Seizures & Epilepsy in Children

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Introduction

Epilepsy “common” neurological problem.

50 million people have epilepsy globally; 20% of them are children.

80% of 3.5 million new cases of epilepsy from developing countries*.

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Why General Pediatrician should be aware of Epilepsy?

60% of later age epilepsy has its “roots” in childhoood*.

60 to 70% of childhood epilepsy are manageable in community.

50% of children with epilepsy have co-morbidities**.

Why General Pediatrician should be aware of Epilepsy?

1/3rd children in cities & 85% children in rural areas with epilepsy either don't attend or are expelled from schools.

Treatment gap is more than 80%.

Universally accepted high mis-diagnosis.

Definitions

Seizure

A transient occurrence of signs and/or symptoms resulting from abnormal excessive or synchronous neuronal activity in brain

Epilepsy

Two or more unprovoked seizures more than 24 hrs apart – epileptic tendency

One Seizure with abnormal Neurological Examination Or EEG Or MRI

Situation related

Seizures provoked by illness / metabolic disturbance / toxic event, does not denote epileptic tendency

Prodrome

Mood or Behavior change that precedes a seizure

- A seizure is a temporary involuntary disturbance of brain function that may be manifested as impaired consciousness, abnormal motor activity, sensory disturbances or autonomic dysfunction.
- All seizures are not epilepsy
- 2 large categories
  - Focal/ partial
  - Generalized
- Febrile seizures is a special category

- Acute symptomatic seizures- acute problem affecting bra excitability such as electrolyte imbalance

- Unprovoked seizures

- Remote symptomatic seizures- distant brain injury

- Reflex seizures are usually precipitated by a sensory stimulus such as flashing lights

EPILEPSY

- Disorder of brain characterized by an enduring predisposition to generate seizures and by the neurobiologic, cognitive, psychological
and social consequences of this condition
- 1 unprovoked epileptic seizure+ 2nd such seizure

OR

enough EEG or clinical predisposition

- More than 2 unprovoked seizures in >24 hrs

- Seizure disorder- any one of the several disorders including
  - Epilepsy
  - Febrile seizure
  - single seizures
- secondary to metabolic, infectious , or other etiologies

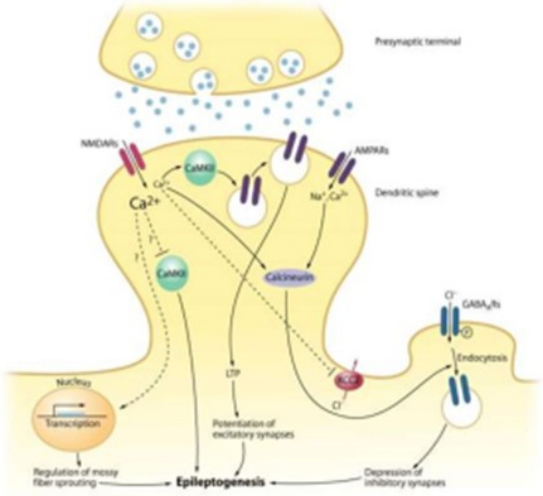
- Epileptic syndrome- manifests one or more seizure types specific age of onset and specific prognosis
- Epileptic encephalopathy- severe EEG abnormality to result in cognitive impairment
- Idiopathic epilepsy- genetic
- Symptomatic epilepsy- by an underlying brain disorder
- Cryptogenic epilepsy(presumed symptomatic epilepsy)- brain disorder which is not underlying known

MECHANISMS OF SEIZURES

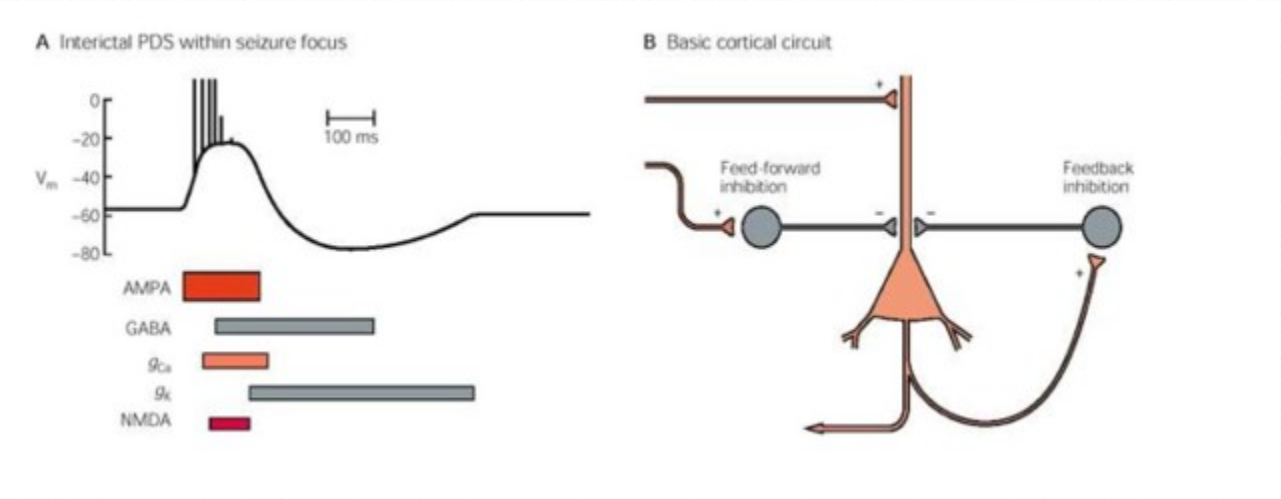
- Underlying etiology- brain tumors, strokes, scarring or mutations
- Mutations can involve voltage gated channels,ligand gated channels
and Ɣ-amino butyric acid A receptors [GABA A]
- Kindling- animal model for temporal lobe epilepsy

- repeated stimulation- activation of metabotropic and ionotropic glutamate receptors as well as TrkB receptor , BDNF and neurotrophin 4

- Increase in intraneuronal Ca
- Activates CaMK2 and calcineurin
- Ca dependant epileptogenic gene expression
- Sprouting of mossy fibres
- Increased excitability and epilepsy

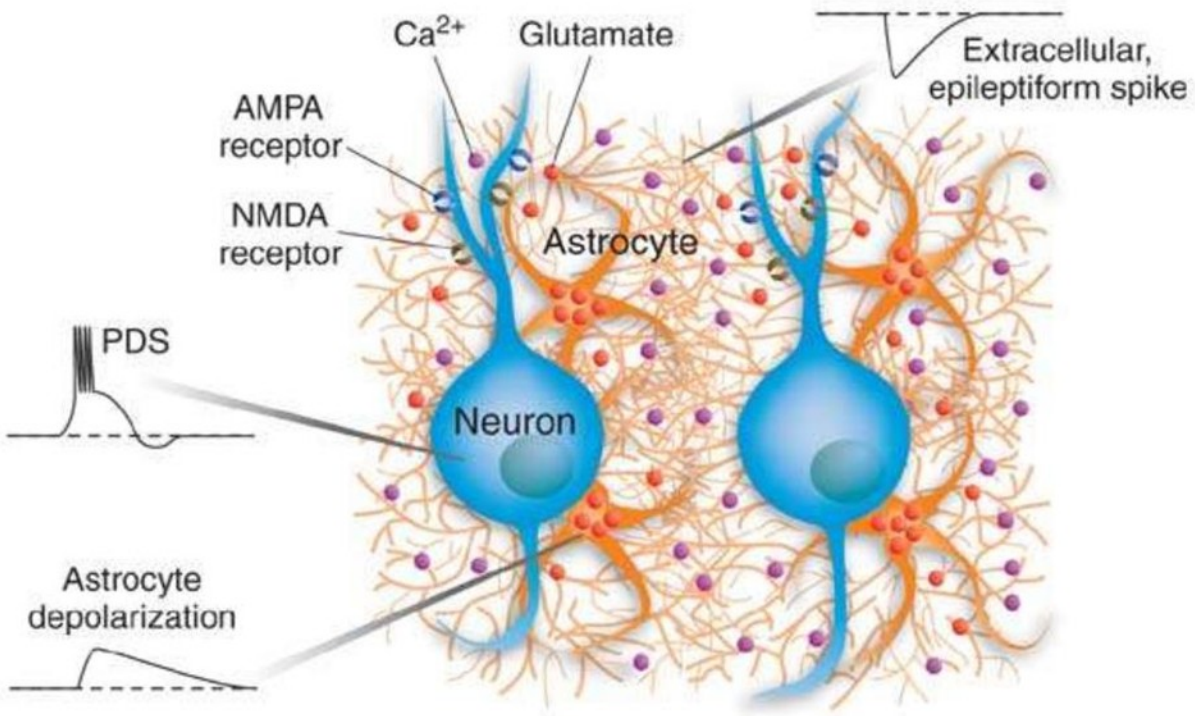


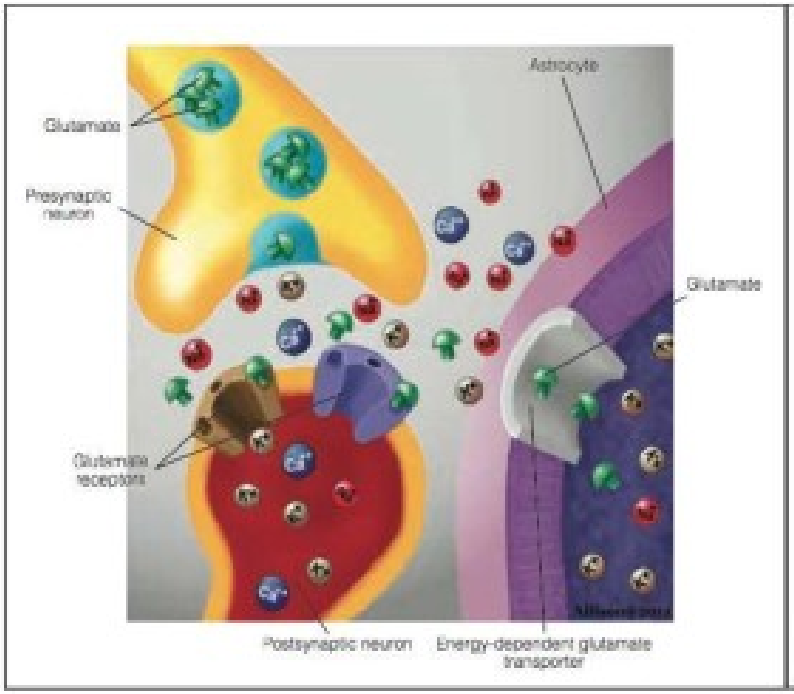
• Epileptic state of increased excitability present in all patients:
• In a seizure focus each neuron has stereotypic synchronized response
  called paroxysmal depolarization shift

