Epilepsy in children

DR. REDAB AL-GHAWANMEH

Objectives

- Definitions
- Epidemiology
- Classification
- Etiology
- Epileptic syndroms
- Treatment
- Prognosis
- Disorders that mimic seizures

Definitions

- Seizure
- Epilepsy
- Status Epilepticus
- Febrile convulsions

Seizure

- Transient occurrence of signs and/or symptoms(change in LOC, motor activity, sensory phenomena or inappropriate behavior) resulting from abnormal excessive or synchronous neuronal activity in the brain

Epilepsy

- Epilepsy: two or more unprovoked attacks, in a time frame longer than 24 h.
- Epileptic syndrome: one or more specific seizure types, has a specific age of onset and specific prognosis.

Status epilepticus

- Continuous seizure >= 30 min, generalized or focal, during which the patient remains unconscious or has two or more sequential seizures without full recovery of consciousness between seizures.
- New definition 5 min
- Treatment of Status epilepticus

Febrile convulsion

- A Seizure is association with febrile illness in the absence of CNS infection, or acute electrolyte imbalance in children between 6-60m without prior afebrile seizures.
- Age: 6-60 months (peak 18)
- 2-5% of children
- Most common form of seizures in children.

Febrile convulsion –cont

- Classification:

-Simple: generalized

<15 min

not recurring in 24 h

-complex: focal

>15 min

multiple

Febrile convulsion –cont

- After first febrile seizure: 30% recurrence of a second febrile seizure.
- 50% after two or more seizures.

Risk factors for recurrence of FC

- Age<12 m
- Family history of febrile convulsion.
- Height of temperature (the lowest the highest the risk)
- Duration of fever (the shorter the higher the risk)
- Male gender
- Daycare attendance

Risk of developing epilepsy

- Risk of developing epilepsy after single FC is not different than the risk in the general population
- Complex febrile convulsion
- Neurodevelopmental abnormalities.
- Family history of epilepsy
- Duration of the fever before the seizure

Treatment of FC

- Management of the acute febrile illness
- Counseling and education of the family
- Treatment of the seizure: consider rectal diazepam as rescue treatment
- Prophylactic treatment: debate

Epidemiology of epilepsy

- Birth-16 year: 4-10% experience at least one seizure, mostly in childhood.
- Cumulative risk(risk to develop epilepsy throughout an individual lifetime): 3-5%

ILAE 2017 Classification of Seizure Types Expanded Version[1]

Focal Onset

Aware | Impaired Awareness

Motor Onset
- automatisms
- atonic[2]
- clonic
- epileptic spasms[2]
- hyperkinetic
- myoclonic
- tonic

Non-Motor Onset
- autonomic
- behavior arrest
- cognitive
- emotional
- sensory

focal to bilateral tonic-clonic

Generalized Onset

Motor
- tonic-clonic
- clonic
- tonic
- myoclonic
- myoclonic-tonic-clonic
- myoclonic-atonic
- atonic
- epileptic spasms[2]

Non-Motor (absence)
- typical
- atypical
- myoclonic
- eyelid myoclonia

Unknown Onset

Motor
- tonic-clonic
- epileptic spasms

Non-Motor
- behavior arrest

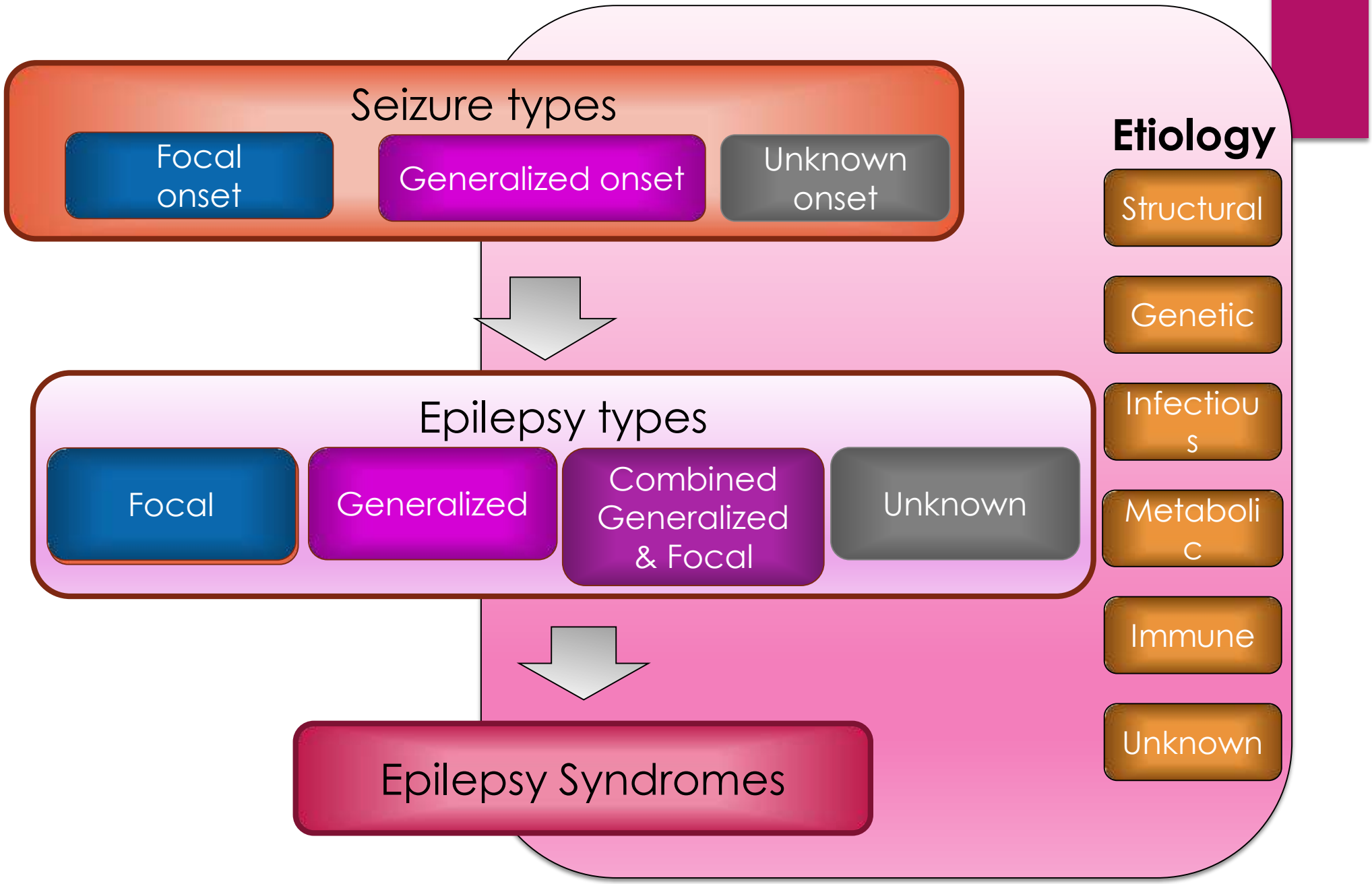
Unclassified[3]

