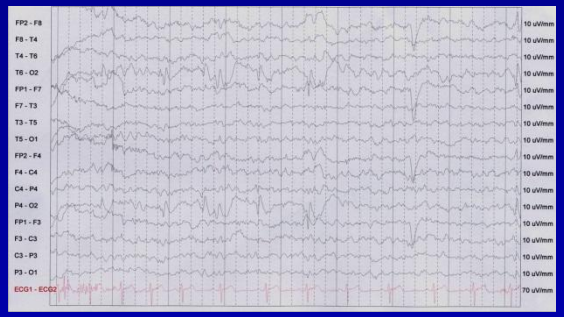
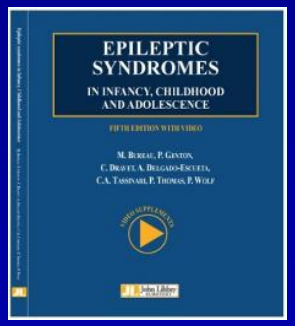
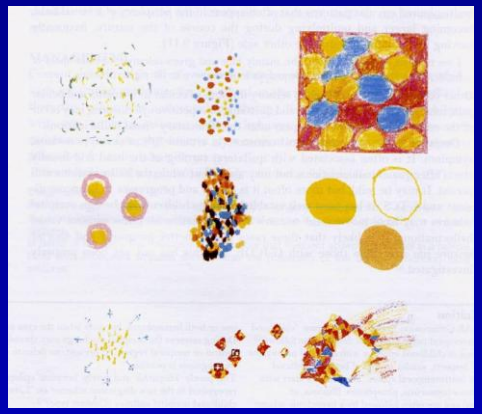






Epilepsy Syndromes

& some new concepts in classification

Sameer Zuberi
Paediatric Neurologist
Royal Hospital for Children
Glasgow

Electro-clinical syndromes

- ***A complex of clinical features, signs and symptoms that together define a distinctive, recognisable clinical disorder.***

- Identifiable on basis of typical age of onset, seizure types, specific EEG characteristics, and other features.

- The diagnosis has implications for treatment, management, and prognosis. They are largely but not exclusively genetic in nature

Do we include imaging, genetics and other aspects of aetiology?

Recognised in ILAE Classifications from 2001

The Blue Book - first published in French 1984 and English 1985

Review the utility and difficulties with the concept?

Do we need to redefine what we mean by epilepsy syndrome?

Have some syndromes outlived their usefulness?

Do we need to re-assess / change the words we use to describe syndromes?

One syndrome may have more than one aetiology

&

a single aetiology may be associated with one syndrome

www.epilepsydiagnosis.org

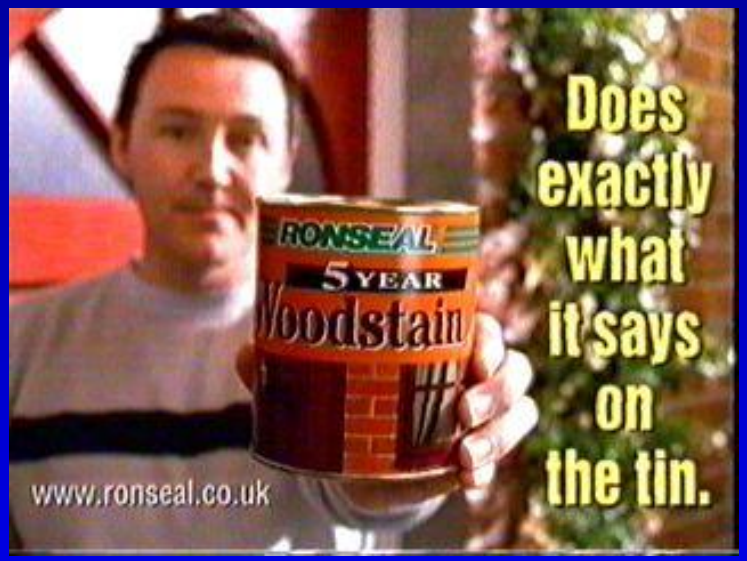
What does the clinician want from a developing classification of the epilepsies?