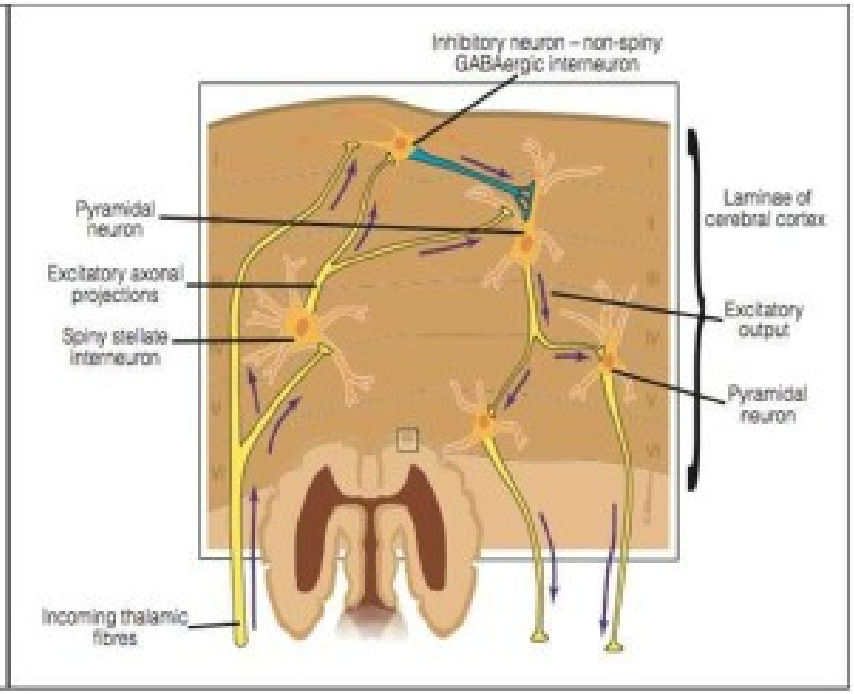


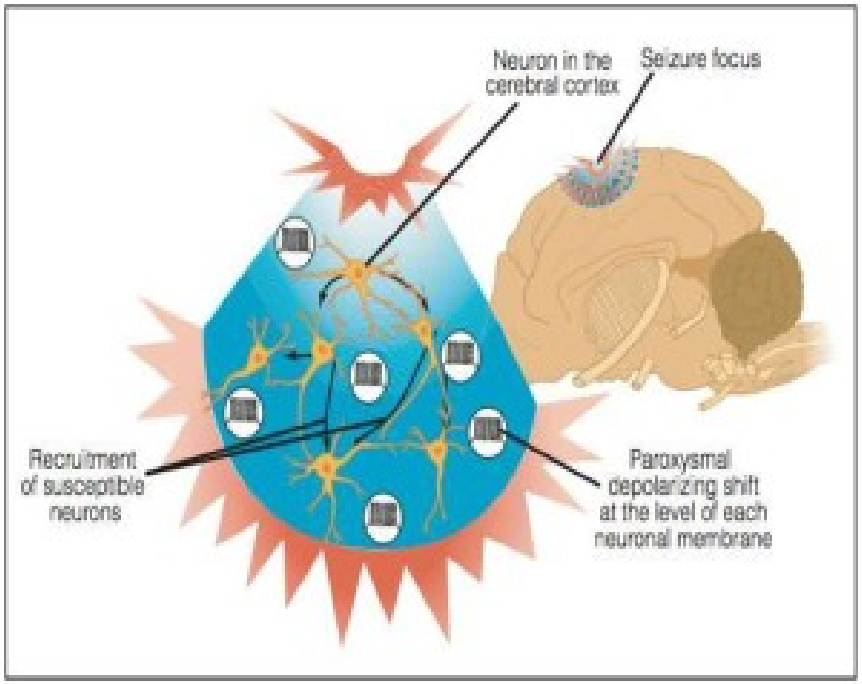
- Sudden depolarization(glutamate and Ca channel activation)

- Afterhyperpolarisation(K channels and GABA receptors)




Glutamate
Presynaptic neuron
Astrocyte
Glutamate
Glutamate receptors
Postsynaptic neuron
Energy-dependent glutamate transporter


Inhibitory neuron – non-spiny GABAergic interneuron
Pyramidal neuron
Excitatory axonal projections
Spiny stellate interneuron
Laminae of cerebral cortex
Excitatory output
Pyramidal neuron
Incoming thalamic fibres


Neuron in the cerebral cortex
Seizure focus
Recruitment of susceptible neurons
Paroxysmal depolarizing shift at the level of each neuronal membrane

• T-type Ca channels on thalamic relay neurons are activated resulting
in spike wave pattern

• 4th process

• Seizure related neuronal injury

• In MRI after prolonged febrile and afebrile status epilepticus
• Acute swelling in hippocampus and long term hippocampal atrophy with sclerosis

• Apoptosis and necrosis of
neurons

- Things that can cause membrane instability:
  - Deficiency in Oxygen
  - Deficiency in Glucose
  - Decrease in Calcium

Incidence

Seizures affect 4 to 7 % of children.

Up to 1% of all ED visits are peds sz

- Peds febrile: 1 in 125 visits (0.8%)
- Peds afebrile: 1 in 500 visits (0.2%)

Epileptic seizures affect 1-2% of the population & 4% of children*.

Developing countries have higher prevalence.

**Hauser WA. Epidemiology of epilepsy in children. Neurosurg Clin N Am. 1995;6:419–29*

Incidence

40% develop epilepsy before the age of 16 yrs.

Focal epilepsies commoner than generalized.

In about 20% classification changes on follow up*.

90% of childhood epilepsies are classifiable into syndromes*

*A Practical Guide to Childhood Epilepsies. Vol. 1. Oxford: Medicine; 2006. pp. 17–20.

- Mean age 3.2 yrs, median age 1 year
- 61% by age 3
- Etiology age dependent
  - 25% is febrile SE
  - Before age 1, 75% due to acute insult
  - Epilepsy, fever, CNS infection common

Etiology of seizures

Causes for Epileptic seizures

- Idiopathic (70-80%) – cause unknown but presumed genetic
- Secondary
  - Cerebral malformations
  - Cerebral vascular occlusion
  - Cerebral damage (ex; congenital infections, hypoxic-ischaemic encephalopathy

- Cerebral tumour
- Neurodegenerative disorders
- Neurocutaneous syndromes
  - Neurofibromatosis
    - Tuberous sclerosis

Causes for Non-epileptic seizures

- Febrile seizures
- Metabolic
  - Hypoglycaemia
  - Hypocalcaemia
  - Hypomagnesaemia
  - Hypo/hyper natraemia

- Head trauma
- Meningitis/Encephalitis
- Poisons/Toxins

CAUSES

Table 1. Various Causes Of Seizures

Developmental Or Congenital	Withdrawal Syndromes
• Genetic (inborn errors of metabolism) • Congenital anomalies, birth injury, hypoxic encephalopathy, stroke, brain hemorrhage, vascular injury, brain contusion • Degenerative cerebral diseases • Neurocutaneous syndromes	• Alcohol • Anticonvulsants
Mechanical or Traumatic	**Drugs, Medications**
• Ventriculoperitoneal (VP) shunt malfunction • Head trauma (accidental, child abuse)	• Cocaine, amphetamines, alcohol • Phenothiazines • Sympathomimetics • Lithium • Isoniazid (INH) • Lindane • Acetylsalicylic acids (ASA), salicylates • Theophylline
Metabolic	**Infectious**
• Hypocalcemia • Hypomagnesemia • Hyponatremia • Hypercarbia • Hypoxia • Pyridoxine deficiency • Hypoglycemia • Inborn errors of metabolism	• Meningitis (bacterial, viral, fungal) • Encephalitis • Neurocysticercosis • Brain abscess • Shigella gastroenteritis
Poisonings	**Idiopathic**
- Organophosphates - Lead	- Epilepsy

- Most common- febrile seizures
- Infections
- Metabolic disorders
- Drugs

Risk factors

- A family history of epilepsy
- A previous history of epilepsy; whether the child is
- Taking antiepileptic drugs (AEDs)
- Presence of conditions associated with electrolyte (magnesiu

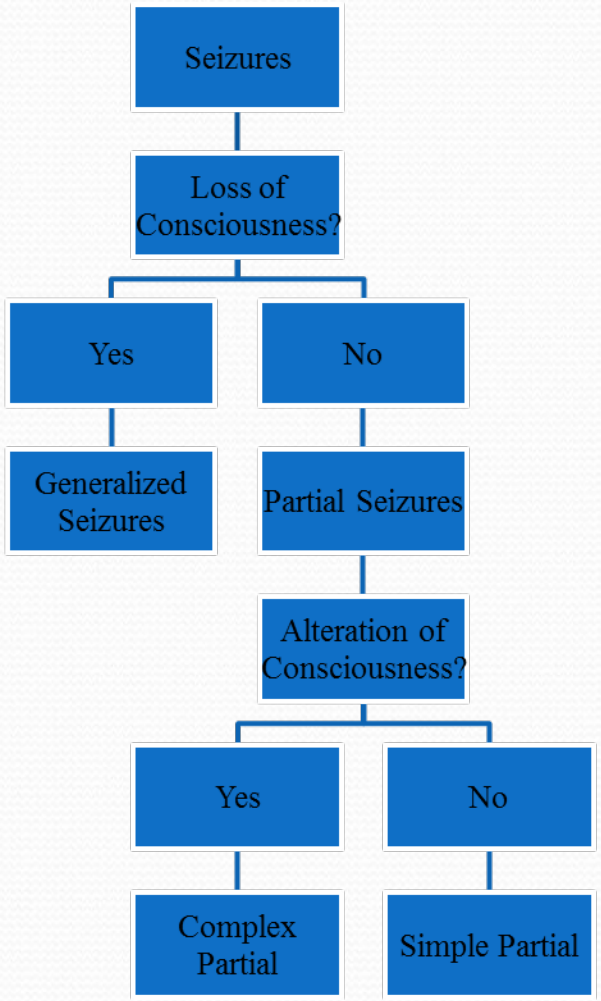
phosphate, or calcium) disturbances, such as diarrhea or rick

- Presence of acidosis associated with hypoxia
- Provoking factors, such as sleep deprivation, fevers, illness o infections, fatigue, decreased physical health, alcohol ingesti emotional stress, flashing lights, menstrual cycle, missed mea

Classification of Seizures

- Traditionally divided into “ grand mal” and “petit mal” seizures
- ILAE classification of epileptic seizures in 1981 based on clinical observation and EEG findings
- Seizures were divided into partial and generalized seizures based on loss of consciousness
- Partial seizures were divided into simple partial and complex partial based on alteration of consciousness

Classification of Seizures



ILAE Classification of Seizures

Partial Seizures	Generalized Seizures
Complex Partial Seizures (CPS) – With automatism – Without automatism Simple Partial Seizures (SPS) – Motor o With march o Without march o Versive o Postural o Phonatory – Sensory o Somatosensory o Olfactory o Visual o Auditory o Gustatory o Vertiginous – Autonomic – Psychiatric o Dysphasic o Déjà vu or jamais vu o Cognitive o Affective o Illusions o Structured hallucinations Secondary Generalized Tonic-Clonic	Tonic-Clonic (primary tonic-clonic) Absence Myoclonic Clonic Tonic Atonic Atypical Absence Infantile Spasm

Classification of Epilepsy

- ILAE classification of epilepsy and epileptic seizures in 1989
- Depends on 2 distinctions;
  - Location of pathology (Localized or generalized)
  - Know or presumed etiology
    - Idiopathic
    - Symptomatic
    - Cryptogenic

ILAE Classification of Epilepsy

	Localization-Related (named by location)	*Generalized (named by disease)*
Idiopathic	Benign Rolandic epilepsy (Benign childhood epilepsy with centro-temporal spikes) Benign occipital epilepsy of childhood Autosomal dominant nocturnal frontal lobe epilepsy Primary Reading Epilepsy	Benign Neonatal Convulsions (+/- familial) Benign myoclonic epilepsy in infancy Childhood absence epilepsy Juvenile absence epilepsy Juvenile myoclonic epilepsy Epilepsy with GTCs on awakening Some reflex epilepsies
Symptomatic	Temporal lobe Frontal lobe Parietal lobe Occipital lobe (Rasmussen's encephalitis) (Most Reflex epilepsies)	Early myoclonic encephalopathy Early infantile epileptic encephalopathy with suppression- burst (Ohtahara's syndrome) Cortical abnormalities -malformations -dysplasias Metabolic abnormalities - amino acidurias - organic acidurias - mitochondrial diseases - progressive encephalopathies of childhood West's Syndrome Lennox-Gastaut Syndrome
Cryptogenic	(Any occurrence of partial seizures without obvious pathology.)	Epilepsy with myoclonic-astatic seizures Epilepsy with myoclonic absence

Types of seizures

Focal seizures :

i. Originate from localized area of one hemisphere*.

ii. Common causes include

a. Idiopathic

b. Cryptogenic

c. Structural

iii. In young infants & children focal seizures are very subtle.