1 Definitions, other seizure types and descriptors are listed in the accompanying paper and glossary of terms.

2 These could be focal or generalized, with or without alteration of awareness

3 Due to inadequate information or inability to place in other categories

From *Fisher et al. Instruction manual for the ILAE 2017 operational classification of seizure types. Epilepsia doi:*



Continuum/Epilepsy Overview and Revised Classification of Seizures and Epilepsies
Alison M. Pack, MD, MPHEpilepsy p. 306-321April 2019, Vol.25, No.2doi:
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Epilepsia

SPECIAL REPORT

ILAE classification and definition of epilepsy syndromes with onset in neonates and infants: Position statement by the ILAE Task Force on Nosology and Definitions

Sameer M. Zuberi[1] | Elaine Wirrell[2] | Elissa Yozawitz[3] | Jo M. Wilmshurst[4] | Nicola Specchio[5] | Kate Riney[6,7] | Ronit Pressler[8,9] | Stephane Auvin[10] | Pauline Samia[11] | Edouard Hirsch[12] | Santiago Galicchio[13] | Chahnez Triki[14] | O. Carter Snead[15] | Samuel Wiebe[16] | J. Helen Cross[17,18] | Paolo Tinuper[19,20] | Ingrid E. Scheffer[21] | Emilio Perucca[22,23] | Solomon L. Moshé[24,25,26] |

- *It divided syndromes in two groups: self-limited epilepsy syndromes and developmental and epileptic encephalopathies
- *It introduced the concept of epilepsy syndromes determined primarily by etiology.

