1. West Syndrome:

It affects infants usually in 4-7 months age group. Seizure type consist of salam attacks. EEG shows burst suppression with distorted background activity. Prognosis is poor, even after treatment with steroids.

2. Lennox Gastaut Syndrome:

Children of 1-8 year of age group show this picture consisting of more than one variety of generalised seizures, presence of mental retardation and generalised sharp and slow wave complexes of less than 3 Hz and some times sleep bursts of fast rhythm.

Refractoriness to common anti-epileptic drugs is the rule.
3. Epilepsy with myoclonic astatic seizures:

Age of onset is usually 7-12 years. Frequently male children are affected. Seizure type consist of myoclonic akinetic absence with clonic and atonic components. Frequently these patients may land into status epilepticus of generalised tonic-clonic type. Regular fast spike wave and polyspike wave abnormalities are noticed on EEG.

Epilepsies and Syndromes undetermined whether focal or generalised

1. Epilepsy with continuous spike wave activity during sleep.

Seizure type consist of Generalised tonic clonic seizures during sleep and atypical seizures. Observed EEG findings consist of continuous diffuse spike-wave during sleep.

2. Acquired epileptic childhood aphasia (Landau Kleffner syndrome) Seizure type consist of verbal auditory agnosia with secondarily generalised partial motor seizures. EEG findings consist of multi focal spikes and spike wave complexes. Course is benign usually with complete remission before the age of 15 years.

EEG in the Diagnosis of epilepsy

Definitive diagnosis of epilepsy requires demonstration of ictal EEG changes simultaneously with occurrence of seizure, which is rarely possible except in patients with absence seizures, juvenile myoclonic seizures and partial seizures with minor motor seizure.
Due to paroxysmal nature of the disease a normal EEG does not exclude epilepsy. The probability of positive EEG findings is related to several factors.

a. Duration of recording.
b. Wake-sleep study.
c. Activation procedures used.
d. Times of EEG recording.

In a single routine EEG, positive findings are usually observed in around 60% cases with sleep deprivation, but if 5 EEG recordings have been taken epileptiform changes may be observed in 90% cases.

e. Use of additional electrodes.
f. Anticonvulsant therapy.

**EEG abnormalities in epilepsies**

They can be grouped in three categories:

1. Non-specific abnormalities
2. Specific abnormalities
3. Background abnormalities

1. **Non-specific abnormalities**
   a) 3 Hz delta paroxysms
      i. Frontal intermittent rhythmic Delta activity (FIRDA).
      ii. Occipital intermittent rhythmic Delta activity (OIRDA)
   b) Runs of bilaterally synchronous delta.
   c) Bursts of bilaterally synchronous theta.
d) 8-12 Hz activity of higher amplitude than the background alpha
activity.

2. **Specific epileptiform abnormalities.**
   a. Spikes
   b. polyspikes
   c. High voltage sharp waves
   d. spike-wave complexes
   e. polyspike-wave complexes
   f. sharp wave discharges.

3. **Background abnormalities.**
   a. mild degree of diffuse slowing while on anticonvulsant
   therapy.
   b. Diffuse marked slowing—may occur during post ictal period for
   few hours to 1-2 days.
   c. background slowing other than above usually correlates with
   mental subnormalities.

Following newer investigation have facilitated the diagnosis of
epilepsies.

A) **NEUROPHYSIOLOGICAL INVESTIGATIONS:**
1. Routine EEG
2. Ambulatory cassette recording
3. EEG telemetry
   a. Cable telemetry.
   b. Radio telemetry.
4. EEG recording with closed circuit television by split screen
   technique.
5. Computrisied EEG neuromapping.
B) **NEURORADIOLOGICAL PROCEDURES**

1. Plain radiography.
2. CT scanning.
3. MRI imaging.
4. Isotope encephalography
5. PET scanning.
6. SPECT scanning.

EEG abnormalities reported in partial epilepsies are sequential spikes and sharp waves, Sinusoidal waves Rhythmic waves and attenuation of spontaneous activity. In a study designed to assess EEG morphology of partial epileptic seizures, Warren T Blume et al (1984) studied 66 consecutive patients with electrographic partial seizures. Different EEG phenomenon noted were:

1. Attenuation, sinusoidal waves.
2. Spikes, spike-waves, polyspike-waves, sharp waves, saw toothed waves.

The second group was referred as repetitive epileptiform potentials (REPs).

They noted: Total patients = 66

Sinusoidal waves in 20 patients (30%)

REPs in 17 patients (25%)

Both in 29 patients (45%)
ONSET AND MORPHOLOGY OF EEG CHANGES

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<th>Evolution</th>
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<tr>
<td>Sinusoidal only 31</td>
<td>Reps appear (11)</td>
</tr>
<tr>
<td>Reps only -25</td>
<td>Reps only 17</td>
</tr>
<tr>
<td>Both (sinusoidal &amp; Reps) -10</td>
<td>Sinusoidal appear-8</td>
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Spikes and sharp waves were the most common repetitive epileptic potentials encountered.

Attenuation of spontaneous activity with onset of seizure was noted in 11% of cases. Gibbs (1964) reported 80% incidence of temporal foci in clinical psychomotor epilepsy.

Many workers have tried to develop methods of deriving components of local activity in certain areas. Bo Hjorth (1976) made an important contribution by formulating source sensing derivations connected between EEG amplifiers and recording channels, derivations of this type constitute better description of local activity than do the conventional, common reference and bipolar derivations.

The model implied that the observed surface pattern is a secondary effect of primary field components perpendicular to surface.

Smith et al (1985) have demonstrated the reliability of an invasive method for successfully localising the local components of scalp EEG. Dipole localisation method (DLM) is a computer assisted, mathematical method based on electrical field theory.