* Epilepsia. 1999;40:439–44.

Focal seizures

ILAE 1989 classification*

- Simple partial seizure (SPS) when consciousness is preserved.
- Complex partial seizure (CPS) when consciousness is impaired or lost.
- Partial seizure evolving secondary to generalized.

**Commission of classification and terminology of the International League Against Epilepsy. Proposal for revised classification of epilepsies & epileptic syndromes. Epilepsia. 1989;30:389–99*

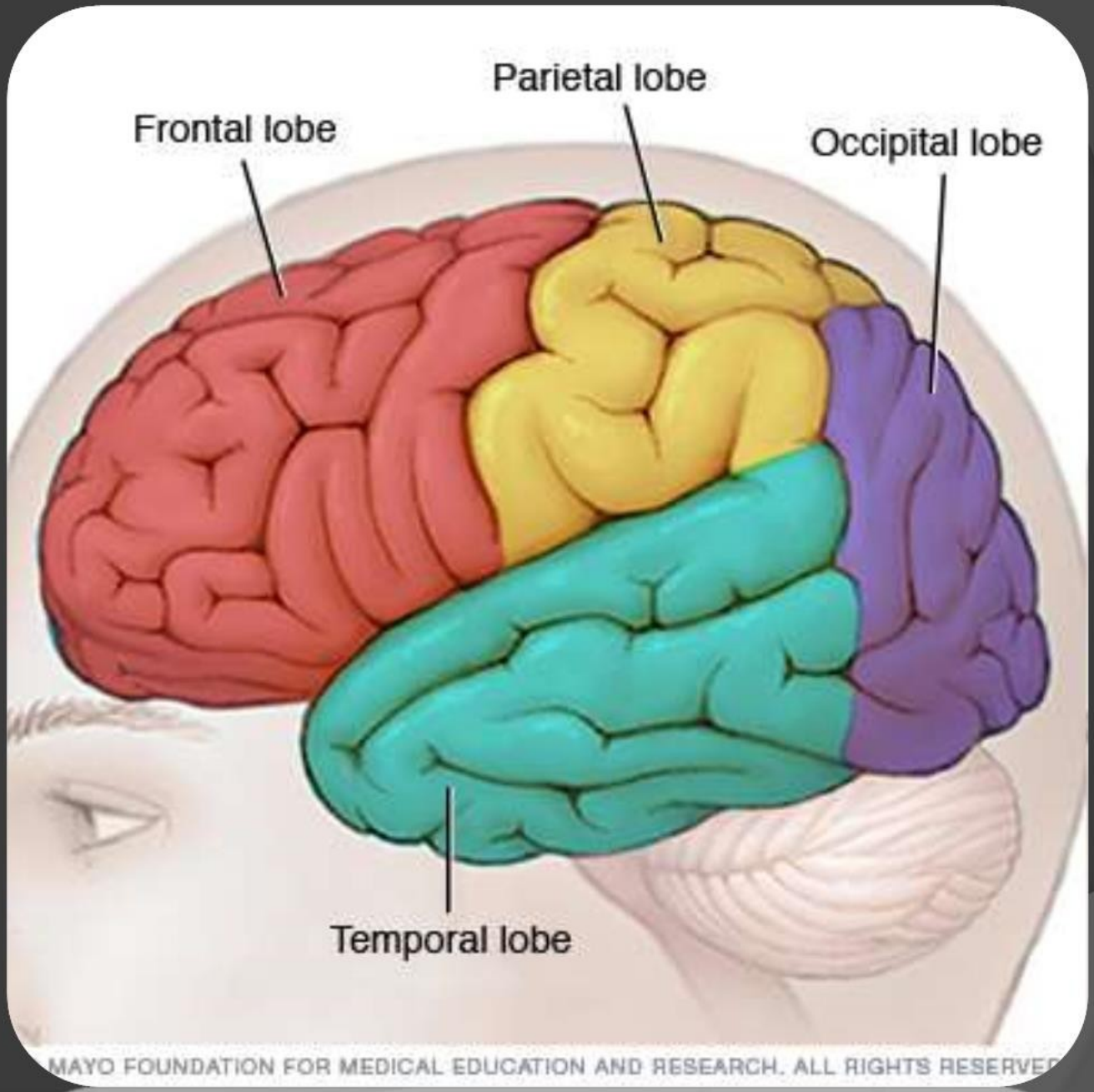
This classification is still used as most physician are familiar with it, yet newer guidelines don't include these terms.

Focal seizures

Characterized by aura, behavioral arrest, focal motor activity & versive eye movements.

SPS can have motor, sensory, autonomic or psychiatric symptoms without loss of sensorium.

CPS is characterized by aura, altered sensorium, motor activity & automatism.


Parietal lobe
Frontal lobe
Occipital lobe
Temporal lobe
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Frontal	Motor phenomena
Temporal	Auditory or sensory (smell or taste) phenomena
Occipital	Positive or negative visual phenomena
Parietal	Contra lateral altered sensation

Partial Seizures

- Simple seizures (no LOC)
  - Focal motor (Jacksonian)
  - Sensory or somatosensory
  - Autonomic
  - Psychic
- Complex (impaired consciousness)
  - Involves some cognitive, affective sx
  - Temporal lobe, psychomotor seizures

Simple Partial Seizure

- Usually lasts 5-10 seconds; most less than a minute
- Symptoms dependent on cortical area involved
- No loss of consciousness
- No postictal state
- Difficult to differentiate between psychiatric disorders (key is paroxysmal nature and duration of seizure)
- EEG - normal or focal spikes

Complex Partial Seizure

- Most common type of seizures
- Variable duration, but typically less than 3 minutes
- Appears awake, but not responsive - often stare or have automatisms
- If restrained, may become hostile or aggressive
- Postictal period - somnolence, confusion, and headache up for up to several hours
- No memory of what took place during seizure
- EEG - focal activity spreading to involve one or both hemispheres

Focal seizures

Evaluation

– Age of onset

– History

– Characteristic

EEG.

These children have normal neuro-development

& don’t require extensive workup.

Focal seizures evaluation

No neuro-imaging required in benign childhood epilepsy with centro-temporal spikes (BCECTS)/Rolandic Epilepsy.

In other children EEG & neuro-imaging is required.

Request a prolonged or sleep deprived EEG or video EEG to increase the yield.

EEG shows focal slowing or focal spikes & wave from involved lobe.

Generalized seizures

Synchronous involvement of both cerebral hemispheres.

Important to differentiate them from focal seizure with very fast secondary generalization

Generalized seizures

ILAE 1989 clissification* :

- Typical & atypical absence
- Myoclonic
- Tonic
- Clonic
- Tonic - clonic

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Generalized tonic clonic seizures

Tonic phase is characterized by sudden loss of consciousness, generalized body stiffness, open eyes & shrilled cry lasts for 10 to 30 sec followed by clonic phase for 1 to 2 min.

Clonic phase has alternative rhythmic clonic contractions & relaxation of the body, cyanosis, salivation, incontinenence & is always followed by post ictal phase of variable duration lasting for minutes to few hours.

In post ictal phase initially there is stupor followed by agitation confusion & child may have headache & vomiting.

Generalized Tonic-Clonic Seizure

- Usually lasts 1-2 minutes
- Abrupt loss of consciousness, often preceded by scream
- All muscles become stiff (tonic) followed by twitching/jerking movements (clonic)
- Expect cyanosis, mouth injuries, or other bodily injuries
- Can be preceded by any partial seizure
- Postictal period - usually deep sleep with hyperventilation then gradual wakening with complaint of headache
- EEG - series of generalized, high-amplitude spikes

Generalized tonic clonic seizures

GTCS can be

a. Primarily generalized

b. Focal with secondary generalization

c. Part of other epileptic syndromes.

• Etiology is idiopathic (mainly genetic) while child with secondary generalization may have underlying focal lesion.

Generalized tonic clonic seizures

EEG may be normal or show generalized spike & wave followed by slowing in post ictal phase,

Focal findings indicate underlying focal structural lesion.

Neuro-imaging must be done in all children with first unexplained GTCS..

Generalized tonic clonic seizures

Evaluation after first seizure:

i. Careful history & examination

ii. Rule out

a. neuro-infections.

b. Febrile seizures.

c. Focal seizures by history of aura

d. Focal onset or post ictal Todd's paresis

e. Non epileptic events

Precipitating factors are sleep deprivation, photosensitivity.

GENERALISED SEIZURES AND RELATED SYNDROMES

- **ABSENCE SEIZURES**
- Start at 5-8 yrs of age
- No aura, last for few seconds, flutter or upward rolling of eyes
- No post ictal period
- Hyperventilation can precipitate
- Atypical absence- associated myoclonic component

tone changes are difficult to treat

precipitated by drowsiness

1-2 hz spike and slow wave discharges

• Juvenile absence- at later age

- 4-6 hz spike and slow wave, polyspike and slow wave discharges

a/w juvenile myoclonic epilepsy

Absence Seizure

- Usually lasts between 5-10 seconds; but frequently in clusters
- Considered a seizure disorder of childhood
- Absence before age 5 associated with mental retardation and tendency for future seizures
- Sudden staring with impaired consciousness with eye blinking and lip smacking for longer seizures
- EEG - characteristic generalized, 3 per second, spike and wave

Seizure type	Older AED	Newer AED	Drugs that worsen the seizure
Generalized	Sodium Valporate Phenytoin Carbamazipine	Clobazam Limotrigene Topiramate	
Focal	Carbamazipine Phenytoin	Oxcarbazepine Limotrigene Clobazam Topiramate Lacosamide Vigabatrin	
Absence	Sodium Valporate Ethosuximide	Limotrigene Levitracetam	Carbamazepine Oxcarbamazepine Phenytoin Vigabatrin
Myoclonous	Sodium Valporate Benzodizepines	Limotrigene Topiramate Levitracetam Zonisamide	Carbamazepine Limotrigene
Infantile spasms	ACTH Sodium Valporate Benzodizapine	Vigabatrin Clobazam Topiramate Zonisamide Levitracetam	Phenytoin Carbamazepine
Mixed	Sodium Valporate	Limotrigene Clobazam Topiramate	

Other Generalized Sz Types

- Neonatal seizures
- Benign childhood epilepsy (Rolandic)
- Infantile spasms (West syndrome)
- Lennox-Gastaut syndrome
- Atonic seizures
- Febrile seizures

Neonatal Seizure

- Occur in first 28 days of life
- Most occur shortly after birth
- Subtle sz: lip smack, eye mvmt, apnea
- **Causes**: Perinatal asphyxia, metabolic abn, Hypoglycemia, hypocalcemia,CNS infection, hemorrhage, lesion