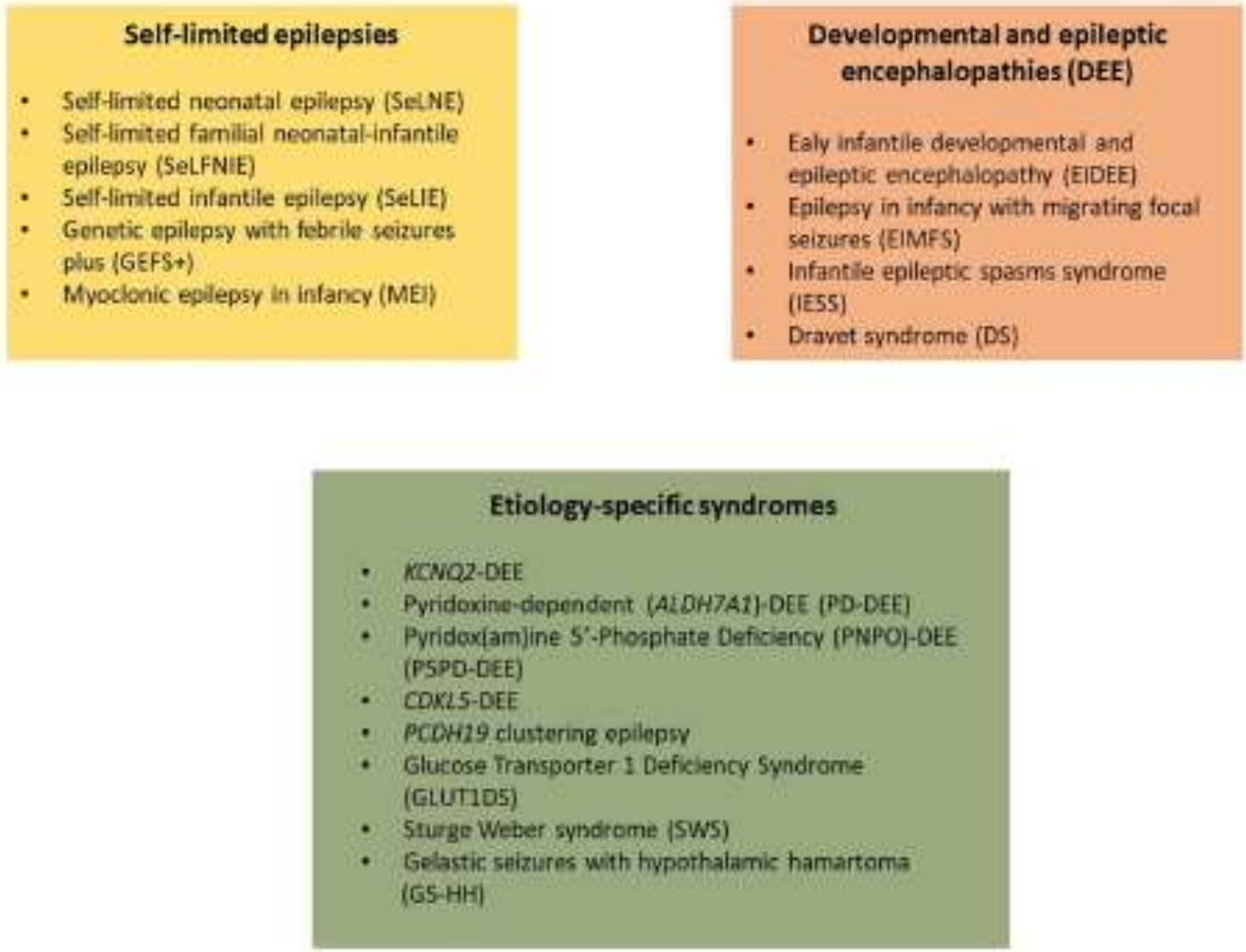


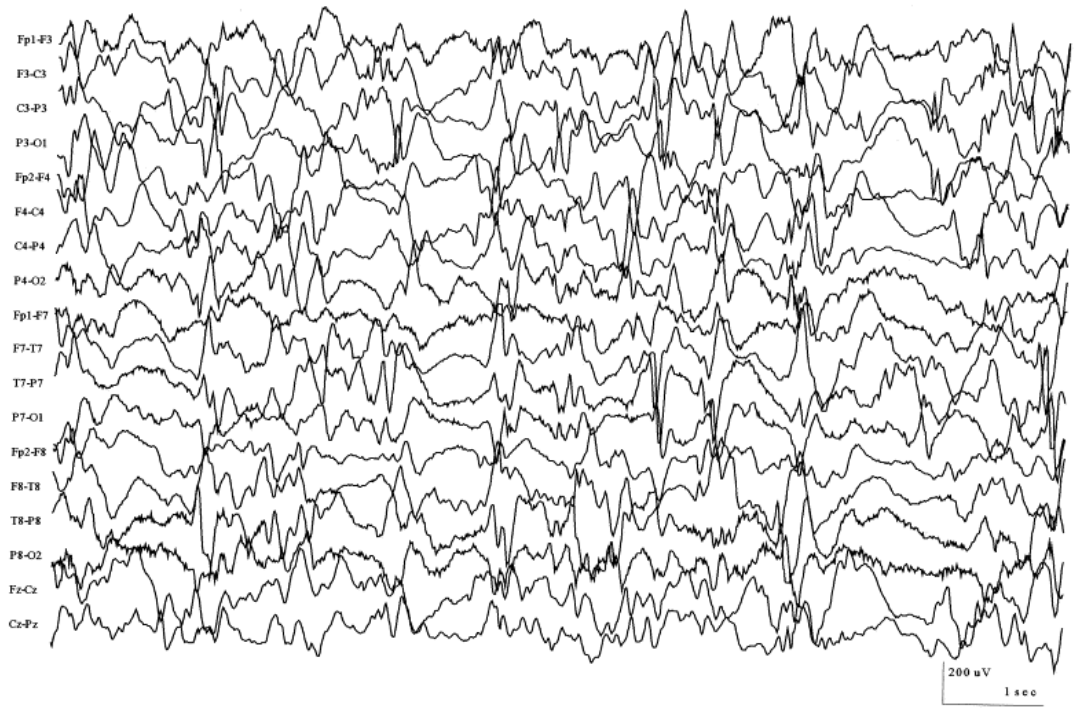
FIGURE 1 Organization of epilepsy syndromes that begin in the neonates and infants. Syndromes are broadly divided into Self-Limited Epilepsies (where there is likely to be spontaneous remission) and Developmental and Epileptic Encephalopathies (disorders where there is developmental impairment related to both the underlying aetiology independent of epileptiform activity and the epileptic encephalopathy). Etiology-specific epilepsy syndromes are due to specific genetic, structural, metabolic, immune or infectious etiologies, and have consistent electroclinical features, management, and prognostic implications. Most etiology-specific syndromes that begin in the neonatal or infantile period are DEEs. ALDH7A1, aldehyde dehydrogenase 7 family member A1; CDKL5, cyclin-dependent kinase-like 5; KCNQ2, potassium voltage-gated channel subfamily Q member 2; PCDH19, protocadherin19; PNPO, Pyridoxamine 5'-Phosphate Oxidase

Infantile epileptic spasms syndrome (IESS)

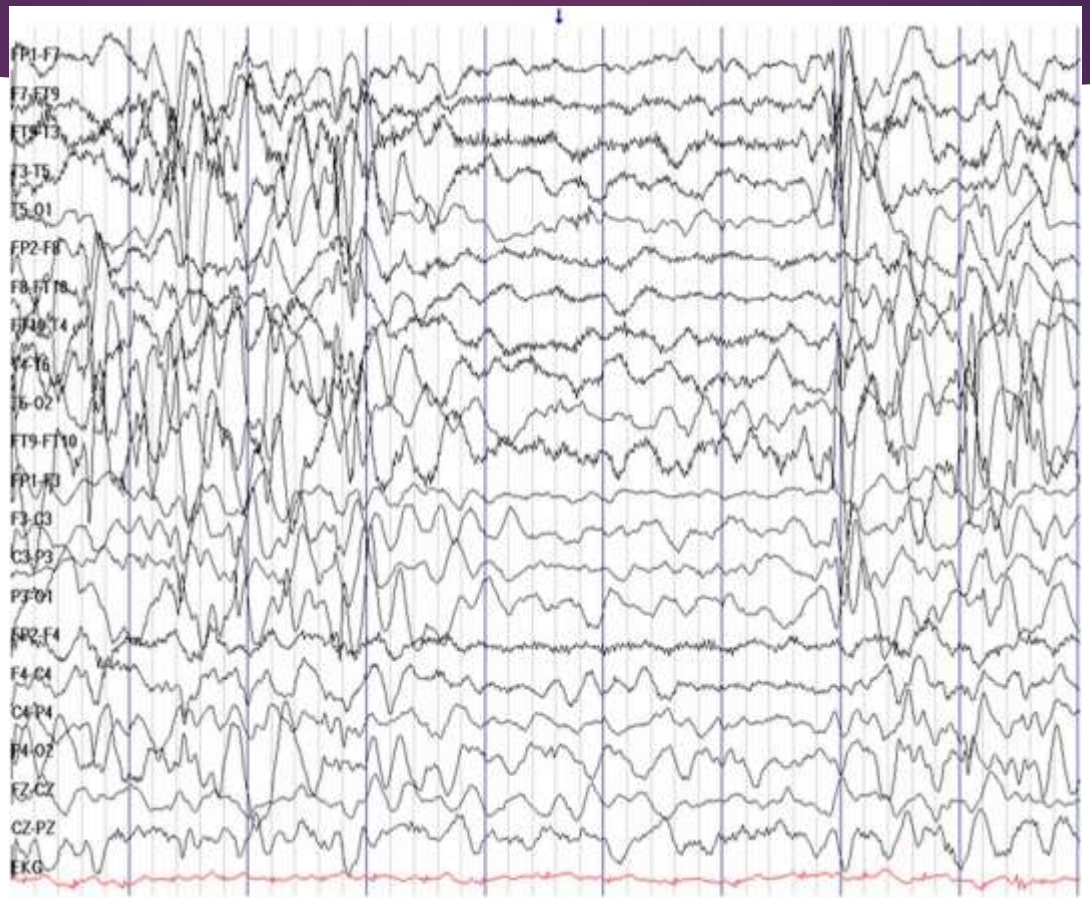
IESS is a term proposed to encompass both West syndrome as well as infants presenting with epileptic spasms who do not fulfil all the criteria for West syndrome

West syndrome classically referred to the triad of epileptic spasms, hypsarrhythmia, and developmental stagnation or regression. However, infants with IESS often lack one of these three criteria.

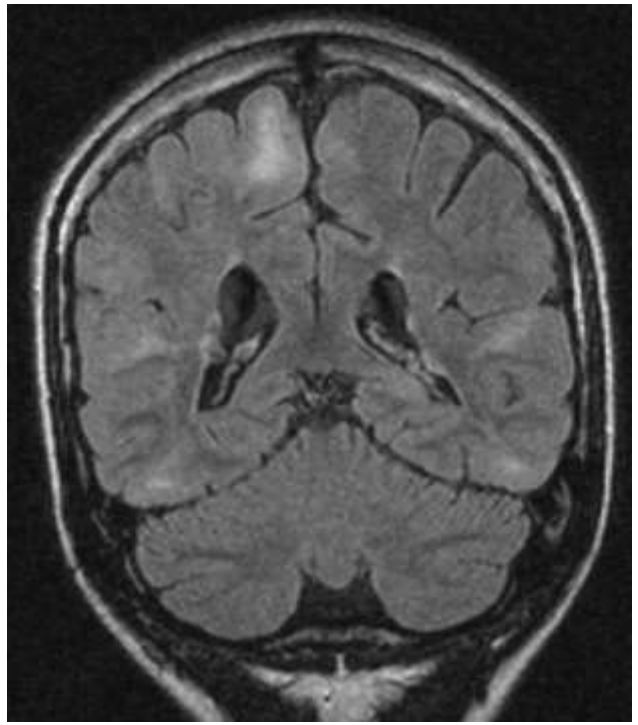
hypsarrhythmia



Spasms


FP1-F7
F7-FT9
FT9-T3
T3-T5
T5-O1
FP2-F8
F8-FT10
FT10-T4
T4-T6
T6-O2
FT9-FT10
FP1-F3
F3-C3
C3-P3
P3-O1
FP2-F4
F4-C4
C4-P4
P4-O2
FZ-CZ
CZ-PZ
EKG

Cortical tubers in tuberous sclerosis



Infantile epileptic spasms syndrome (IESS)..