A scheme that helps in patient management

Provides an aetiological perspective to help make a specific diagnosis

Helps organise the clinician's thoughts

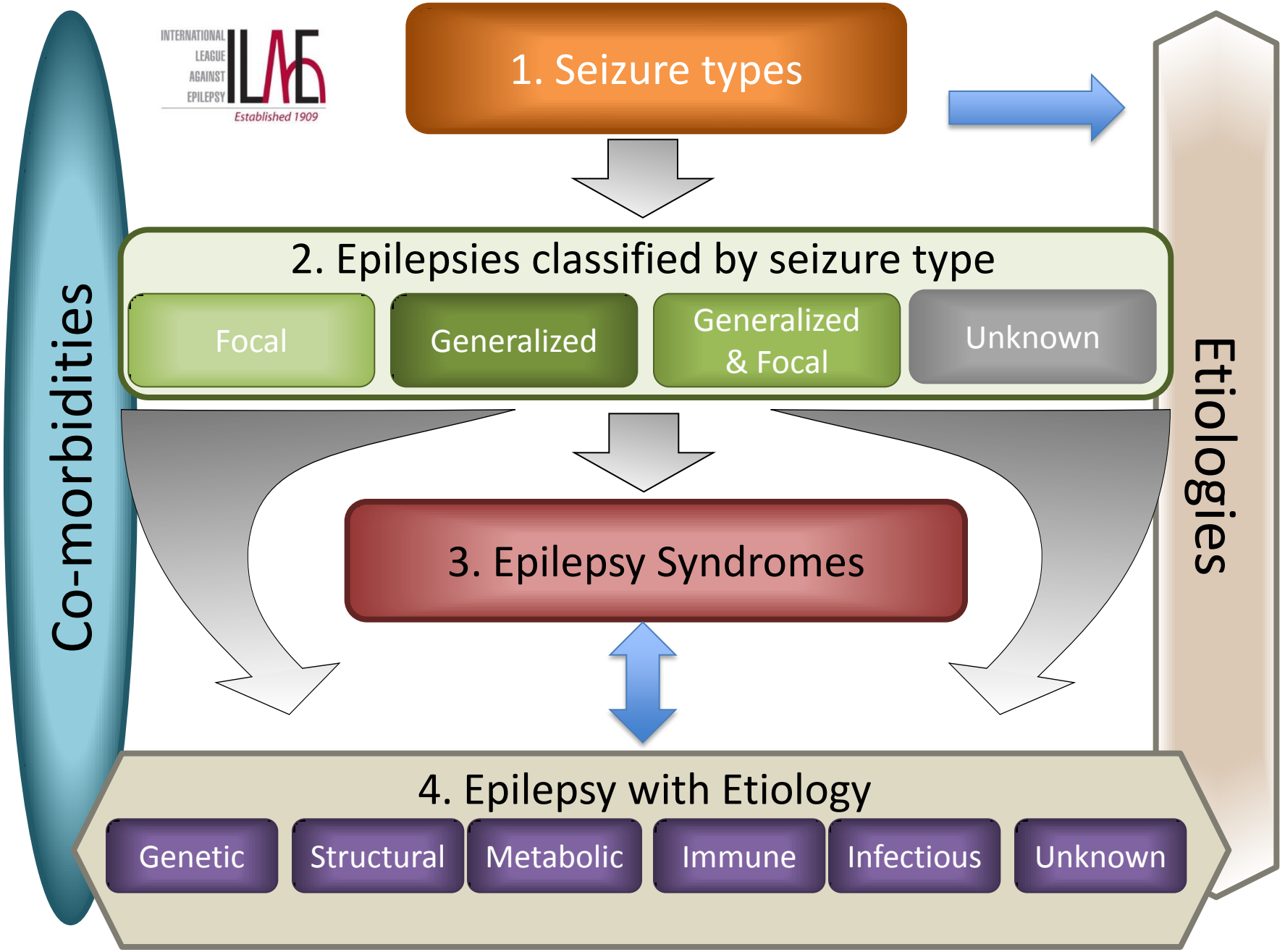
Aids in treatment decisions

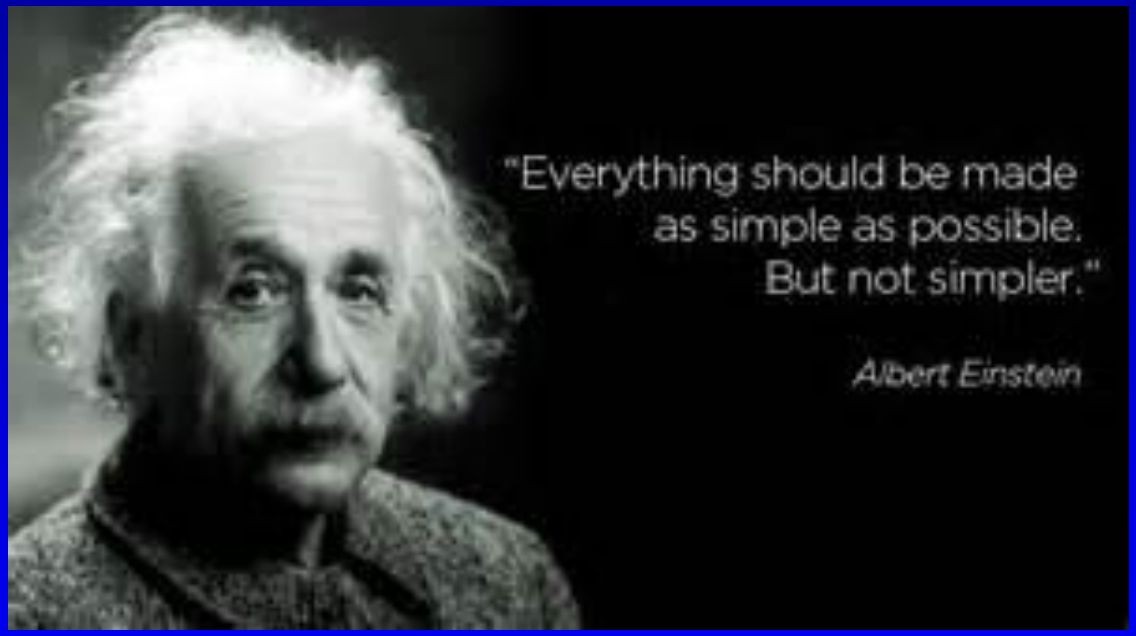


Helps in communication with the patient and family

That makes sense to the general practitioner /physician / paediatrician

Aids research & communication between physicians


INTERNATIONAL LEAGUE AGAINST EPILEPSY
ILAE
Established 1909
1. Seizure types
2. Epilepsies classified by seizure type
Focal
Generalized
Generalized & Focal
Unknown
3. Epilepsy Syndromes
4. Epilepsy with Etiology
Genetic
Structural
Metabolic
Immune
Infectious
Unknown
Co-morbidities
Etiologies


"Everything should be made
as simple as possible.
But not simpler."
Albert Einstein

Words matter

« Mal nommer les choses,

c'est ajouter au malheur du monde »

→ not finding the right words can add to the misfortune of the world

Albert Camus

Electro-clinical syndromes

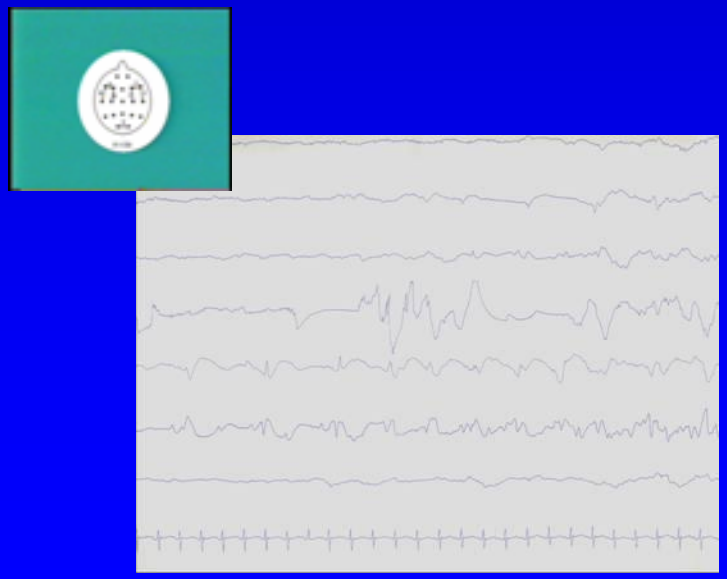
- Neonatal period
- Infancy
- Childhood
- Adolescence – Adult
- Without specific age relationship

- Are these epilepsy syndromes?
  - Mesial temporal lobe epilepsy
  - Rasmussen syndrome
  - Gelastic seizures with hypothalamic hamartoma

Neonatal epilepsy syndromes

- Benign familial neonatal seizures
- Early myoclonic encephalopathy
- Ohtahara syndrome

Benign Familial Neonatal Seizures



- Well neonates until seizures begin on day 2 or 3
- Premature infants have delayed onset until term
- Seizures - tonic, apnoea, clonic, may have focal features, autonomic features
- EEG
  - Interictal - normal or focal/multifocal abnormalities
  - Ictal - diffuse flattening then focal or generalized spikes
- Autosomal dominant, penetrance 85%
- Later Febrile Seizures 5%, epilepsy 11%
- Respond well to carbamazepine