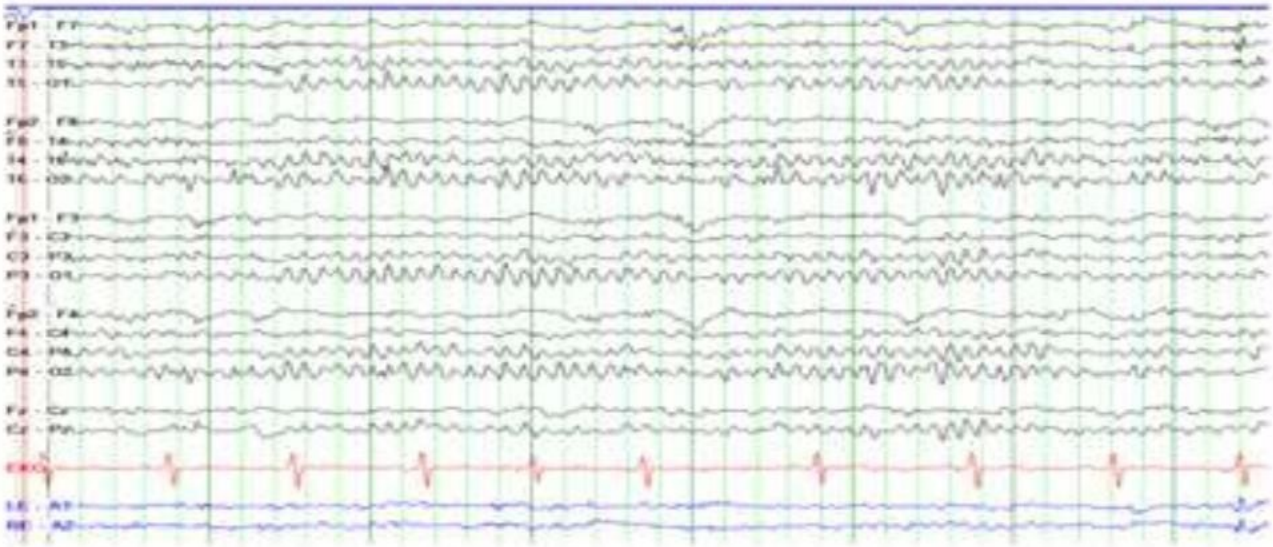
Benign Childhood Epilepsy

- Rolandic epilepsy
- Onset between 3 and 13 years of age
- Often occurs upon awakening
- Facial mvmts, grimacing, vocalizations
- EEG diagnosis

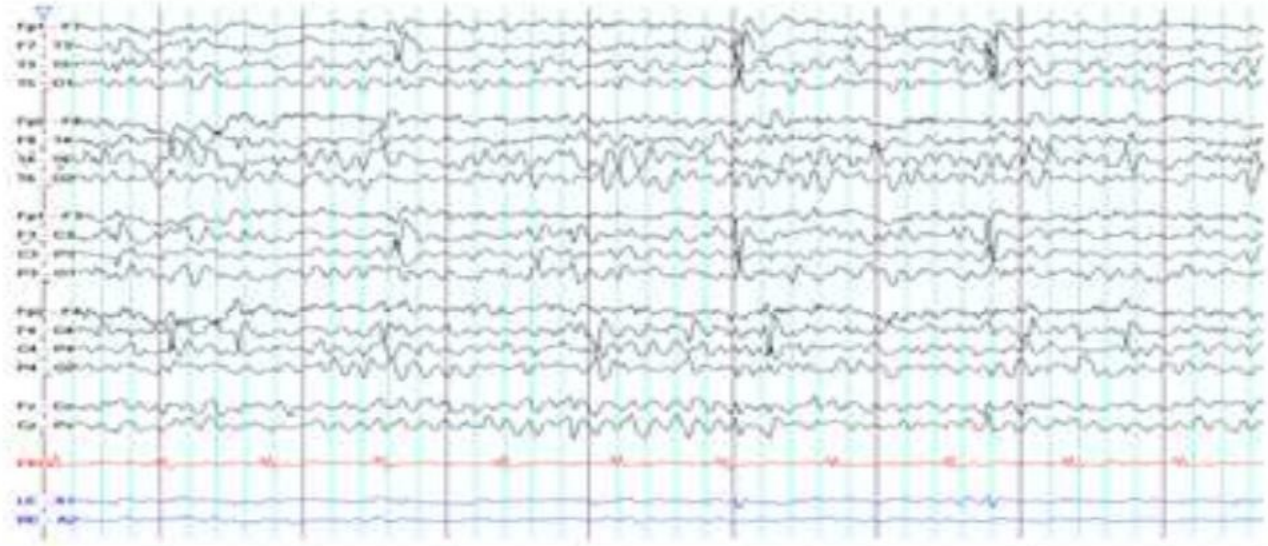
Benign epilepsy with centrotemporal spikes (BECTS)

- Age of onset 4-10 y
- Tonic clonic seizures in sleep
- Abnormal feelings in tongue & face
- Focal sharp waves in EEG
- Benign so important to recognize

Normal EEG Awake



Benign Rolandic Epilepsy



Infantile Spasms

- West syndrome
- Occurs up to one year
- May be symptomatic or idiopathic
- Sudden tonic movements of the head, trunk, extremities
- Must do full work-up, incl metabolic
- Caution, AED hepatotoxicity a risk

Atonic Seizures

- Astatic or akinetic seizures
- Sudden loss of motor tone
- Child falls to the floor
- May have myoclonic jerks
- No clear generalized seizure
- No etiology of apparent syncopal episode

Febrile Seizures

- Age: 6 months to 5 years
- Related to rapid rise in temperature
- Brief, self-limited generalized sz
- Complex: Focal, > 10-15 min, flurry
- 25% recurrence, esp if in child < 1 yr old
- Risk of epilepsy not significantly greater

Juvenile Myoclonic Epilepsy

- aka janz syndrome
- Common in teens, young adults
- Etiology of generalized TC seizures
- History of staring spells
- History of AM clumsiness, myoclonus
- Sleep deprivation precipitant
- Phenytoin: worse myoclonus, absence sz

- **BENIGN GENERALISED EPILEPSIES**
- Petit mal epilepsy- starts in midchildhood
- Benign myoclonic epilepsy of infancy- onset of myoclonic and other during 1st yr with 3 hz spike and slow wave
- Febrile seizure plus syndrome- febrile seizures and multiple types of generalized seizures in multiple family members

- SEVERE GENERALISED EPILEPSIES
- Intractable seizures and developmental delay
- Early myoclonic infantile encephalopathy(EMIE)- starts during 1st 2 months, severe myoclonic jerks and burst suppression pattern
- Early epileptic infantile encephalopathy(EIEE, Ohtahara syndrome)- tonic seizures, brain malformations or syntaxin binding protein1 mutations)

- Severe myoclonic epilepsy of infancy(dravet syndrome)- focal febrile status epilepticus--- to myoclonic and others
- West syndrome- starts between ages 2 and 12 months

triad of infantile spasms, developmental regression, hypsarrythmia

- Lennox-gastaut syndrome- 2 and 10 yrs

  - triad of developmental delay, multiple seizure types, 1-2 hz spike and slow waves, polyspike bursts in sleep

  Myoclonic astatic epilepsy- milder

Progressive myoclonic epilepsies- progressive dementia, worsening myoclonic and other seizures

- Type 1 or Unvericht Lundborg disease- slowly progressive, starts in adolescence
- Type 2 or Lafora body disease- early childhood onset, quickly progressive, fatal

  -photosensitivity, manifests PA S positive lafora inclusions in muscle or skin biopsy

- Myoclonic encephalopathy in non progressive disorders- epileptic encephalopathy in angleman syndrome
- Landau-Kleffner syndrome- m.c in boys

-mean onset 5 ½ yrs

- loss of language skills
- aphasia, auditory agnosia, normal hearing
- high amplitude spike and wave(bitemporal)
- CT and MRI- normal results
- PET- u/l or b/l hypo/hypermetabolism

- Valproic acid is DOC
- leviteracetam/ or nocturnal diazepam therapy(0.2-0.5mg/kg PO)
- If seizures persist- prednisone- 2mg/kg /day for 1 month

tapered to 1mg/kg/day- 1month

- Speech therapy
- subpial transection
- methylphenidate

Evaluation of Patient

History :

i. Seizures –

. Age of onset

. Generalized/ focal; single/ multiple;

. Aura

. Sequence

. Loss of sensorium

. Injury due to fall

. Post-ictal deficit

. Recall of the event

Clinical Evaluation of Seizures

HISTORY is the most important part of the clinical evaluation. Pointed questions are often needed.

Obtain as accurate of a description from patient and witness(es).

Clinical Evaluation of Seizures

Before the seizure…

- Was there an aura?
- Was there an identifiable trigger?
- If there is a history of seizure, what are known precipitants or triggers.

Clinical Evaluation of Seizures

During the seizure…

- Was there signs of impaired consciousness?
- What was the patient actually doing?
- Was there loss of urine or stool?
- How long did the episode last?
- If h/o seizures, was this a typical/atypical episode?

Clinical Evaluation of Seizures

After the seizure…

- For the observer, was the patient postictal? If no observer, did patient know where he/she was, what had happened immediately after episode?
- If postictal, how long was it?
- Did the patient have any complaints when s/he became more awake?

Clinical Evaluation of Seizures

Other history to obtain besides event history:

- Medical history: febrile seizures, head injury, CVA, malignancy, infectious diseases ,drug use or abuse
- Family history: febrile seizures, epilepsy in close relative, h/o neurological disorders
- Social history: travel, occupation, substance abuse

- When the environment is more calm, do a complete history and physical exam
- Spend time on a thorough neurological exam
- Correct any suspected underlying causes

Evaluation of Patient

ii. History of fever, diarrhea, trauma, perinatal insult.

iii. Development – normal/ delay/ regression.

iv. Birth history

v. Family history of epilepsy, febrile seizures.

Evaluation of Patient

Examination :

- Clinical & neurological examination
- Head circumference
- Dysmorphism
- Cutaneous marker- hypo/ hyper pigmented lesions, facial nevus
- Focal deficit
- Fundus examination

Laboratory Data

- UECs, Ca, Mg, CBC with differential, toxicology screens, RBS
- Drug levels if patient is on an anticonvulsant.

Consider…

- Lumbar pucture
- Holter monitoring and/or other cardiac evaluation
- Neurology consult

Investigations

EEG :

– Recommended in all patients with paroxysmal event.

– Helps to distinguish seizure from non- seizure, classification of seizure type & syndrome, deciding treatment & focus localization.

– Video EEG needed for differentiating true seizures from non- seizure paroxysmal disorders & pre-surgical evaluation.

Investigations

Time to do EEG :

- Ideally should be done 3-4 days after seizure to avoid post ictal slowing.
- Sleep deprived EEG increases the yield.
- Photic stimulation & hyper- ventilation helps

No need to stop AED before EEG.

Investigations

Radiology :

– MRI better than CT- Head CT can be used if suspect mass lesion, hemorrhage, or large stroke. Also used if MRI is contraindicated.

– Indications for neuro- imaging :

- Seizures in early infancy
  - Focal seizures (not indicated in benign partial seizure)
  - Developmental delay

– neuro- imaging not Indicated in :

- Non-epileptic events
  - Febrile seizures
  - BCECT (Rolandic) epilepsy
  - Absence seizure

Pre-requisites before starting AED

Is it epileptic or non epileptic event?

What is the seizure type?

Is it an epileptic syndrome?

What is the etiological diagnosis?

Whether provoked or unprovoked seizures?

Non-epileptic events

20 to 30% difficut to treat epilepsy are in fact NEE (Non-epileptic events)

Video EEG in doubtful cases.

Differentials vary according to the age of the patient.