- Treatment

  -ACTH, most effective

  -High dose oral corticosteroid(S/E: immunosuppression, HTN, diabetes)

  -Vigabatrin particularly in tuberous sclerosis(S/E: irreversible visual field deficit with prolonged use

- Prognosis – developmental delay, many will have seizures later in life, can evolve to Lennox Gastaut Syndrome

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Epilepsia

SPECIAL REPORT

International League Against Epilepsy classification and definition of epilepsy syndromes with onset in childhood: Position paper by the ILAE Task Force on Nosology and Definitions

Nicola Specchio[1] | Elaine C. Wirrell[2] | Ingrid E. Scheffer[3] | Rima Nabbout[4] | Kate Riney[5,6] | Pauline Samia[7] | Marilisa Guerreiro[8] | Sam Gwer[9] | Sameer M. Zuberi[10] | Jo M. Wilmshurst[11] | Elissa Yozawitz[12] | Ronit Pressler[13] | Edouard Hirsch[14] | Sam Wiebe[15] | Helen J. Cross[16] | Emilio Perucca[17,18] | Solomon L. Moshé[19] | Paolo Tinuper[20,21] | Stéphane Auvin[22]

- The ILAE produced a classification of epileptic syndromes presenting in childhood
- Syndromes with onset in childhood are divided into three categories: self-limited focal epilepsies, generalized epilepsies, and developmental and/or epileptic encephalopathies
- Each syndrome has mandatory seizure types, EEG features, age at onset, and findings from key investigations
- Precise identification of an epileptic syndrome can provide useful information on prognosis and management

TABLE 1 Childhood epilepsy syndromes

Self-limited focal epilepsies		Genetic generalized epilepsies		DEEs	
Epilepsy syndromes with focal seizures	**Formerly known as**	**Epilepsy syndromes with generalized seizures**	**Formerly known as**	**DEEs**	**Formerly known as**
SeLECTS	Childhood epilepsy with centrotemporal spikes, (benign) Rolandic epilepsy, (benign) epilepsy with centrotemporal spikes	CAE[a]	Pyknolepsy, petit mal	EMAtS	Doose syndrome
SeLEAS	Panayiotopoulos syndrome, early onset (benign) occipital epilepsy	EEM	Jeavons syndrome	LGS	No changes
COVE	Late onset (benign) occipital epilepsy or idiopathic childhood occipital epilepsy–Gastaut type	EMA	Bureau and Tassinari syndrome	DEE-SWAS EE-SWAS Landau–Kleffner syndrome (subtype of EE-SWAS)	Epileptic encephalopathy with continuous spike-and-wave in sleep, atypical (benign) partial epilepsy (pseudo-Lennox syndrome)
POLE	Idiopathic photosensitive occipital lobe epilepsy			FIRES HHE	AERRPS, DESC No changes

Note: This table includes identified syndromes of this age group and not all epilepsy types.

Abbreviations: AERRPS, acute encephalitis with refractory, repetitive partial seizures; CAE, childhood absence epilepsy; COVE, childhood occipital visual epilepsy; DEE, developmental and/or epileptic encephalopathy; DEE-SWAS, developmental epileptic encephalopathy with spike-and-wave activation in sleep; DESC, devastating epileptic encephalopathy in school-aged children; EEM, epilepsy with eyelid myoclonia; EE-SWAS, epileptic encephalopathy with spike-and-wave activation in sleep; EMA, epilepsy with myoclonic absence; FIRES, febrile infection-related epilepsy syndrome; HHE, hemiconvulsion–hemiplegia–epilepsy syndrome; LGS, Lennox–Gastaut syndrome; POLE, photosensitive occipital lobe epilepsy; SeLEAS, self-limited epilepsy with autonomic seizures; SeLECTS, self-limited epilepsy with centrotemporal spikes.