“Benign” Epilepsies

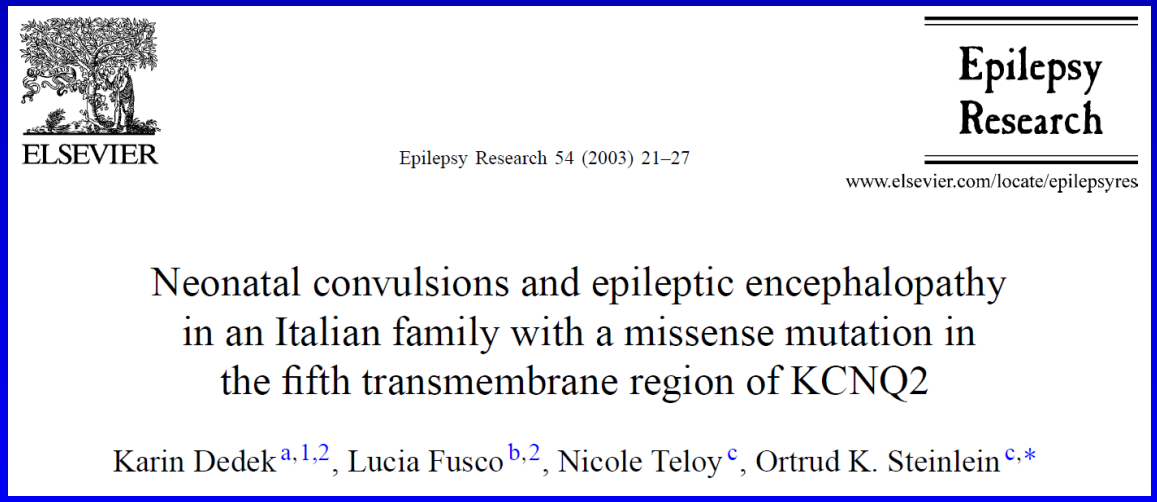
- The seizures are self-limited; they will remit with or without treatment at a predictable age in the vast majority of cases

- The seizures are generally not disabling and are not associated with an epileptic encephalopathy (though mild cognitive impairment can occur)

Do you consider mild cognitive impairment benign?

BFNS with a KCNQ2 mutation

In some families 30% recurrence rate of seizures in later life



ELSEVIER

Epilepsy Research 54 (2003) 21–27

Epilepsy Research

www.elsevier.com/locate/epilepsyres

Neonatal convulsions and epileptic encephalopathy in an Italian family with a missense mutation in the fifth transmembrane region of KCNQ2

Karin Dedek[a,1,2], Lucia Fusco[b,2], Nicole Teloy[c], Ortrud K. Steinlein[c,*]

De novo mutations may be associated with a severe phenotype – Ohtahara syndrome – early onset epileptic and developmental encephalopathy

“Benign” Epilepsies

- The seizures are self-limited; they will remit with or without treatment at a predictable age in the vast majority of cases

- The seizures are generally not disabling and are not associated with an epileptic encephalopathy (though mild cognitive impairment can occur)

Do you consider mild cognitive impairment benign?

“Benign” Epilepsies

- May be associated with
  - significant cognitive problems
  - psychiatric disorders
  - behavioural problems
  - migraine
  - sudden unexpected death can occur in a benign epilepsy (SUDEP)

Is it right to tell a family their child has a benign epilepsy?

Infantile onset syndromes

- Migrating partial seizures of infancy -
- West Syndrome
- Myoclonic epilepsy in infancy
- Benign infantile seizures
- Benign familial infantile seizures
- Dravet Syndrome
- Myoclonic encephalopathy in non-progressive disorders

(Benign) Myoclonic Epilepsy in Infancy

- The prefix benign has been taken out of the latest classification
- Brief epileptic myoclonic attacks with onset 6 months to 3 years
- Infants have normal neurological examination and normal development prior to onset of epilepsy
- 20% infants have history of simple febrile convulsions typically preceding the myoclonias

(Benign) Myoclonic Epilepsy in infancy

- Myoclonic attacks involving upper limb and head, rarely lower limbs. Falls would be atypical.
- Multiple episodes per day.
- May be triggered by noise or startle, surprise is important.
- Series of rhythmic jerks – last 5-10s

What about outcome?

Cognitive outcome

- *Lin et al* –
  - 1/10 cases had moderate mental retardation
  - Mean delay in onset to treatment 2 months
- *Rossi et al* – 5/11 cases had cognitive problems
  - 1 child moderate mental retardation
  - 1 child mild mental retardation
  - 1 child specific learning difficulties
  - 2 children attention deficit / concentration problems
  - Mean delay in onset to treatment 10 months
- *Pratz et al-*
  - 3/7 cases had neuropsychological problems
- Mukhin et al –
  - “High frequency of intellectual disorders”

Dravet Syndrome (Severe Myoclonic Epilepsy of Infancy)

Charlotte Dravet 1978

- Onset in the first year of life with febrile seizures
- Prolonged unilateral or generalized clonic seizures
- Other seizure types evolve by 1- 4 years
  - Myoclonus
  - partial seizures
  - atonic seizures
  - atypical absences
- Hyperthermia often precipitant (bathing, fever)

Genes which have been associated with Dravet Syndrome like phenotypes

SCN1A	**sodium channel neuronal type 1, α subunit**
SCN2A	sodium channel neuronal type 2, α subunit
SCN1B	sodium channel neuronal type 1, β subunit
SCN8A	sodium channel neuronal type 8, α subunit
SCN9A	sodium channel neuronal type 9, α subunit
PCDH19	protocadherin 19
GABRA1	Gamma-amino butyric acid (GABA-A) receptor α1 subunit
GABRB3	Gamma-amino butyric acid (GABA-A) receptor β3 subunit
GABRG2	Gamma-amino butyric acid (GABA-A) receptor γ2 subunit
HCN1	hyperpolarisation-activated, cyclic nucleotide-gated channel
CHD2	chromodomain helicase DNA binding protein 2
STXBP1	syntaxin binding protein 1
KCNA2	Kv1.2 potassium channel
TBC1D24	TBC1 Domain family, member 24 protein

14 of 231 "*epilepsy genes*"

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GABRA1 and *STXBP1*: Novel genetic causes of Dravet syndrome

Neurology® 2014;82:1245-1253

Table 1 Clinical features of patients with Dravet syndrome who underwent whole-exome sequencing or in whom mutations were identified by targeted resequencing