Non-epileptic events

Age related differentials include –

- Newborns (startle, jitteriness, sleep myoclonus)
- Infants (breath holding spells, sandifer syndrome)
- Beyond infancy (syncope, pseudoseizures, migraine variants, night terrors)
- Tremors, ticks & stereotypes can occur at all ages.

Clinical Characteristics of Pseudoseizures

- **Strongly Suggest**
  - Prolonged duration of the event (10-30 minutes)
  - Preservation of consciousness despite whole body jerking
  - Bizarre and asynchronous motor movements
  - Pelvic thrusting movements
  - Not stereotyped
- **Strongly Against**
  - Injuries sustained during spells
  - Tongue laceration (especially the sides of the tongue)
  - Incontinence

Seizure type

Important to determine seizure type & for diagnosing epileptic syndrome, etiological diagnosis & choosing most appropriate AED.

Childhood Epilepsy Syndromes (CES)

- Conditions with common features such as age of onset, seizure type, EEG pattern & prognosis.
- Comprises 10% of childhood epilepsy.

Seizure type

Childhood Epilepsy Syndromes (CES)

– Benign syndromes like benign rolandic epilepsy requires only EEG for diagnosis, & has good outcome while infantile spasm has multi-factorial etiology & requires specific treatment like steroids & vigabatrin.

– Correct diagnosis of CES helps in specific treatment & timely referred to pediatric neurologist.

Benign syndrome	Age of onset	Clinical features	Drugs	Outcome
Childhood absence epilepsy (CAE)	5 to 7 yrs	Multiple absence seizures Hyperventilation 3HZ/s spike wave discharge	Sodium valproate Ehosuxamide Limotrigene	Good Remits by puberty
BCECT	4 to 12 yrs	Partial seizure Hemifacial motor In sleep Central focal spikes in EEG	No Rx for infrequent seizures For frequent seizures carbamazepine, oxcarbamazepine	Good Remits by puberty
Juvenile Myoclonic Epilepsy (JME)	12 to 18 yrs	Myoclonic jerks on awakening Positive family history EEG-spikes/poly spike waves	Sodium valproate Prefer Limotrigene, levitracetam in girls	Good Life long treatment
Catastrophic Epilepsy (*West syndrome*)	4 mo to 1 yr	Spasms in cluster Developmental delay	ACTH Vigabatin Sodium valproate Clobazam Pyridoxine	poor
Severe Myoclonic Epilepsy (Dravet Syndrome)	6 mo to 1 yr	Recurrent partial febrile seizure Psychomotor regression Myoclonic seizures	Sodium valproate Clobazam Topiramate	poor
Lennox Gestaut Syndrome	1 to 8 yrs	Multiple seizure types Psychomotor retardation Generalized slow spike waves	Sodium valproate Limotrigene, Topiramate Zonisamide	poor

Provoked vs unprovoked seizures

Febrile seizures :-

– Seizure during fever in absence of neuro infection in a neurologically normal child.

– Occur between 3months to 5 yrs of age, with peak incidence at 18 months. Onset after 6 years is unusual.

Febrile seizures

– Classified as

- Simple (SFS)
- Complex (CFS)

– SFS don’t require EEG or MRI, LP indicated in all infants with fever & seizures.

– Management includes treatment of acute attack, excluding neuro-infections, finding cause of fever, prophylaxis of future episodes & parental counseling.

Febrile seizures

SFS have good prognosis without any residual effect & remits with age.

Indications for intermittent prophylaxis :

– Frequent seizures in short period.

– Seizure lasting for more than 15 min.

– Clobazam (0.75 to 1 mg/kg/day for 3 days)

Febrile seizures

Continuous prophylaxis has limited role, given in

- Failed intermittent prophylaxis.
  - Frequent complex febrile seizures.
  - Valproic acid or phenobarbitone used.
  - Valproic acid preferred over phenobarbitone due to behavioral side effects of latter.
  - Continued for 2yrs seizure free period or 5 yrs of age whichever is earlier.

Management

Who needs treatment?

If yes, which drug, how long?

What are drug side effects?

Seizure Management - Medication

When to start medication? Definitely start if:

- there is a structural lesion, such as tumor, AV malformation, infection
- EEG with a definite epileptic pattern
- history of brain injury or stroke, CNS infection, significant head trauma
- Todd's postictal paresis
- Status epilepticus on presentation

Otherwise, get neurology consult.

Treatment of Seizures

- Provoked Seizures
  - Treatment directed to the provoking factor
- Unprovoked Seizures
  - First Seizure
    - Usually no treatment
    - Treatment can be initiated if risk of recurrence is high or if a second seizure could be devastating
  - Second Seizure
    - Diagnosis of epilepsy is established and risk of a third Seizure is high
    - Most physician treat at this stage
    - In children, some may wait for a third seizure

Management

Management of first unprovoked seizure :

- Good history & examination.
- Mostly a developmentally normal child with 1st idiopathic GTCS doesn't require long-term AED.
- Neuro- imaging must be done to rule out granuloma

Indications of AED after 1st seizure :

- Focal seizures
- Myoclonic seizures & absence seizures
- 1st episode of status- epilepticus
- Underlying structural lesion
- Severe parental anxity

Management

Drug treatment :

– No ideal AED.

– Has to be individualized

Choice of AED depends upon

. Type of seizure & epileptic syndrome

. Drug safety & tolerability

. Age & gender

. Ease of administration

. Lifestyle, pre- morbid condition

Management

Principle of therapy :

– Monotherapy is best option & is mandatory when treatment is started first.

– Always start monotherapy at low dose & titrate slowly until seizure remit or adverse effects emerge.

– Monotherapy in appropriate dosage controls seizure in 70 to 80% cases.

Management

Principle of therapy :

– If seizures are uncontrolled by first drug, choose alternate monotherapy & readually withdraw 1st drug.

– If seizures are still not controlled, refer to child neurologist. May require polytherapy, ketogenic diet or surgery.

– Before lebelling drug failure, always check compliance; rule out NEE & progressive neurological disorders.

Treatment of Established Epilepsy

- First Line
  - Approved Anti-Epileptic Drugs (AEDs)
- Second Line (intractable epilepsy)
  - Epilepsy Surgery
  - Vagus Nerve Stimulation Therapy
  - Experimental Therapy
    - AEDs
    - Implanted Devices

Management

Duration of AED :

– Generally given for minimum 2 yrs seizure free period in most of idiopathic generalized tonic epilepsies.

– AED is required for longer duration in symptomatic epilepsy, epileptic syndromes & if there are multiple risk factors for recurrence.

– In juvenile myoclonic epilepsy, small dose AED are required lifelong.

Management

How to taper AED :

- Always individualized.
- Slow taper over 3-6 months after consultation with parents & explaining risk of recurrence.
- Most epilepsies remit, relapse are reported in 11-41% greatest risk is in 1-2 yrs after drug discontinuation.
- Restart previous AED, most patients will remit again.
- Risk factors for relapses- symptomatic epilepsies, structural brain lesions, abnormal neurological signs & EEG

Management

Therapeutic drug monitering (TDM):

– Not indicated in well controlled patients without adverse drug effects.

– Indications for TDM :

- Suspicion of non-compliance
- Intractable epilepsy
    - Signs of toxicity
    - Patients on poly therapy

Antiepileptic Drugs (AED)

First Generation	Second Generation	Unconventional
Carbamazepine (Tegretol) Clonazepam (Klonopin) Clorazepate (Tranxene) Ethosuximide (Zarontin) Phenobarbital Phenytoin (Dilantin) Primidone (Mysoline) Valproic acid (Depakote)	Felbamate (Felbatol) Gabapentin (Neurontin) Lamotrigine (Lamictal) Levetiracetam (Keppra) Oxcarbazepine (Trileptal) Pregabalin (Lyrica) Tiagabine (Gabitril) Topiramate (Topamax) Zonisamide (Zonegran)	Adrenocorticotropic hormone (ACTH) Acetazolamide (Diamox) Amantadine (Symmetrel) Bromides Clomiphene (Clomid) Ethotoin (Peganone) Mephenytoin (Mesantoin) Mephobarbital (Mebaral) Methsuximide (Celontin) Trimethadione (Tridione)

Current guidelines for treatment of some common seizures in children

Type of Epilepsy	Treatment
Newly diagnosed focal seizures	
Symptomatic	Carbamazepine or lamotigine
Idiopathic	Carbamazepine or sodium valporate
Epilepsy with GTCS	Sodium valporate Lamotigine
Epilepsy with absence seizure	Sodium valporate lamotigine
Epilepsy with myoclonic Seizure	
Symptomatic	Sodium valporate with clobazam
Idiopathic	

Ongoing Therapies

Table 20–1
Classification of Epileptic Seizures

SEIZURE TYPE	FEATURES	CONVENTIONAL ANTISEIZURE DRUGS	RECENTLY DEVELOPED ANTISEIZURE DRUGS
PARTIAL SEIZURES:			
Simple partial	Diverse manifestations determined by the region of cortex activated by seizure (*e.g.*, if motor cortex representing left thumb, clonic jerking of left thumb results; if somatosensory cortex representing left thumb, paresthesias of left thumb results), lasting approximating 20 to 60 seconds. **Key feature is preservation of consciousness.**	Carbamazepine, phenytoin, phenobarbital, primidone, valproate	Gabapentin, lamotrigine
Complex partial	Impaired consciousness lasting 30 seconds to two minutes, often associated with purposeless movements such as lip smacking or hand wringing.	Carbamazepine, phenobarbital, phenytoin, primidone, valproate	Gabapentin, lamotrigine
P n secondarily zed tonic-clonic	Simple or complex partial seizure evolves into a tonic-clonic seizure with loss of consciousness and sustained contractions (tonic) of muscles throughout the body followed by periods of muscle contraction alternating with periods of relaxation (clonic), typically lasting 1 to 2 minutes.	Carbamazepine, phenobarbital, phenytoin, primidone, valproate	Gabapentin, lamotrigine
GENERALIZED SEIZURES:			
Absence seizure	Abrupt onset of impaired consciousness associated with staring and cessation of ongoing activities typically lasting less than 30 seconds.	Clonazepam, ethosuximide, valproate	Lamotrigine
Myo lonic seizure	A brief (perhaps a second), shocklike contraction of muscles which may be restricted to part of one extremity or may be generalized.	Valproate	
nic seizure	As described above for partial with secondarily generalized tonic-clonic seizures except that it is not preceded by a partial seizure.	Carmbamazepine, phenobarbital, phenytoin, primidone, valproate	

Seizures and Drugs.