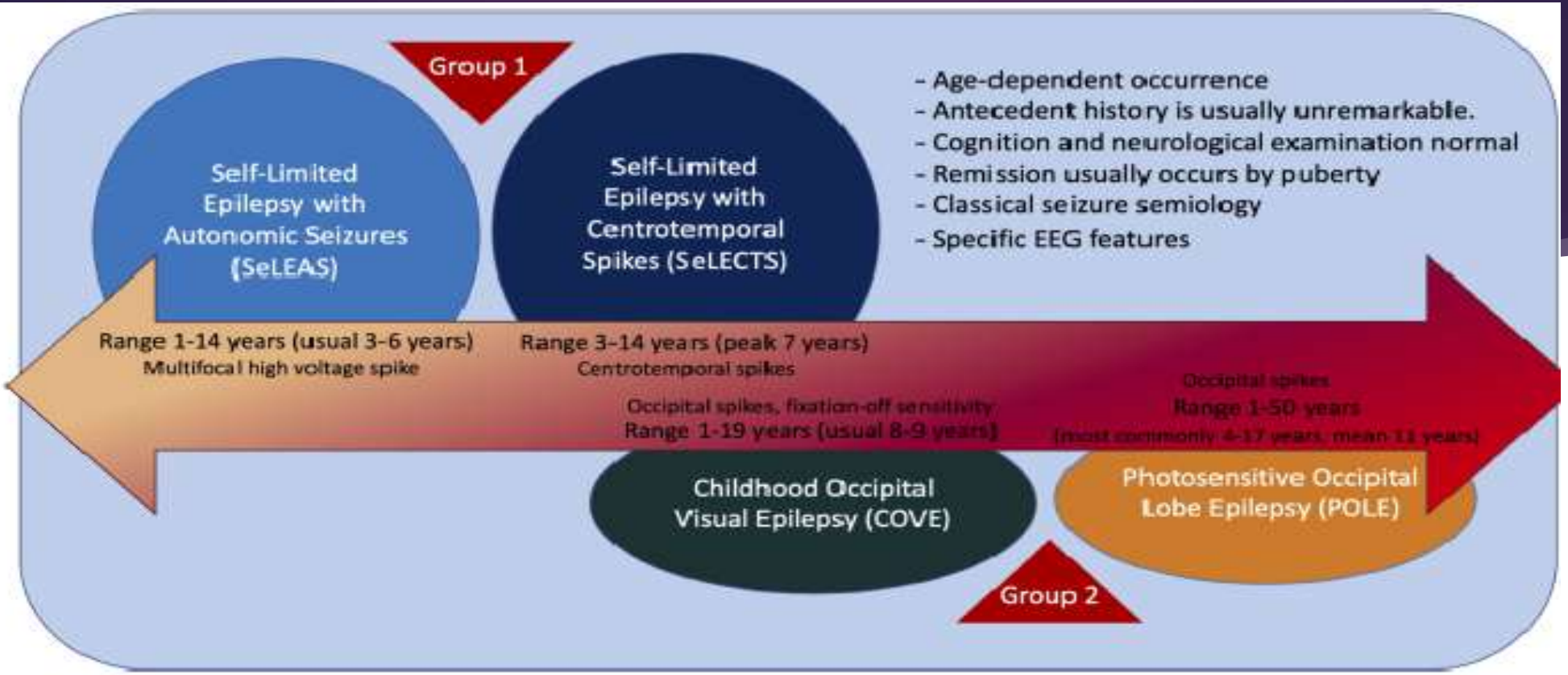

FIGURE 1 Self-limited focal epilepsies of childhood (SeLFE) syndromes are a group of conditions characterized by age-dependent occurrence in otherwise normal children. Cognition and neurological evaluation are typically normal. Remission occurs in almost all patients by puberty. Presumed genetic factors have an important role. Seizure semiology and electroencephalographic (EEG) features are specific for each of the syndromes included in this group. Within the SeLFEs, we recognize two levels of syndromes, based on the long-term prognosis. The first subgroup (Group 1) includes two syndromes: the former syndromes of childhood epilepsy with centrotemporal spikes or benign epilepsy of childhood with centrotemporal spikes or benign Rolandic epilepsy, now renamed as self-limited epilepsy with centrotemporal spikes; and of Panayiotopoulos syndrome or early onset benign occipital epilepsy, now renamed as self-limited epilepsy wi autonomic seizures. The second subgroup (Group 2) includes two syndromes: the former syndromes of late onset benign occipital epilepsy or Gastaut syndrome or idiopathic childhood occipital epilepsy–Gastaut type, now renamed as childhood occipital visual epilepsy; and of idiopathic photosensitive occipital lobe epilepsy, now renamed as photosensitive occipital lobe epilepsy. In Group 1, remission is expected all cases. In Group 2, remission is highly likely; however, a few patients may have persistence of seizures after adolescence. In the figure ar

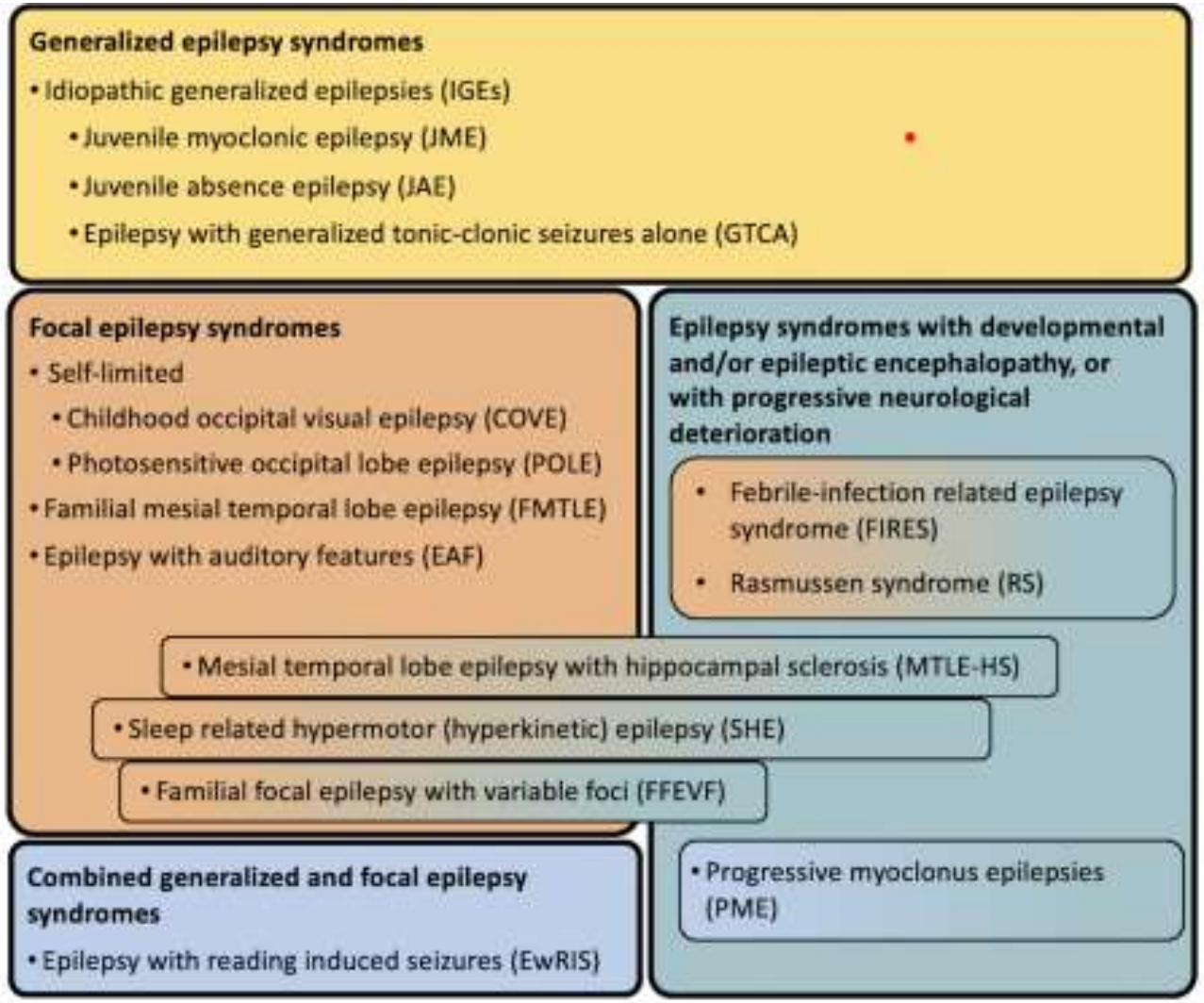

FIGURE 1 The epilepsy syndromes that begin at a variable age grouped by epilepsy type and whether they are associated with developmental and/or epileptic encephalopathy (D and/or EE) or progressive neurological deterioration. Some patients with the focal epilepsy syndromes MTLE-HS, SHE, and FFEVF may have cognitive, neurologic, or psychiatric impairment related to their etiology or epilepsy (D and/or EE). All patients with established PME (a combined generalized and focal epilepsy syndrome) and FIRES and RS (focal epilepsy syndromes) will have D and/or EE or progressive neurological impairment. The authors note that other epilepsy syndromes may be identified in the future

Self-limited focal epilepsy syndromes with presumed complex inheritance

- Self-limited focal epilepsies (SeLFEs) account for up to 25% of all pediatric epilepsies.
- They have age-dependent onset and remission, characteristic seizure semiologies, specific EEG features (with normal EEG background), are drug-responsive, and cognition is typically normal.
- The etiology is genetic, supported by a higher incidence of epilepsy in families and familial predisposition to the EEG trait. However, no genes have been identified, and the etiology is presumed complex inheritance at a susceptible age.
- SeLFEs predominantly begin in childhood, but two syndromes can begin at a variable age: COVE and POLE.
- Although remission is expected in these syndromes, it may not occur in all patients.