Patient	Age, y/ sex	Seizure onset age (mo), seizure type	Seizure types	Fever sensitivity	Intellect (regression)	EEG	MRI	Likely causative gene	Testing method
T20744	2/F	8, Brief H	FDS, H, SE, TCS	Present	Mild delay (no)	Normal	Normal	*GABRA1*	WES
T16706	7/F	11, Febrile, 20 min TCS	Ab, FDS, H, Myo, TCS	Present	Moderate ID (yes)	GSW	Normal	*GABRA1*	Targeted resequencing
T23532	18/M	11, Febrile, 10 min H	Ab, At, FDS, H, SE, T, TCS	Present	Moderate ID (yes)	Focal discharges	Calcified subependymal nodule in left lateral ventricle	*GABRA1*	Targeted resequencing
Co05	18/M	8, H SE	Ab, At, FDS, H, Myo, SE, TCS	Present	Mild ID (unknown)	GSW, MFD, PPR	Normal	*GABRA1*	Targeted resequencing
T1915	11[a]/M	11, Afebrile cluster of TCS	At, FDS, Myo, SE, T, TCS	Present	Severe ID, deceased aged 11 y (yes)	MFD	Normal	*STXBP1*	WES
EP1807	21/M	6, Febrile FDS	Ab, FDS, Myo, TCS	Present	Severe ID (yes)	MFD	Atrophy	*STXBP1*	Targeted resequencing
T21717	6/F	12, Brief febrile TCS	Ab, At, H, Myo, T, TCS	Present	Learning difficulties (no)	Normal	ND	*STXBP1*	Targeted resequencing
T888	23/F	6, Afebrile H SE	Ab, FDS, H, Myo, SE, TCS	Present	Moderate ID (no)	GSW, PPR	Normal	*SCN1A*	WES
T1895	17[a]/M	11, Febrile SE	aAb, At, FDS, Myo, NCS, SE, TCS	Present	Severe ID, deceased aged 20 (yes)	GSW, PPR, MFD	Normal	*SCN1A*	WES
T17775	7/F	3, 10 min afebrile TCS	Ab, At, FDS, H, Myo, NCS, SE, TCS	Present	Severe ID (yes)	GSW, PSW, MFD	Normal	*SCN1A*	WES
T22809	3/M	6, Febrile SE	Ab, Myo, SE, TCS	Present	Mild ID (yes)	Normal	Normal	*SCN1B*	WES
T20038	10/F	6, Brief febrile TCS	TCS	Present	Mild ID (no)	Normal	Normal	None	WES
T16860	26/M	2, Brief TCS	Ab, At, FDS, Myo, SE, TCS	Present	Mild ID (unknown)	Focal discharges	Cerebellar atrophy	None	WES
T1911	8/M	7, TCS	At, FDS, Myo, T, TCS	Present	Severe ID (yes)	GSW, MFD	Normal	None	WES
T3892	9/M	4, Febrile SE	Myo, FDS, H, SE, TCS	Present	Moderate ID (no)	GSW, PSW, MFD	Normal	None	WES
T863	11/F	6, Ab	Ab, At, H, Myo, NCS, SE, T, TCS	Present	Mild ID (yes)	Normal	Delayed myelination	None	WES
T19264	9/F	14, Febrile TCS	Ab, FDS, H, Myo, SE, TCS	Present	Severe ID (no)	GSW, PPR	Normal	None	WES
T2985	39/M	6, Febrile, 15 min H	At, FDS, H, Myo, SE, TCS	Present	Moderate ID (no)	GSW, MFD	Normal	None	WES

Whole exome sequencing in 13 & targeted resequencing in 67 SCN1A negative individuals with Dravet syndrome – 11 significant findings. Is this really Dravet syndrome?

Epileptic patients with de novo *STXBP1* mutations: Key clinical features based on 24 cases

[1]Chloé Di Meglio, [2]Gaetan Lesca, [1]Nathalie Villeneuve, [3,4,5]Caroline Lacoste, [3,5]Affef Abidi, [3,4,5]Pierre Cacciagli, [6]Cécilia Altuzarra, [7,8]Agathe Roubertie, [9]Alexandra Afenjar, [9]Florence Renaldo-Robin, [10]Bertrand Isidor, [11]Agnes Gautier, [12]Marie Husson, [13]Claude Cances, [14]Julia Metreau, [15]Cécile Laroche, [16]Mondher Chouchane, [17]Dorothée Ville, [17]Stéphanie Marignier, [17]Christelle Rougeot, [18]Marine Lebrun, [19]Anne de Saint Martin, [20]Alexandra Perez, [21]Audrey Riquet, [3,4,5]Catherine Badens, [4]Chantal Missirian, [3,4,5]Nicole Philip, [1]Brigitte Chabrol, [3,5]*Laurent Villard, and [1,3,5]*Mathieu Milh

Epilepsia, **(*):1–10, 2015
doi: 10.1111/epi.13214

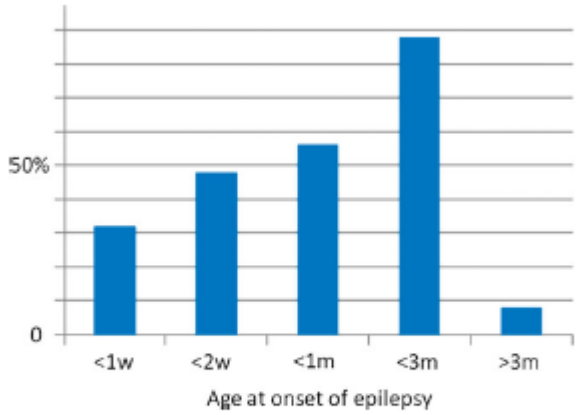

Figure 1.
Cumulative distribution of mutated patients according to age of onset of epilepsy.
Epilepsia © ILAE

Key Points

- We describe 24 patients carrying a mutation of *STXBP1*, from a large cohort of 284 epileptic patients
- *STXBP1* mutations are found in approximately 10% of patient with an early onset epileptic encephalopathy, with a rate of 25% in Ohtahara syndrome
- Half of the mutated patients evolve to West syndrome, but only one of 24 patient was diagnosed with West syndrome from the beginning of the epilepsy
- Epilepsy improves after the first year of life in most mutated patients
- All of them have moderate to severe developmental delay with normal head circumference

Do STXBP!1 encephalopathy cases really have Dravet syndrome?

Genetic Heterogeneity in Dravet Syndrome: the role of *SCN2A*, *SCN1B*, and *GABRG2* variants

Joe Symonds[1], Andreas Brunklaus[2], Sarah Aylett[2], Kunle Ayonrinde[3], PS Baxter[4], Anita Devlin[5], Rachael Ellis[6], Frances Elmslie[7], Lina Nashef[8], Ki Pang[5], Eleanor Reavey[6], Marcus Reuber[9], Sameer Zuberi[1], Mark Rees[10], Seo-Kyung Chung[10]

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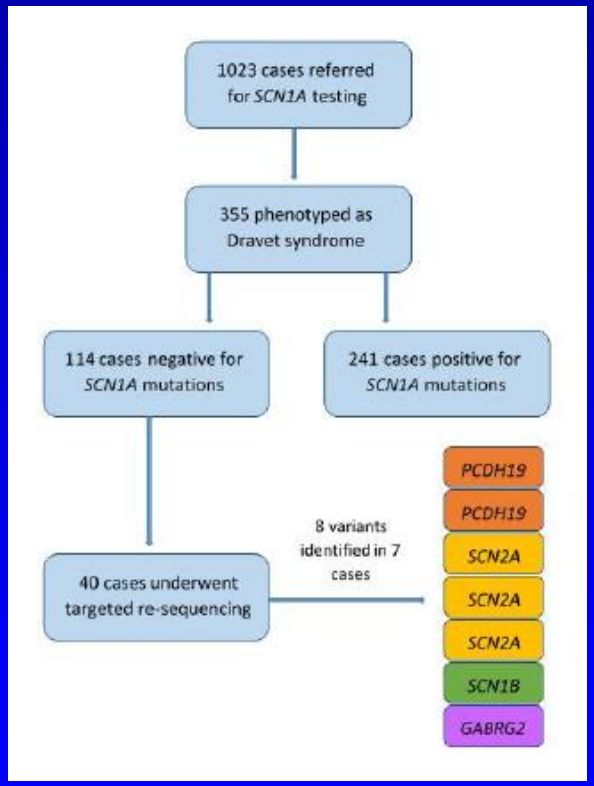
DRAVET Syndrome UK



PCDH19: The 2 cases (reported previously - BPNA 2013 poster) had an initial presentation typical of Dravet syndrome, but further follow-up showed that the epilepsy was characterised by clusters of focal seizures, separated by long seizure-free periods.