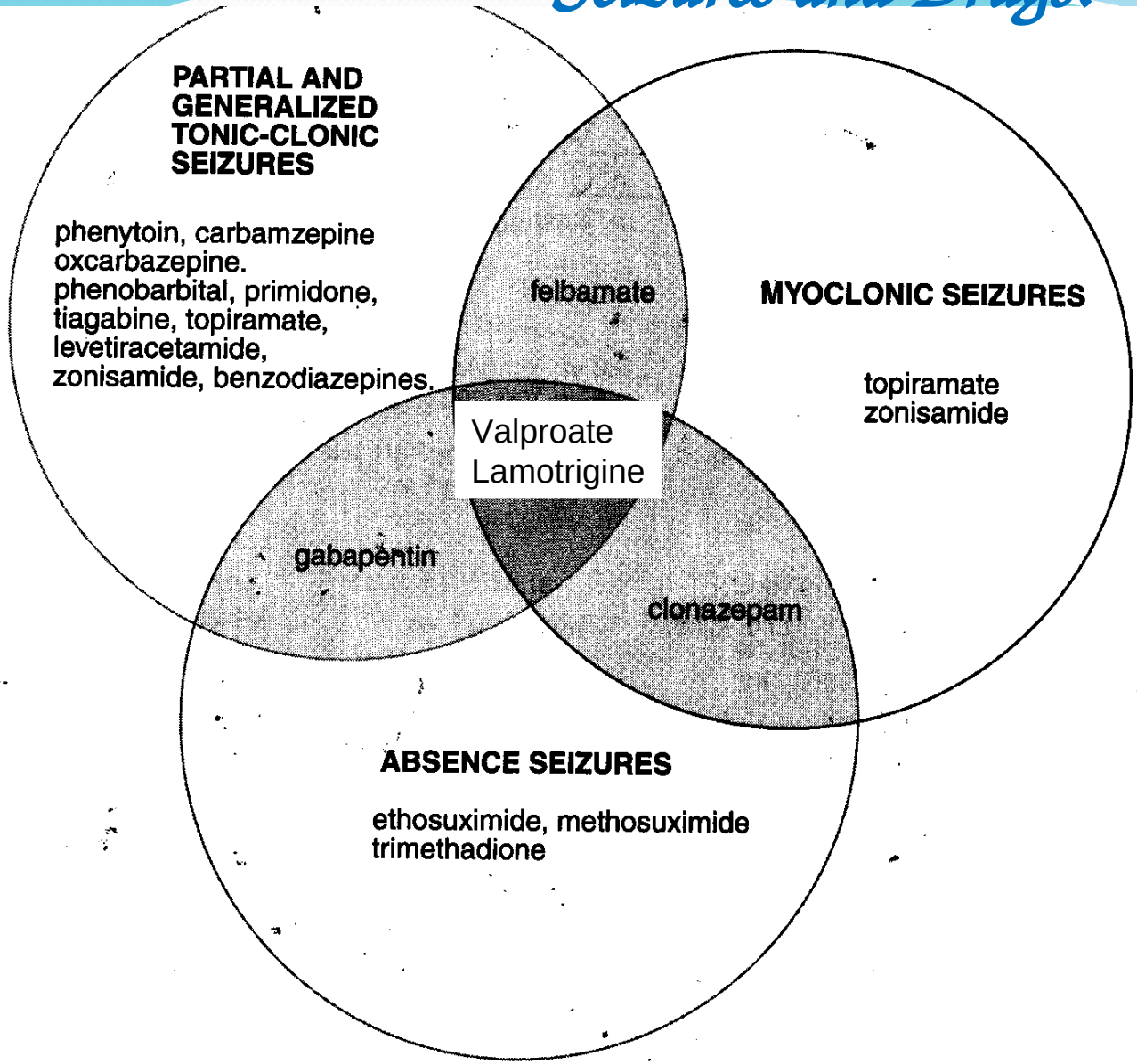


Fig. 16.2. The antiseizure drugs used in treatment of the various seizures.

Selecting AEDs

- *Type of Seizures*
- *Co-morbid conditions*
- Side Effect Profile
- Pharmacokinetics
- Cost
- Compliance

Side Effects

- Phenobarbital-sedation
- Valproate-weight gain, liver toxicity, decreased platelets, pancreatitis
- Sodium abnormalities-ox-carbazepine
- Lamotrigine-Steven's Johnson syndrome
- Topiramate-weight loss, language dysfunction, kidney stones, glaucoma
- Levetiracetam-irritability,agitation

Parental counseling

Explain the nature of disease, regular treatment & follow up.

Maintain seizure diary, video record if possible.

Demonstrate domiciliary management of seizure.

Emphasize to treat the child like other children.

Maintain normal lifestyle with caution.

Explain that most children are normal & their mental development would not be affected by drugs or disease.

Avoid precipitants like sleep deprivation & alcohol.

Algorithm for epilepsy treatment

Most appropriate First line monotherapy in low dose

Seizure uncontrolled

Gradual increase the dose

If seizure recurs,
increase to maximum tolerated dose/
side effects

Add alternate 2nd drug

1st drug taper & stop

Check compliance
Re-evaluate
Rule out progressive disorder
NEE

Seizure persists

Timely referral/ polytherapy

ACTH

- Preferred drug for the management of infantile spasms
- Dose and duration of therapy are not uniform.
- Prednisone is equally effective.
- A common schedule includes ACTH, 20 U/day (IM) for 2 wk, and if no response occurs, the dosage is increased to 30 and then 40 U/day IM for an additional 4 wk.
- Unless seizure control is complete, ACTH is replaced with oral prednisone, 2 mg/kg/24 hr for 2 wk.
- If the seizures persist, prednisone is given for an additional 4 wk.

- Side effects of ACTH include hyperglycemia, hypertension, electrolyte abnormalities, gastrointestinal disturbances, infection (including Pneumocystic carinii pneumonia), and transient brain shrinkage observed by CT scanning.
- ACTH and prednisone are equally effective for the treatment of cryptogenic and symptomatic seizures, and control can be expected in ≈70% of patients.

KETOGENIC DIET

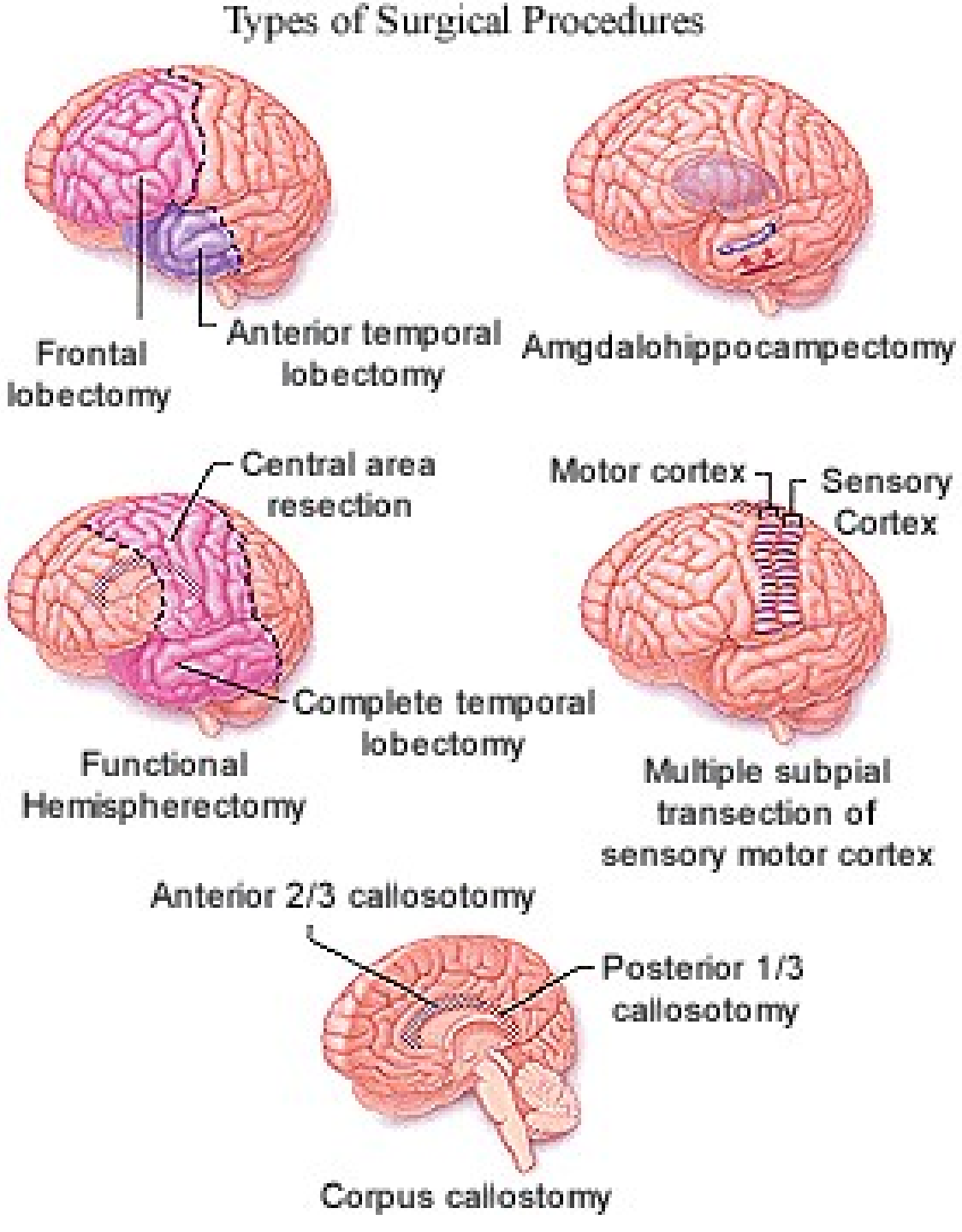
- Mechanism of action of the ketogenic diet is unknown,though some evidence shows that it exerts an anticonvulsant effect secondary to elevated levels of β-hydroxybutyrate and acetoacetate resulting from the ketosis.
- The use of valproic acid is contraindicated in association with the ketogenic diet, because the risk of hepatotoxicity is enhanced.

Treatment of Medically Intractable Epilepsy

- An epilepsy that is not responding well to medical treatment
- Most expert agree if a patient fails adequate trial of 2 AEDs, his/her epilepsy is intractable
- 25-35% of all epilepsies are intractable
- Medical treatment should be continued and other options should be explored

Treatment of Intractable Epilepsy "Other Options"

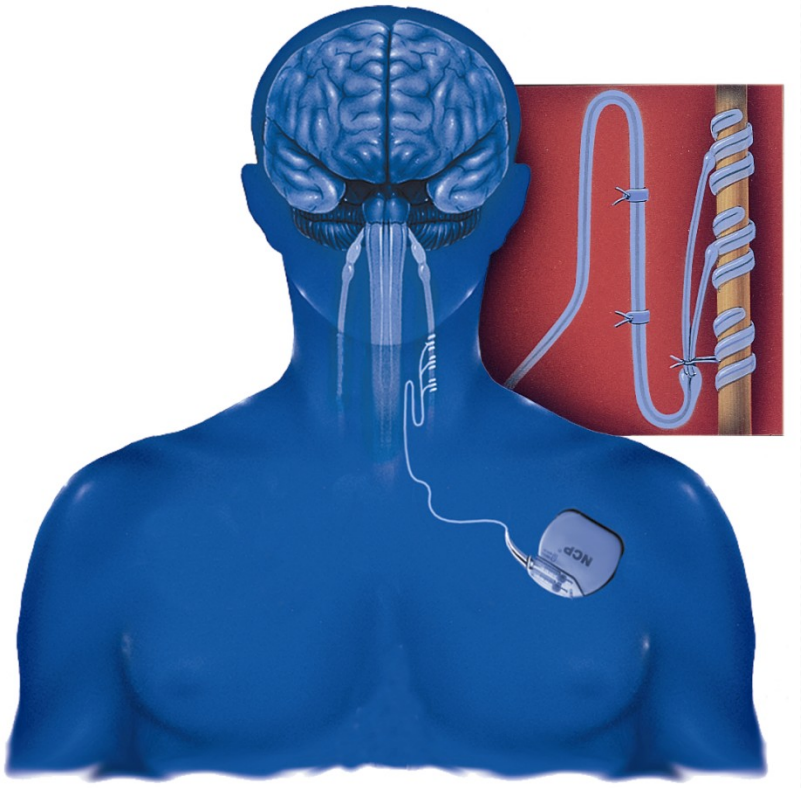
- Epilepsy Surgery
  - Removal of seizure focus
  - Requires extensive evaluation
  - Results are superior to medical treatment in patients who are good candidate
  - Surgery is associated with a small risk; however, the benefit justifies the risk
- Vagus Nerve Stimulator (VNS)
  - Not superior to medical treatment
  - Advantage: compliance, no side effects
  - Disadvantage: expensive