Self-limited epilepsy with centrotemporal spikes/ SeLECTS

- Focal seizures with dysarthria, sialorrhea, dysphasia, and unilateral clonic or tonic–clonic movement of mouth in wakefulness or sleep and/ or nocturnal focal to bilateral tonic–clonic seizures in sleep only
- If seizures occur during sleep, they are seen within 1 h of falling asleep or 1–2 h prior to awakening
- Remission by mid to late adolescence
- No developmental regression
- EEG: High-amplitude, centrotemporal biphasic epileptiform abnormalities

Self-limited epilepsy with autonomic features

- Focal autonomic seizures, with or without impaired awareness
- Autonomic symptoms often involve prominent retching and vomiting, but may also include malaise, pallor, flushing, abdominal pain, and pupillary or cardiorespiratory changes
- Remission by early to mid adolescence
- No developmental regression

COVE/ Childhood occipital visual epilepsy

- characterized by frequent brief focal aware sensory seizures with visual phenomena during wakefulness, often followed by headache.
- Onset up to age 19 years has been described.
- The EEG shows a normal background with interictal occipital sharp- or spike-andwave, seen mainly in sleep.
- Remission occurs in 50%–80% of patients within 2–7 years after onset with or without administration of antiseizure medication

POLE /photosensitive occipital lobe epilepsy

- characterized by photic-induced focal aware sensory seizures with visual phenomena.
- Onset in adulthood has been described.
- There is a strong female predominance.
- The EEG shows normal background, with interictal occipital spike- or polyspike-and-wave, facilitated by eye closure and intermittent photic stimulation.
- Generalized spike-and-wave can also be seen.

Familial mesial temporal lobe epilepsy FMTLE

- It has been estimated that FMTLE accounts for almost one fifth of newly diagnosed cases of nonlesional mesial temporal lobe epilepsy.
- Age at seizure onset varies between 3 and 63 years, with symptoms usually starting in adolescence or adulthood.
- A female predominance has been reported.
- Individuals with FMTLE generally have normal intellectual development and no associated neurological abnormalities.

FMTLE…

- Patients typically present with focal aware seizures mainly consisting of intense déjà vu, which is reported by >70% of affected individuals.
- Manifestations commonly associated with déjà vu include dreamy perceptions, fear or panic, slow motion, visual or auditory illusions, and autonomic manifestations (a rising visceral or epigastric sensation, nausea, tachycardia, sweating, flushing, or pallor).
- In most patients with the typical form of FMTLE, seizures are mild and occur infrequently.
- Good response to treatment

Etiology-specific epilepsy syndromes

- Etiology-specific epilepsy syndromes can be identified when there is an etiology for the epilepsy that is associated with a clearly defined, relatively uniform and distinct clinical phenotype in most affected individuals (clinical presentation, seizure types, comorbidities, course of illness, and/or response to specific therapies), as well as consistent EEG, neuroimaging and/or genetic correlates.

Mesial temporal lobe epilepsy with hippocampal sclerosis

- Age at seizure onset :typically in adolescent and young adult years
- There is no sex predominance.
- Antecedent, birth, and neonatal history is typically normal.
- Early developmental milestones are within normal limits.
- A past history of febrile seizures in early childhood may be found, and prolonged febrile seizures in childhood may cause HS.

- Cognitive comorbidity is recognized, with deficits in verbal memory associated with MTLE-HS affecting the dominant (usually left) mesial temporal lobe and deficits in visual memory associated with MTLE-HS affecting the nondominant temporal lobe
- often drug-resistant.
- Epilepsy surgery, in selected etiologies, may transform outcome from uncontrolled drug-resistant seizures to full remission
- The best surgical outcome is seen when the structural abnormality is well defined on imaging

Seizurs: Focal aware or impaired awareness seizures occur with semiological features referable to medial temporal lobe networks.

Imaging HS: characterized by decreased hippocampal volume (best seen on coronal)with increased hippocampal signal intensity