SCN2A: The 3 cases had onset of generalised clonic seizures at 9, 12, and 13 months. Atonic or "drop" seizures were seen in all 3. Other seizure types observed were myoclonic seizures (2/3), atypical absences (2/3), tonic seizures (2/3), and focal seizures with impairment of awareness (2/3). Only 1/3 had a febrile seizure (FS) at presentation. All cases demonstrated cognitive decline.

SCN1B: This case presented at 3 months of age with a generalised tonic-clonic seizure, later developing recurrent FS, focal seizures, myoclonic seizures, and atypical absences. There was cognitive decline.

GABRG2: This case presented at 10 months with a simple FS, followed soon after by clusters of FS. Other seizure types included afebrile status epilepticus, recurrent hemiclonic seizures, autonomic seizures, and atypical absences. She was seizure–free from 47 months but was diagnosed with autism.

Immediate suppression of seizure clusters by corticosteroids in PCDH19 female epilepsy

Norimichi Higurashi [a,b], Yukitoshi Takahashi [c], Ayako Kashimada [d], Yuji Sugawara [d], Hiroshi Sakuma [e], Yuko Tomonoh [f], Takahito Inoue [f], Megumi Hoshina [g], Ruri Satomi [h], Masaharu Ohfu [i], Kazuya Itomi [j], Kyoko Takano [k], Tomoko Kirino [l], Shinichi Hirose [b,f,*]

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Seizure 27 (2015) 1–5

Table 1
Details and efficacy of corticosteroid therapy.

Pt no	*PCDH19* mutation	Age at onset (m)	Age at CS TX	CS	Route & dose	Target symptom	Simultaneous TX	Usual duration of Sz cluster	Result	Present intellect
1	p.L719*	13	2y4m	mPSL	IV, 30 mg/kg, 3d	Sz cluster	MDL CBZ CZP VPA LTG LEV	Days ~2 wk	Disappeared after 1st IV	Normal 5y1m
			2y10m	mPSL	IV, 30 mg/kg, 3d	Sz cluster			Disappeared after 1st IV Recurred in 2 wk w/fever[a]	
			2y11m	mPSL	IV, 30 mg/kg, 3d	Sz cluster			Disappeared after 1st IV Recurred in 1wk w/fever	
			3y0m	mPSL	IV, 10 mg/kg, 3d	Sz cluster			Disappeared after 1st IV	
			3y4m	BET	Oral, 0.01 mg/kg, 3d When fever appeared	Sz prevention			No or mild recurrence	
			4y1m	PSL	Oral, 1–1.5 mg/kg, 3d When fever appeared	Sz prevention			No or mild recurrence	
2	p.K120Rfs*3	10	10m	mPSL	IV, 30 mg/kg, 3d	Sz cluster	MDL PB ACV IVIG EDV	–	Disappeared after 1st IV Recurred in 1wk	Moderate delay 3y
3	p.D417H p.D596Y	5	1y11m	mPSL	IV, 20 mg/kg, 2d	Sz cluster	MDL fPHT CLB LEV KBr DZP	Days ~2wk	Disappeared after 1st IV	Normal 2y8m
			2y1m	mPSL	IV, 20 mg/kg, 3d	Sz cluster			Disappeared after 1st IV	
			2y2m	mPSL	IV, 20 mg/kg, 2d	Sz cluster			Disappeared after 1st IV	
			2y5m	mPSL	IV, 10 mg/kg, 1d *fol. by* 20 mg/kg, 1d	Sz cluster			Disappeared after 2nd IV	
			2y7m	mPSL	IV, 20 mg/kg, 1d	Sz cluster			Disappeared after 1st IV Recurred in 9d w/flu	
			2y7m	mPSL	IV, 20 mg/kg, 2d	Sz cluster			Disappeared after 1st IV	
4	p.D596G	6	1y0m	mPSL	IV, 30 mg/kg, 3d	Encephalopatic symptoms	CBZ fPHT LDC PB	1d	Disappeared after 1st IV	Hyperactive 1y6m
5	p.D45Gfs*43	8	11y5m	PSL	IV, 0.35 mg/kg x1 *fol. by* Oral, 1 mg/kg[b]	Sz cluster	KBr CZP	Half a day	Disappeared after 1st IV	Moderate delay 11y8m
			11y6m	PSL	IV, 0.35 mg/kg x1 *fol. by* Oral, 1 mg/kg	Sz cluster			Disappeared after 1st IV Recurred in 1wk w/fever	
			11y6m	PSL	IV, 0.35 mg/kg x1 *fol. by* Oral, 1 mg/kg	Sz cluster			Disappeared after 1st IV	
			11y8m	PSL	IV, 0.35 mg/kg x2 *fol. by* Oral, 1 mg/kg	Sz cluster			Disappeared after 2nd IV	

The phenotypic spectrum of *SCN8A* encephalopathy

Neurology 84 February 3, 2015

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Chipping away at the channels

Can we fashion a syndrome?

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Neurology® 2015;84:446–447

5/17 delayed from birth

Onset birth to 18m

Fever rarely a trigger to seizures (2/17)

7/17 spasms

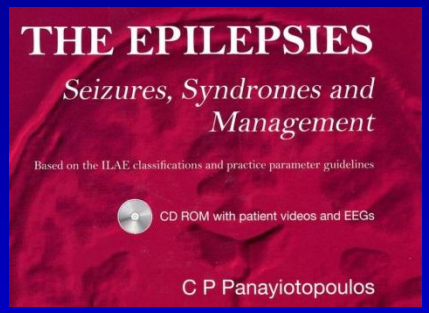
Cortical blindness progresses over time

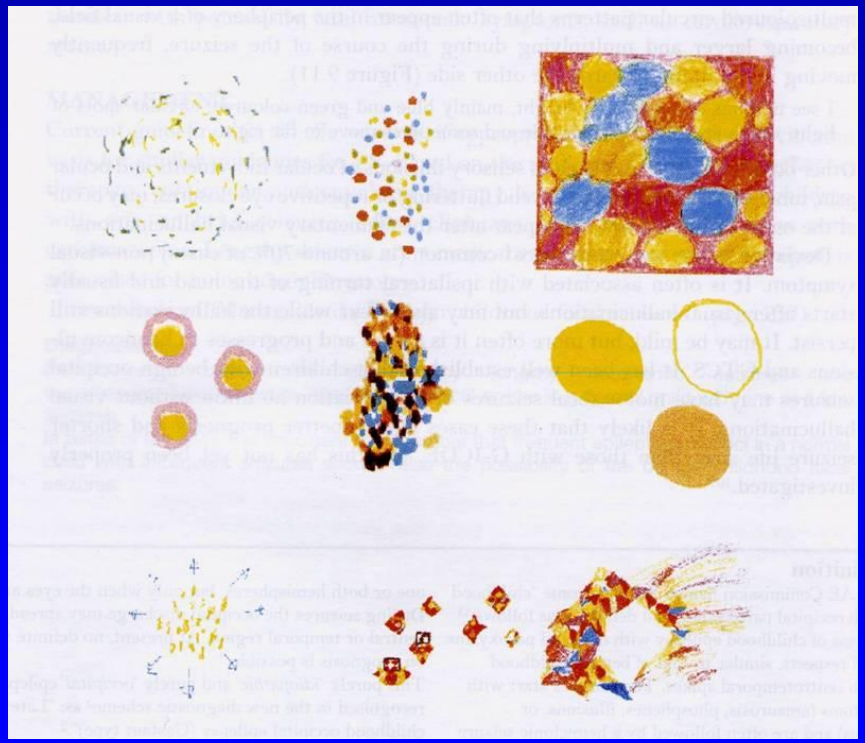
Possible response to sodium channel blockers