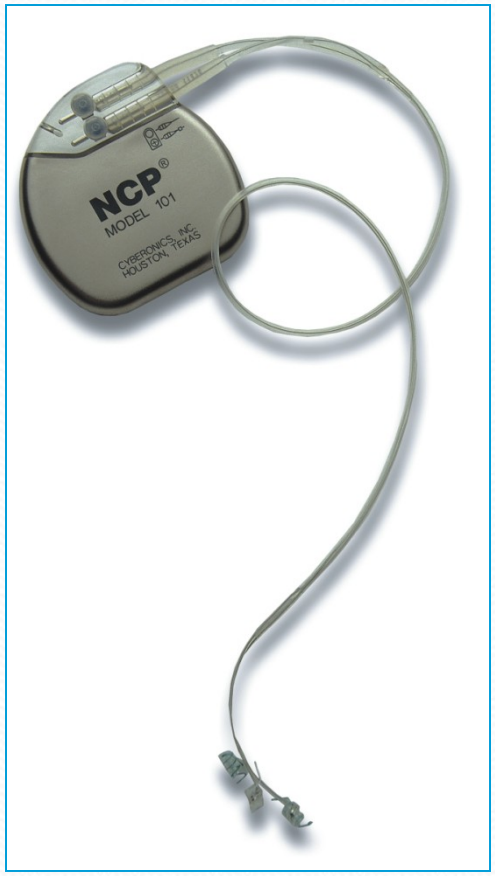

Types of Surgical Procedures
Frontal lobectomy
Anterior temporal lobectomy
Amgdalohippocampectomy
Central area resection
Complete temporal lobectomy
Functional Hemispherectomy
Motor cortex
Sensory Cortex
Multiple subpial transection of sensory motor cortex
Anterior 2/3 callosotomy
Posterior 1/3 callosotomy
Corpus callostomy

A Randomized Controlled Trial of Surgery in TLE (Wiebe et al, NEJM 2001)

- 80 patients with TLE were randomized equally to medical treatment or anterior temporal lobectomy (36/40 underwent surgery) and followed for 1 year
- **After one year; 58% (64%) of the surgical group patients were free of seizures that alter awareness vs. 8% in the medical group**
- Complications related to surgery occur in 4 patients; 1 thalamic infarct caused LT thigh sensory loss , 1 wound infection, 2 verbal memory decline
- One patient in the medical group died (unexplained), none in the surgical group
- Complications not related to surgery (depression, psychosis) were similar in both groups

Vagus Nerve Stimulator (VNS)



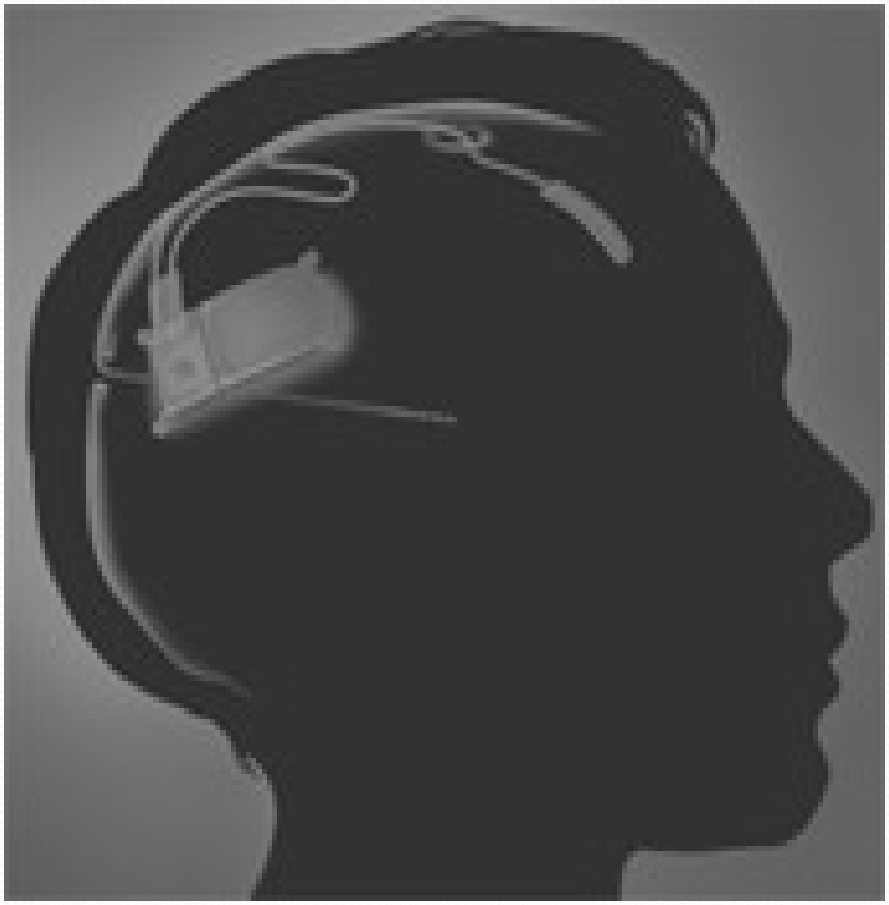


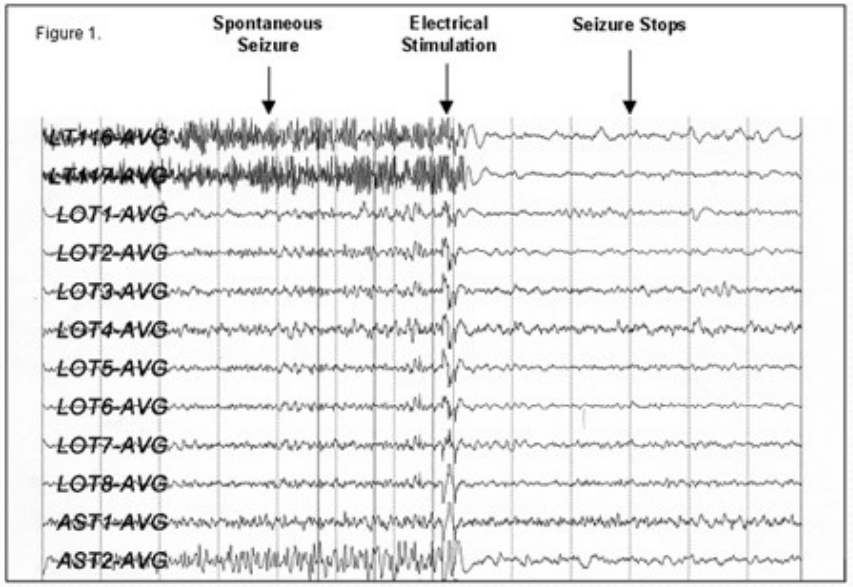
- Animal experiments have suggested that electrical stimulation of the left vagal nerve will interrupt or prevent seizures.
- In a double-blind trial of 60 children, 16 of whom were younger than 12 yr of age, left vagal nerve stimulation produced by an implanted device reduced seizures by 31–42% over 18 mo, suggesting that it may be a safe adjunctive therapy for patients refractory to other therapies.

Experimental Treatment - AEDs

- Brivaracetam
- E 2007
- GW 273225
- Retigabine
- Rufinamide
- RWJ-333369
- SPM 927
- Seletracetam
- Talampanel

Experimental Treatment- Responsive Neurostimulator (RNS)





Experimental Treatment – Deep Brain Stimulator (DBS)

Status Epilepticus

• Status epilepticus is defined commonly as repeated seizures without a return to consciousness lasting longer than 30 minutes.

• Most types of epileptic seizures can be manifested as status epilepticus.

• The 2 major types of status epilepticus,

A. generalized convulsive status epilepticus (major motor seizures and recurrent generalized tonic-clonic [GTC] convulsions) and

B. nonconvulsive status epilepticus (recurrent nonconvulsive seizures, which include absence status, partial complex status, and simple partial status),

Cont….

•Convulsive status epilepticus is the most common emergency associated with neurologic disease because brain damage and death can result from the systemic consequences of repeated GTC seizures.

•Most persons who experience GTC status epilepticus have localized cerebral disturbances as a cause and therefore have secondary generalized partial seizures.

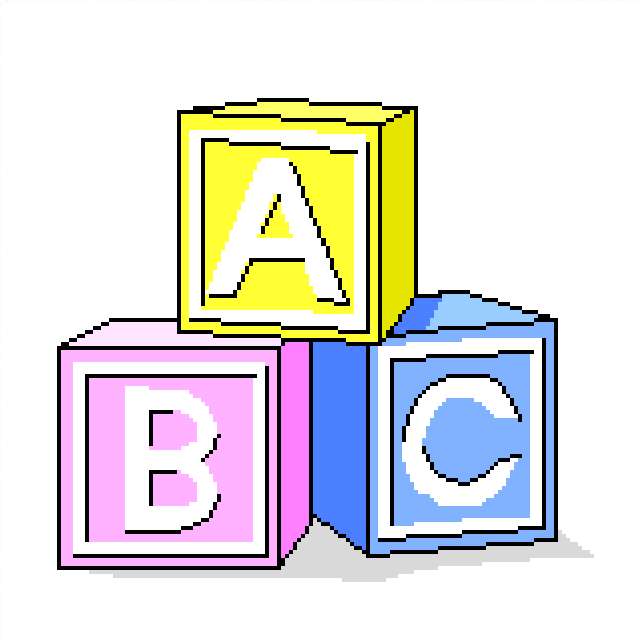
Cont…

- Repeated cerebral epileptic activity can disrupt brain structures or cause permanent neurologic or intellectual deficits.
- Common causes of status epilepticus include CNS infections, toxins, ingestions (including AED ingestion), and drug withdrawal, such as from opiates or benzodiazepines.
- The most common cause of benzodiazepine withdrawal seizures is abrupt discontinuation of clonazepam use in patients undergoing longterm therapy for seizures or anxiety.

Cont…

- Frequent repetitive GTC seizures create a life-threatening systemic condition of hyperpyrexia, failure of cerebrovascular autoregulation, acidosis, and severe hypoxia, causing hypotension, hypoperfusion of the brain, pulmonary edema, electrolyte disturbances, and eventual circulatory collapse.
- Even after cessation of status epilepticus and correction of systemic abnormalities, sepsis from aspiration pneumonia can be a late but life-threatening complication.

Acute Seizure Management



- **A**irway
- **B**reathing
- **C**irculation

- Airway management
  - Gag reflex is suppressed during ictus
  - Vomiting with aspiration
  - Lateral recumbent position
  - Remove dentures
  - Consider endotracheal intubation
    - Persistent seizure
    - Induce with benzodiazepine

Cont…

Treatment

• Therapy must be directed at suppressing all ictal (electrical seizure) activity on EEG.

• Ictal EEG activity can show the following progression:

(1) discrete seizures,

(2) merging of seizures with waxing and waning of amplitude and frequency in variable locations,

(3) continuous ictal activity,

(4) continuous ictal activity intermixed with periods of isoelectric EEG, and

(5) a periodic lateralized or generalized epileptic discharge pattern.

Cont…

- Treatment of status epilepticus consists of correction of glucose, electrolyte, magnesium, and calcium disturbances; control of blood pressure and oxygenation; and the administration of benzodiazepines and a series of routine anticonvulsants.
- At home, caregivers and parents can administer rectal diazepam, which is absorbed rapidly through blood vessels, while they call to emergency medical personnel.