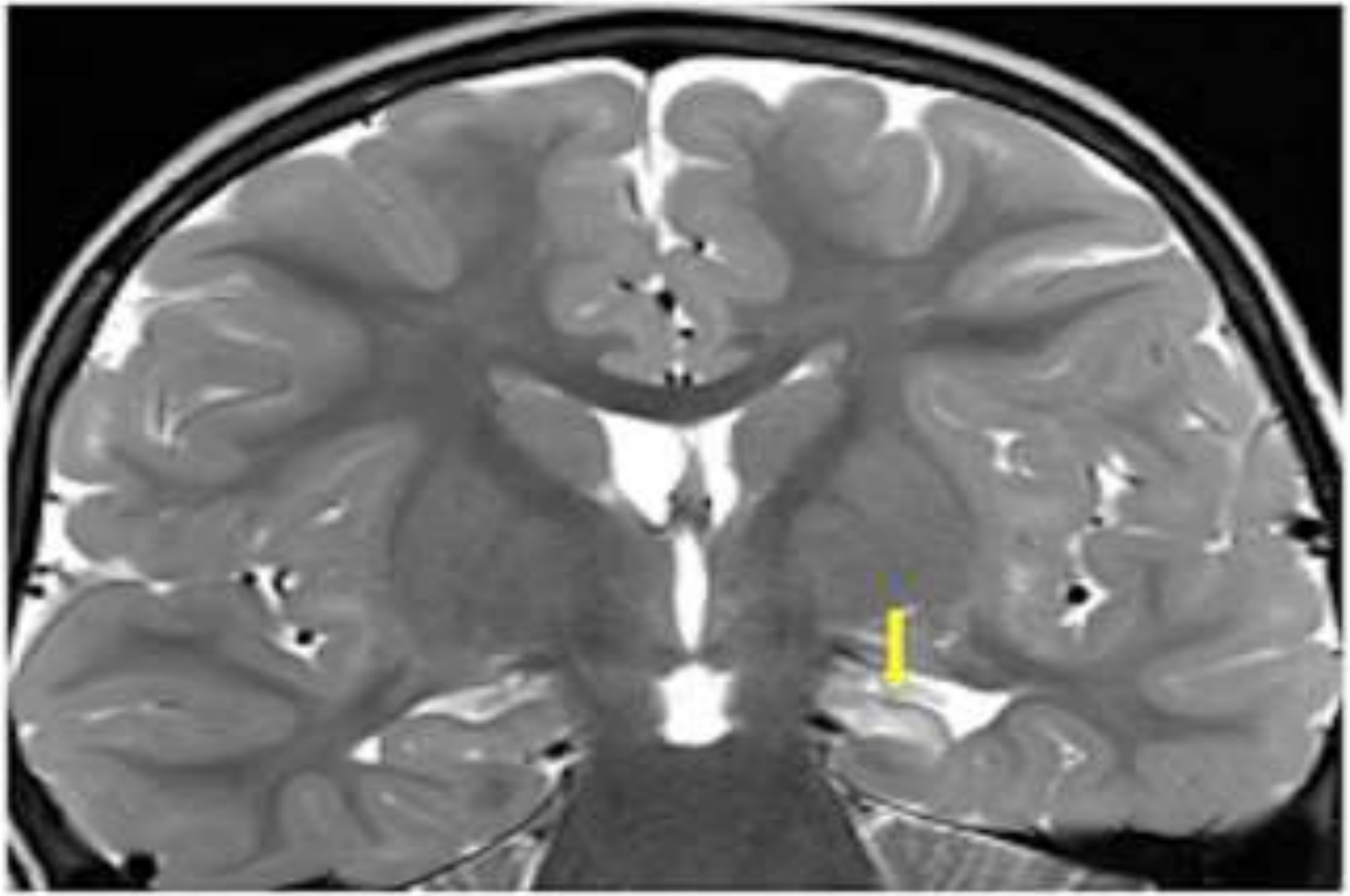

FIGURE 4 T2-weighted imaging in a coronal plane at right angles to the long axis of the hippocampus showing increased signal and loss of volume in the left hippocampus (arrow)

Rasmussen syndrome

- The age at onset is 1–10 years (median = 6 years Both sexes are equally affected.
- Antecedent and birth history and development are usually normal. Over time, cognitive impairment emerges.
- Focal hemispheric seizures that increases in frequency over weeks and months
- frequent drug-resistant seizures and progressive neurological deterioration (hemiparesis, homonymous hemianopia, cognitive impairment).

Imaging MRI

- usually normal in the early phase of the disease, With time, there is progressive atrophy of the affected hemisphere often starting in the insular region, with enlargement of the temporal horn of the lateral ventricle and Sylvian fissure.
- Atrophy is usually seen within the first year of onset and correlates with progressive hemiparesis.

Epilepsy syndromes with developmental and/or epileptic encephalopathy and epilepsy syndromes with progressive neurological deterioration

- The term "DE" applies when there is onset of a condition manifesting with cognitive, neurological, or psychiatric impairment, stagnation, or regression, due directly to the underlying etiology.
- In contrast, an EE is present when the encephalopathy is caused by the epileptic activity.
- The term "developmental and epileptic encephalopathy" (DEE) is used when both factors contribute to the patient's condition.
- The term "DE" can be challenging to apply in an older individual who has completed all development normally. To address this, the Task Force proposes the term "progressive neurological deterioration" instead of DE for such patients who develop cognitive, neurological, or psychiatric impairment due directly to the underlying etiology

Progressive myoclonus epilepsies

- rare, and is caused by a heterogenous group of underlying genetic etiologies.
- It is recognized in the presence of :
  - Myoclonus
  - progressive motor and cognitive impairment
  - sensory and cerebellar signs
  - abnormal background slowing on EEG
  - appear in an individual with prior normal development and cognition.
  - Photosensitivity is a common feature of many etiologies of PME.
- There may be a family history, with autosomal recessive inheritance in most cases, but PME can be sporadic.

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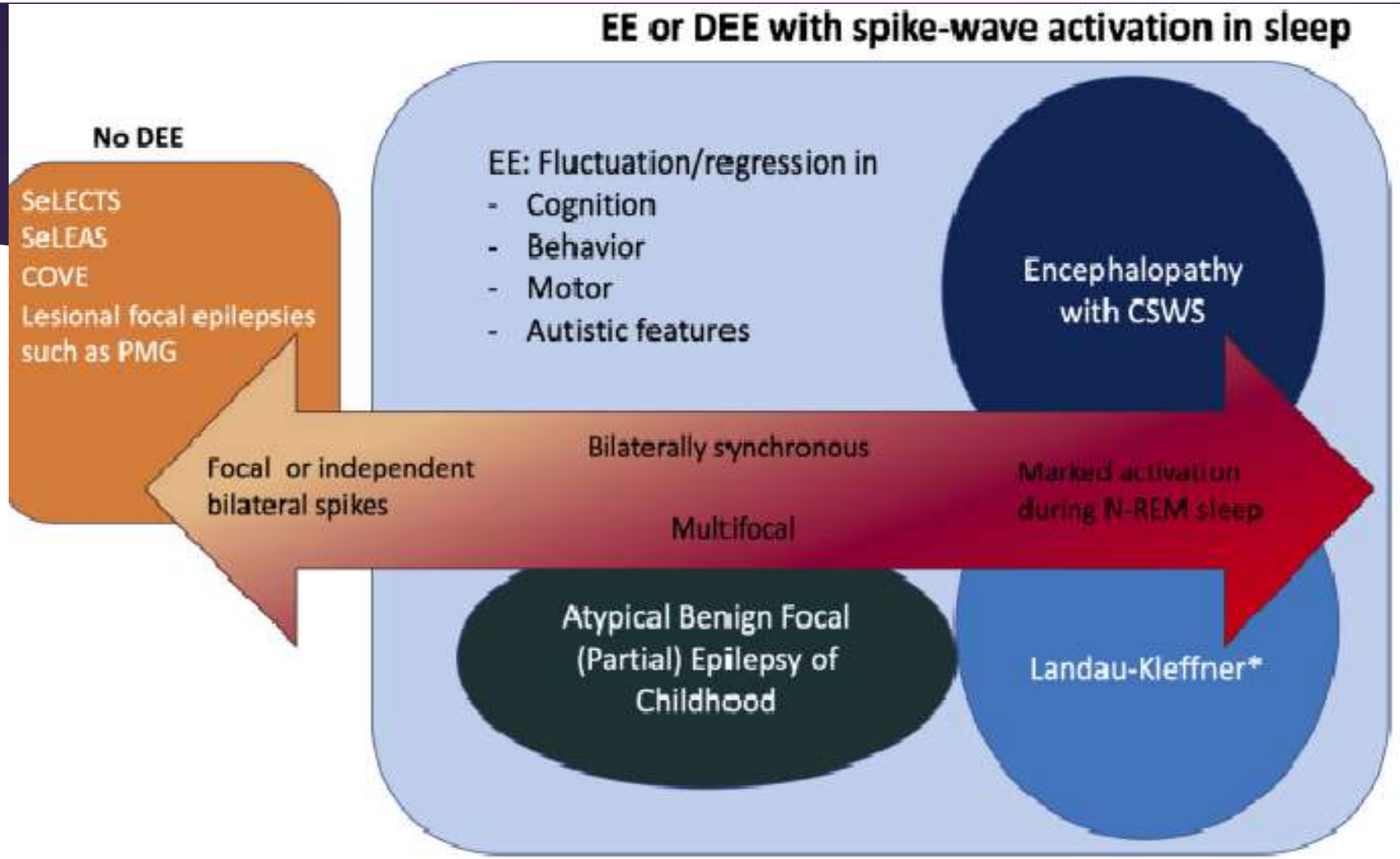
- There may be a family history, with autosomal recessive inheritance in most cases, but PME can be sporadic.