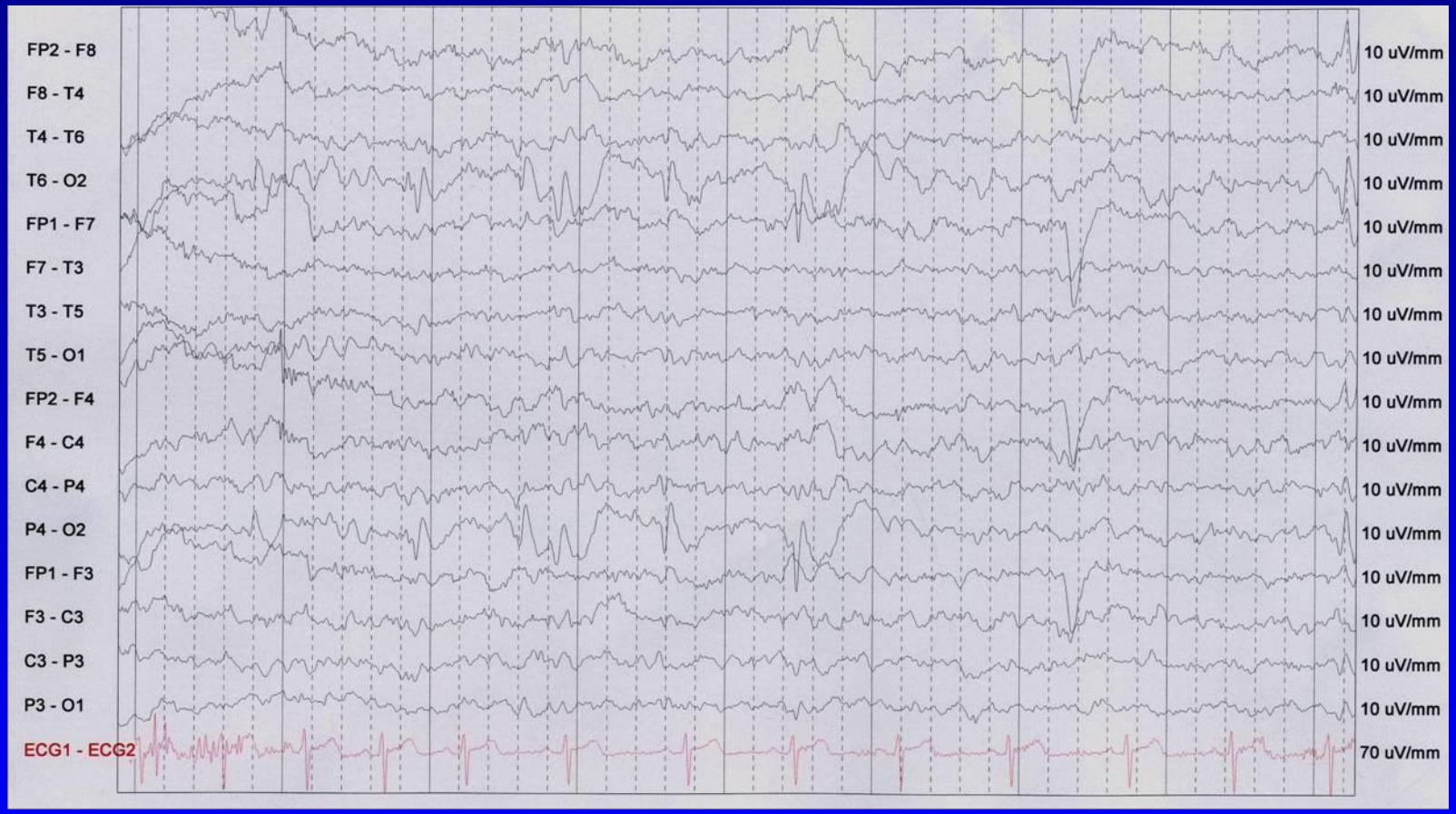
Childhood (benign) occipital epilepsy - Gastaut type

- Onset 3-15y, mean 8y of age
- Elementary visual hallucinations and / or ictal blindness
- 1-3 minutes duration
- Eye and head deviation common
- Fixation off sensitivity on EEG
- MRI is usually done as symptomatic seizures may mimic the benign syndrome
- Carbamazepine usual first line medication

Childhood (benign) occipital epilepsy - Gastaut type







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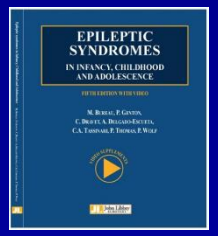


Atypical rolandic epilepsy

"*Seizures typical of BECTS*"

"Atonic or brief inhibitory seizures. Diffuse paroxysms when awake Often associated with atypical absences"

"Frequent almost continuous spike waves in slow wave sleep"

"More or less severe neuropsychological impairment"

"Seizures and EEG abnormalities disappeared before puberty"

Encephalopathy with CSWS

"*Rare nocturnal focal seizures*"