TREATMENT OF SE

- Initial treatment begins with an assessment of the respiratory and cardiovascular systems.
- Best managed in an intensive care unit.
- Secure oral airway and inspected for patency
- Monitor pulse, temperature, respirations, and blood pressure,O_2 Sats
- Remove excessive oral secretions by gentle suction
- Give oxygen via well fitting mask
- Intubate and start assisted ventilation if patient not responding to oxygen via mask/ambu bagging
- Insert an NGT and an IV catheter
- If hypoglycemia is confirmed by Dextrostix, a rapid infusion of 5 mL/kg of 10% dextrose is provided.

- Take blood for a CBC,UECs,Ca, Mag,Phos, lactate, and anticonvulsant levels, if indicated and ABGs
- Blood and urine may be obtained for metabolic studies and toxicology,
- Remember that some drugs potentiate or precipitate status epilepticus (amphetamines, cocaine, phenothiazines, theophylline in toxic levels, tricyclic antidepressants).
- LP unless contraindicated. In this case, appropriate antibiotics should be administered, followed by imaging studies, before a lumbar puncture is attempted.
- If the seizures are refractory to the front-line anticonvulsants or if the patient is paralyzed and is on a respirator, continuous EEG monitoring is important to assess the frequency of seizure discharges, their location, and the response to anticonvulsant therapy.

Acute Seizure Management – Status Epilepticus

Benzodiazepines

- Lorazepam – 0.05–0.1 mg/kg IV administered slowly at 1-2 mg/min up to 10 mg. Can also be given PR or sublingual, same dose.
- Diazepam - 0.1–0.3 mg/kg IV at 2 mg/min up to 20 mg. Can also be given ET or PR at 0.2–0.5 mg/kg.
- Midazolam - 2.5-15 mg IV or 0.15–0.3 mg/kg IV/IM. Very short acting. Buccal or nasal route (0.5 mg/kg) is another option when IV access is not available

BE PREPARED TO INTUBATE!

Lorazepam (Ativan)

- Drug of choice
  - Terminates seizure within 2 minutes
  - Longer duration of action (4 to 6 hours)
  - Can be given IM or IV
  - Must be refrigerated or reconstituted
  - Dose
    - 0.1 mg/kg IV (4 mg in older children/adolescents)
    - Repeat in 10 minutes
    - 0.01 -0.1 mg/kg per hour infusion

Diazepam (Valium)

- Liquid form in room temperature
  - Easily carried by EMS for long periods
  - Rectal gel
  - IV onset is 10 to 20 seconds
  - 50% chance of recurrent seizure within 2 hours
  - Dose
    - 5 to 10 mg every 10 minutes up to 30 mg in 8 hour period
    - Rectal in pediatrics at 0.3 to 0.5 mg/kg

Midazolam (Versed)

- Onset of action is 1 minute
- Can be administered intranasally
- Has the least cardiovascular effects
- Dose
  - 0.2 mg/kg bolus (don’t exceed 2.5 mg)
  - 0.05 – 0.6 mg/kg per hour infusion
  - 0.2 mg/kg intranasally

Cont…

- Intravenous lorazepam (0.1 mg/kg per dose) usually is administered first in treating status epilepticus.
- If the seizures do not break, a second dose of intravenous lorazepam (0.1 mg./kg per dose) is followed by fosphenytoin (20 mg/kg per dose).
- Next, a loading dose of phenobarbital (20 mg/kg per dose) is considered if seizures continue.
- Intubation is a consideration if respiratory depression is observed with either benzodiazepines or phenobarbital.

Phenytoin/Fosphenytoin

- 15 up to 30 mg/kg IV (given in 10 mg/kg increments) at the rate of 1 mg/kg/min.
- Fosphenytoin has advantages over the older formulation because it is water soluble, less irritating after IV injection, and well absorbed after intramuscular injection
- 20-30 minute onset so must also use smaller doses of benzodiazepine
- Give too rapidly and may cause hypotension or arrhythmias.
- If the seizures do not recur, a maintenance dose of 3–9 mg/kg divided into two equal doses daily is begun 12–24 hr later

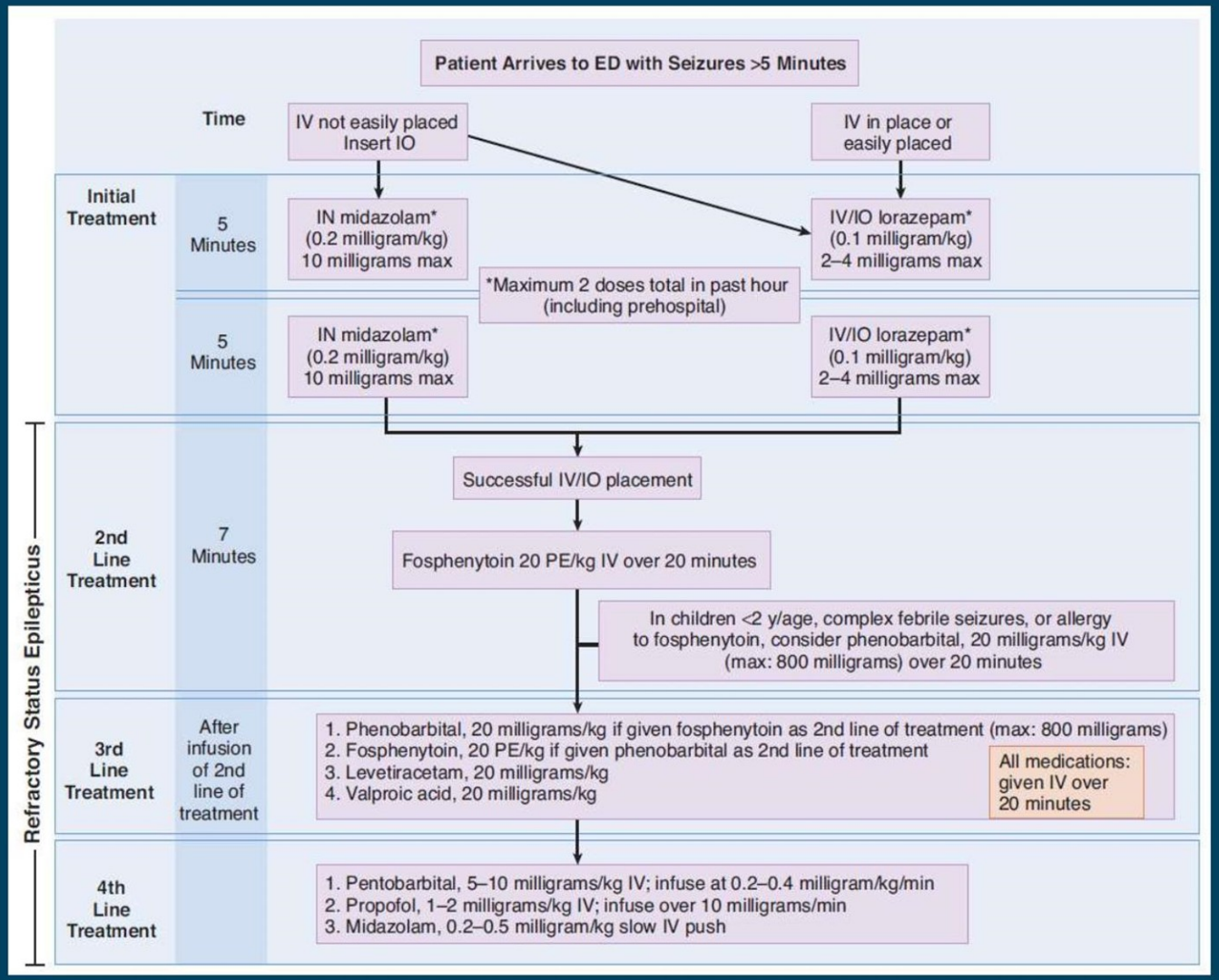
Barbiturates

- Phenobarb given in a loading dose of 15–20 mg/kg or in neonates 20–30 mg/kg IV during 10–30 min. With control of the seizures, the maintenance dose is 3–5 mg/kg/24 hr divided into two equal doses.
- May also be used, but majority of experience with this medication is the ER setting with pediatric patients on in the ICU setting for refractory seizures.
- Still may be useful in adolescentss who are seizing because of phenobarbital withdrawal.
- Be prepared to intubate and support blood pressure.
- Propofol and phenobarbital are acceptable options for treating refractory seizures in ICU setting.
- Get help from a neurologist if you are in the ICU.

- If the status epilepticus is not controlled by the preceding strategy, the physician must make some important therapeutic decisions, because it is likely the transitional period has passed.
- Choices for further drug management include a diazepam infusion, barbiturate coma, paraldehyde, or general anesthesia.
- By this stage, the patient is usually sedated and may show signs of respiratory depression, necessitating elective intubation and assisted ventilation.

- Constant IV infusion of either midazolam (0.20 mg/kg bolus, 20–400 μg/kg/hr infusion) or propofol (1–2 mg/kg, 2–10 mg/kg/hr infusion)
- Barbiturate coma-The initial IV loading dose of thiopental is 2–4 mg/kg and is then titrated to achieve a burst suppression EEG pattern, continued for at least 48 hr, followed by cessation of thiopental until the serum phenobarbital level falls to the therapeutic range.
- Barbiturate coma requires careful monitoring because hypotension due to myocardial depression often requires pressor therapy.

- General anesthesia probably acts by reversing cerebral anoxia and the concomitant metabolic abnormalities, allowing the previously administered anticonvulsants to exert their effect.


Patient Arrives to ED with Seizures >5 Minutes
Time
IV not easily placed
Insert IO
IV in place or
easily placed
Initial Treatment
5 Minutes
IN midazolam*
(0.2 milligram/kg)
10 milligrams max
IV/IO lorazepam*
(0.1 milligram/kg)
2–4 milligrams max
*Maximum 2 doses total in past hour
(including prehospital)
5 Minutes
IN midazolam*
(0.2 milligram/kg)
10 milligrams max
IV/IO lorazepam*
(0.1 milligram/kg)
2–4 milligrams max
Refractory Status Epilepticus
Successful IV/IO placement
2nd Line Treatment
7 Minutes
Fosphenytoin 20 PE/kg IV over 20 minutes
In children <2 y/age, complex febrile seizures, or allergy
to fosphenytoin, consider phenobarbital, 20 milligrams/kg IV
(max: 800 milligrams) over 20 minutes
3rd Line Treatment
After infusion of 2nd line of treatment
1. Phenobarbital, 20 milligrams/kg if given fosphenytoin as 2nd line of treatment (max: 800 milligrams)
2. Fosphenytoin, 20 PE/kg if given phenobarbital as 2nd line of treatment
3. Levetiracetam, 20 milligrams/kg
4. Valproic acid, 20 milligrams/kg
All medications:
given IV over
20 minutes
4th Line Treatment
1. Pentobarbital, 5–10 milligrams/kg IV; infuse at 0.2–0.4 milligram/kg/min
2. Propofol, 1–2 milligrams/kg IV; infuse over 10 milligrams/min
3. Midazolam, 0.2–0.5 milligram/kg slow IV push

Conclusions

Current childhood epilepsy treatment is based on specific epilepsy syndromes.

Conform diagnosis, type of seizure & syndrome, do EEG at the onset & follow principle of monotherapy.

For better compliance, always explain nature of disease to parents.

Goal should be to maintain quality of life.

In resistant cases, early referral will help in offering other options timely.

Reference

- Nelson’s textbook of pediatrics 21st edition
- *Up to date*

Thank You