IGURE 8 Developmental and/or epileptic encephalopathy (DEE) and epileptic encephalopathy (EE) with spike-and-wave tivation in sleep (SWAS) refer to a spectrum of conditions that are characterized by various combinations of cognitive, language, havioral, and motor regression associated with marked spike-and-wave activation in sleep. Regression is seen within weeks from the ectroencephalographic pattern. This syndrome is intended to replace syndromes previously named Epileptic Encephalopathy with ntinuous Spike-and-Wave in Sleep and Atypical Benign Partial Epilepsy (pseudo-Lennox syndrome). *Landau–Kleffner syndrome

Febrile infection-related epilepsy syndrome/ FIRES

- occurs most commonly in school-aged children (mean = 8 years) with a typical range of 2–17 years
- History of nonspecific febrile illness in the 2 weeks preceding seizure onset
- Focal and multifocal seizures that often evolve to bilateral tonic–clonic seizures
- Seizures progress in frequency and severity to culminate in superrefractory status epilepticus
- typically within 2 weeks of onset
- Typically normal development and neurological examination before the onset of seizure
- Acute encephalopathy with onset of frequent seizures
- Normal CSF examination(to exclude infection and autoimmune encephalitis)

What to do

- History and Examination
- Rule out disorders that mimic seizures

- Role of neuroimaging (Brain MRI superior to CT scan): tumors, vascular malformation, inflammation, metabolic disease
- EEG
- Metabolic work up
- Genetic studies

Disorders that mimic seizures

- Arrhythmias
  - Long QT and torsades
  - VTach
  - Anytime there is poor cardiac output impairing cerebral perfusion, you can see motor activity / hypoxic convulsions.
- Breath-Holding Spells
  - Actually not associated with inspiratory hold. The child typically screams/cries and exhales fully.
  - They can loose postural tone and have motor activity.
  - Up to 15% will have generalize hypoxic convulsions

Disorders that mimic seizures

- Migraine Syndromes
  - Basilar Migraine
  - Familial Hemiplegic Migraine
- Gastroesophageal reflux may cause generalized stiffness or posturing.
  - Can have apnea also.
  - Often occurs 20-30 min after a meal.
- Dystonic Reactions
  - Always look at the medication list!!

Disorders that mimic seizures, cont.

- Sleep-Related Phenomena
  - Benign sleep myoclonus
  - Periodic sleep jerks
  - Narcolepsy
  - Sleep terrors
- Tics
- Benign paroxysmal vertigo
- pseudoseizure
- Jitteriness in newborn

Recurrence of unprovoked seizures

- First seizure: 20-40% will have recurrence
- Second seizure: up to 80% will have recurrence
- Recurrence usually within the first 6 m, rare after 2 y
- Factors that enhance recurrence in epileptic patient:

- Poor compliance on medication
- Fever
- change in sleep pattern
- Choice of wrong drug

Treatment goals

- Prevent occurrence of seizure
- Prevent or reduce drug S/E, drug interaction
- Improve quality of life
- Provide simple, cost effective care

Modalities of treatment

- AED: old and new
- Ketogenic diet
- Epilepsy surgeries
- Vagal nerve stimulation

Drugs, old

- Phenobarbital
- Phenytoin
- Valproic acid
- Carbamazepine
- Clonazepam
- ethosuxamide

New drugs

- Lamotrigine
- perampanel
- Topiramat
- Levetiracetam
- Zonisamide
- Felbamate
- Gabapentine
- Oxcarbamazepine
- Vegabatrin
- Tigabine

AED- some S/E

- Valproic acid: weight gain, tremor hair loss, hepatitis, thrombocytopenia
- Carbamezapine: bone marrow suppression, steven Johnson, hyponatremia, liver toxicity
- Ethosuxamide: bone marrow suppression, steven Johnson
- Lamotrigine: steven Johnson, liver toxicity
- Topiramat: kidney stones, glaucoma, hyperhidrosis, weight loss
- Levetracitam: behavioral symptoms

AED- some S/E

- Phenobarbital: sedation, hyperactivity, liver toxicity, steven Johnson
- Phenytoin: gingival hyperplasia, liver toxicity, steven Johnson
- vigabatrin: irreversible visual field deficit
- Zonisamide: hyperhidrosis, fever, kidney stones
- Gabapentine: aggression and hyperexcitability
- Oxcarbazepin: somnolence, headache, hyponatremia
- Felbamate: aplastic anemia, hepatic toxicity, rarely used(lennox Gastaut)

Treatment ..

- When to treat
- Which drug to choose:

- depends on type of seizure and epileptic syndrome

-depends on patient age

-depends if presence of other disease (eg. kidney stones avoid topiramate and zonisamide)

- Old or new
- Refractory epilepsy

Treatment ..

- Monotherapy should be used if possible
- Combination therapy should only be used if monotherapy is ineffective
- Consider AED withdrawal after 2 seizure free years. Bear in mind epileptic syndrome (eg. Not in JME) and child and family psychosocial factors.
- AED withdrawal must take place gradually

Surgical treatment of epilepsy

- Resective surgery: removal of the brain parenchyma that is considerd to be the source of seizures; it is an option where

-Focal onset

-radiological evidence

-minimal predicted morbidity

-must only be performed in specialized centers after an extensive evaluation

Surgical treatment of epilepsy

- Corpus callosotomy
- Hemispherectomy
- Vagal nerve stimulation :-pacemaker behind left carotid, transmit intermittent electrical stimulation to an electrode wrapped around the left vagus nerve decrease in seizure frequency 20-30% -adjunctive treatment to AED - S/E: hoarsness of voice, cough, dysphagia, chest/arm pain or paresthesia
- Deep brain stimulation

Ketogenic diet

- High fat 80%, low carbohydrate, adequate protein 20%
- Supplemented with vitamins and calcium
- Needs admission to hospital few days to induce ketosis
- Common s/e: non compliance, acidosis, constipation, failure to gain weight, increase cholesterol
- Successful control of seizure equal that of AED
- Children who become seizure free will continue to be seizure free when diet discontinued
- Treatment of choice in children with Glucose transport(GLUT1)

protein deficiency. Option for LGS. Contraindicated in FAO disorders

Thank you