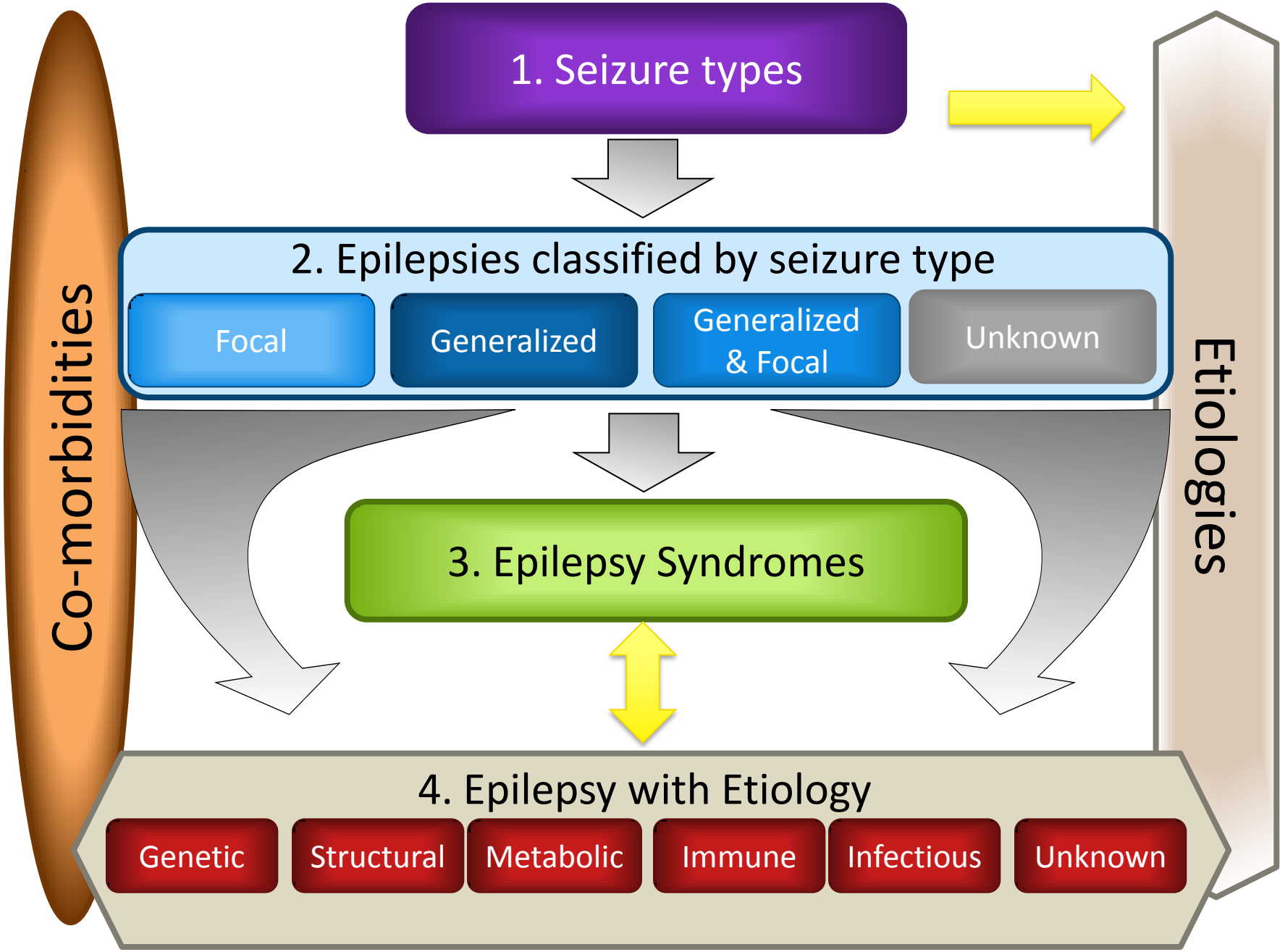
During wakefulness diffuse paroxysms in bursts realising atypical absences often accompanied by inhibitory phenomena."

"Continuous or subcontinuous diffuse spike wakes throughout non REM."

"CSWS accompanied by major neuropsychological impairment"

"Seizures disappeared in all cases"

Described as separate syndromes – are they really distinct?



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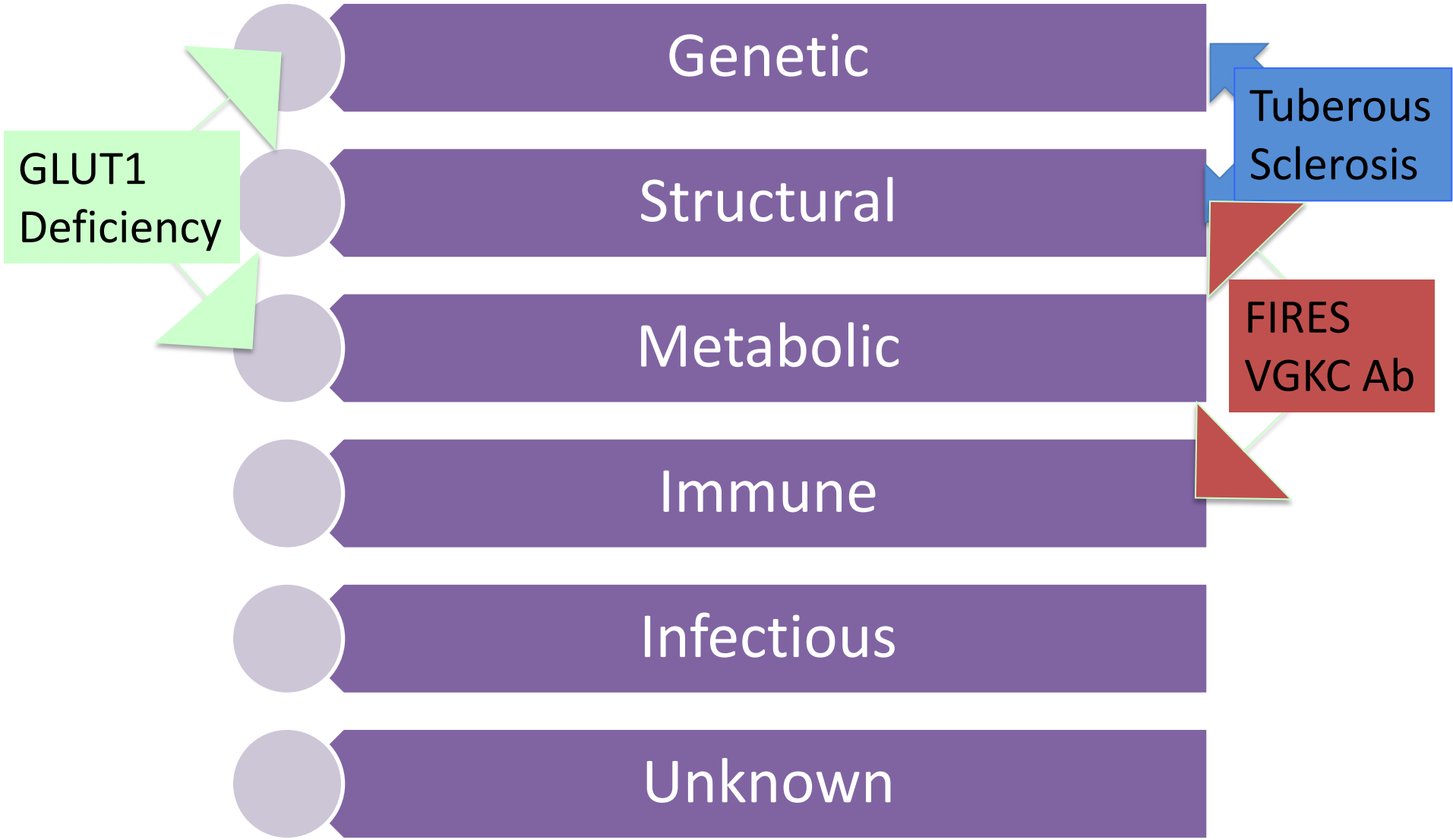
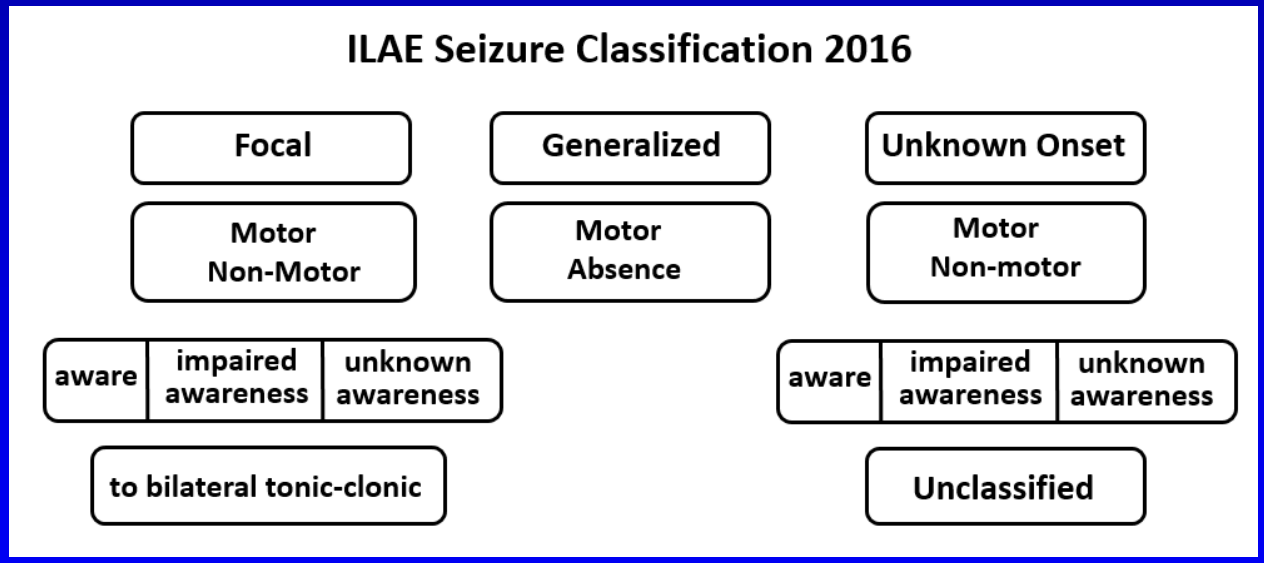
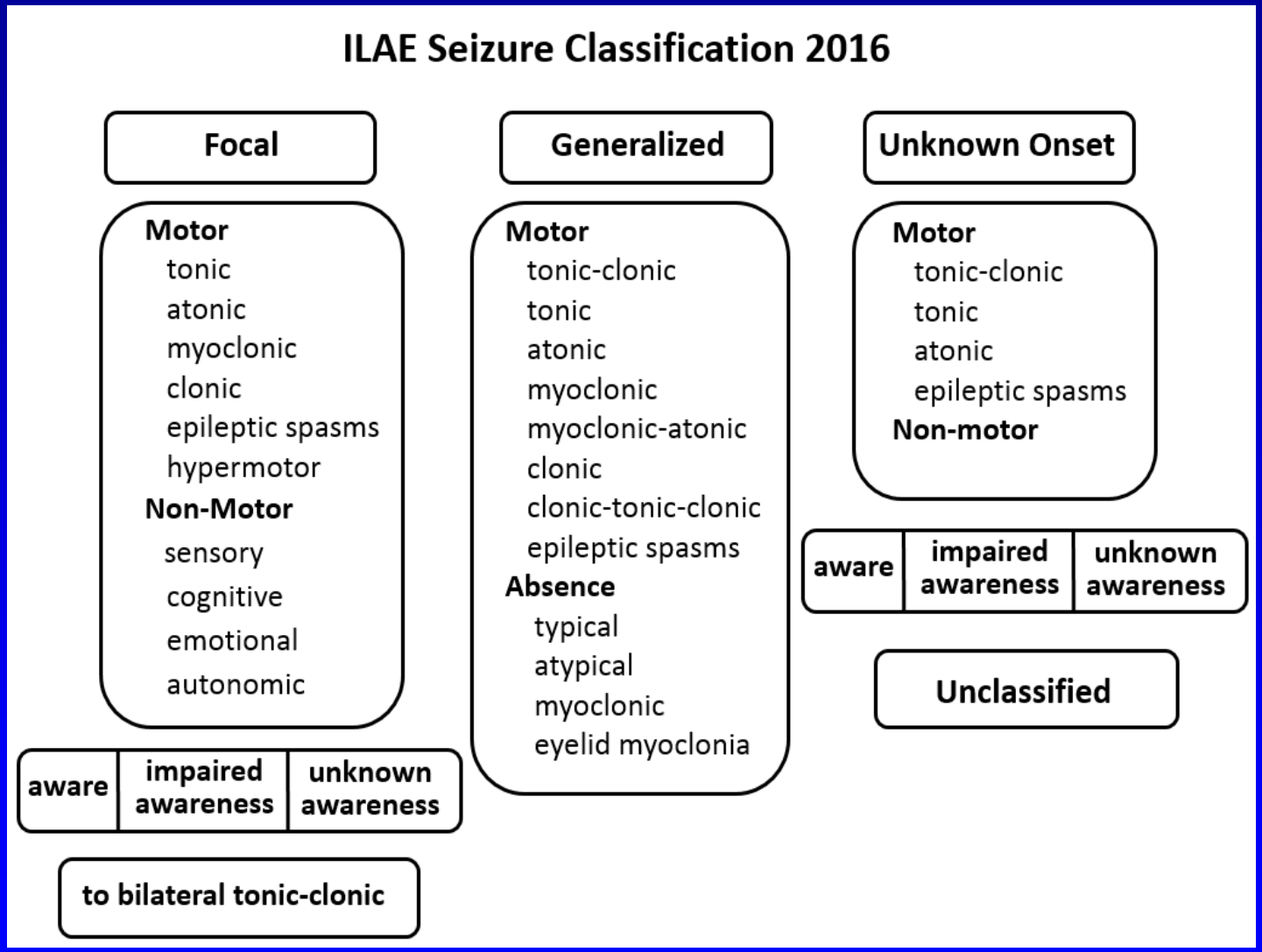
Multiple aetiologies are possible



Classification Seizures - Dec 3 2015.docx




ILAE Seizure Classification 2016
Focal
Motor
tonic
atonic
myoclonic
clonic
epileptic spasms
hypermotor
Non-Motor
sensory
cognitive
emotional
autonomic
aware
impaired awareness
unknown awareness
to bilateral tonic-clonic
Generalized
Motor
tonic-clonic
tonic
atonic
myoclonic
myoclonic-atonic
clonic
clonic-tonic-clonic
epileptic spasms
Absence
typical
atypical
myoclonic
eyelid myoclonia
Unknown Onset
Motor
tonic-clonic
tonic
atonic
epileptic spasms
Non-motor
aware
impaired awareness
unknown awareness
Unclassified

Let it Be?

The Long & Winding Road, B8001, Kintyre, Scotland

The road to Applecross - Wester Ross, Scotland

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