Childhood Epilepsy I
Classification/Syndrome/Approach

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16th July 2010
Outline

- Classification
- Syndromes
- Approach
Morning exercise...please classify
Lumpers and splitters

...debate about lumping together what may or may not be similar forms of epilepsy or splitting apart groups that may represent minor variations of the same form of epilepsy... Implicitly, one must be prepared to split before one can lump. Thus we must always be on guard against unwittingly lumping because we are unaware of certain characteristics on which we should have split.

Anne Berg 2003
The development of a uniform and generally accepted classification of disease is an essential step in the understanding of the underlying processes and in establishing communication through which the results of scientific investigations may be compared and evaluated.
Types of classification

- Clinical – seizures, syndromes, diseases
- Aetiology – genetic, structural, metabolic
- Neurophysiology – localization
- Neuropathology – dysplasia, heterotopia
  - lesional/non-lesional
- Outcome – benign, malignant (epileptic encephalopathy)
Classification of seizures

- Older systems – limited number of seizure types, insufficient
- ILAE – collaboration to standardize terminology and classification
ILAE 1969-1970

- 6 criteria – clinical type, EEG, interictal EEG, anatomic substrate, aetiology, age

- Based on dichotomy – generalized vs partial
Partial – originate in a localized area of one hemisphere
  - simple / complex

Generalized – arise from both hemisphere simultaneously
  - convulsive / non-convulsive

Unclassified
Dichotomy of partial vs generalized:
- not based on undisputable neurophysiological basis
- not always easy to distinguish – clinical or EEG
- origin often difficult to determine
Proposal for Revised Clinical and Electroencephalographic Classification of Epileptic Seizures

From the Commission on Classification and Terminology of the International League Against Epilepsy*

In 1969, the International League Against Epilepsy published a scheme for classification of epileptic seizures. Professor H. Gastaut, then Secretary-General of ILAE and a member of the Commission on Classification, related the history of the work which represents a milestone in efforts at classifying epileptic seizures and has led to Merlis, Dr. D. David Daly of Dallas, Dr. Dieter Janz of Berlin, Dr. J. Kiffin Penry of Bethesda, Dr. Carlo Alberto Tassinari of Marseille. In addition, Dr. Rudolph Dreyer of Bethel, Dr. Antonio V. Escueta of Los Angeles, Dr. K. F. Masuhr of Berlin, Dr. Richard H. Mattson of New Haven, Dr. Roger J. Porter of Bethesda, Dr. Dieter
Dichotomy generalized vs partial maintained

2 features used - clinical features
- EEG features

Partial – simple – without disturbances of consciousness
- complex – with disturbances of consciousness
### Table 1 1981 ILAE Seizure Classification

I. Partial (focal, local) seizures
   A. Simple partial seizures (consciousness not impaired)
      1. With motor signs
         a. Focal motor without march
         b. Focal motor with march (Jacksonian)
         c. Verbal
         d. Postural
         e. Phonatory (localization or arrest of speech)
      2. With somatosensory or special-sensory symptoms (simple hallucinations, eg, tingling, light flashes, and buzzing)
         a. Somatosensory
         b. Visual
         c. Auditory
         d. Olfactory
         e. Gustatory
         f. Vertiginous
   3. With autonomic symptoms or signs (including epigastric sensation, pallor, sweating, flushing, piloerection, and papillary dilatation)
   4. With psychic symptoms (disturbance of higher cerebral function). These symptoms rarely occur without impairment of consciousness and are much more commonly experienced as complex partial seizures
      a. Dysphasic
      b. Dysnomoeic (eg, deja vu)
      c. Cognitive (eg, dreamy states and distortions of time sense)
      d. Affective (fear, anger, and so on)
      e. Illusions (eg, macroopsia)
      f. Structured hallucinations (eg, music and scenes)

B. Complex partial seizures (with impairment of consciousness; may sometimes begin with simple symptomatology)
   1. Simple partial onset followed by impairment of consciousness
      a. With simple partial features followed by impaired consciousness
      b. With automatism
   C. Partial seizures evolving to secondarily generalized seizures (this may be generalized tonic-clonic, tonic or clonic)
      1. Simple partial seizures evolving to generalized seizures
      2. Complex partial seizures evolving to generalized seizures
      3. Simple partial seizures evolving to complex partial seizures evolving to generalized seizures

II. Generalized seizures (convulsive or nonconvulsive)
   A. Absence seizures
      1. Impairment of consciousness only
      2. With mild clonic components
      3. With atonic components
      4. With tonic components
      5. With automatism
      6. With autonomic components (b through f may be used alone or in combination)
      2. Atypical absence may have:
         a. Changes in tone that are more pronounced than in A.1
         b. Onset and/or cessation that is not abrupt
   B. Myoclonic seizures (myoclonic jerks, single or multiple)
   C. Clonic seizures
   D. Tonic seizures
   E. Tone-clonic seizures
   F. Atonic seizures (astatic) (Combinations of the above may occur, for example, B and F, Band D)

III. Unclassified epileptic seizures: Includes all seizures that cannot be classified because of inadequate or incomplete data and some that defy classification in hitherto described categories. This includes neonatal seizures (eg, rhythmic eye-movements, chewing, and swimming movements)
Proposal for Revised Classification of Epilepsies and Epileptic Syndromes

Commission on Classification and Terminology of the International League Against Epilepsy

Preface

Since the Proposal for Classification of Epilepsies and Epileptic Syndromes was presented to the General Assembly of the International League Against Epilepsy (ILAE) in 1985, the Commission on Classification and Terminology of the ILAE has refined and revised the Proposal in light of findings and suggestions emanating from experience in use of the 1985 schema.

The purpose of the International Classification of Epilepsies and Epileptic Syndromes (ICE) is to support records and communication between collaborators. An epileptic syndrome is an epileptic disorder characterized by a cluster of signs and symptoms customarily occurring together; these include such items as type of seizure, etiology, anatomy, precipitating factors, age of onset, severity, chronicity, diurnal and circadian cycling, and sometimes prognosis. Tradistinction to a disease, a syndrome does not necessarily have a common etiology and prognosis. On the other hand, some of the epileptic disorders...
ILAE Classification of Epilepsies and Epileptic Syndromes, 1989

- 2 sets of criteria
  - topographic – localization-related vs generalized syndromes
  - aetiologic – symptomatic vs idiopathic (cryptogenic)
- Idiopathic – not secondary to other d/s but may be genetically determined
- Symptomatic – due to known brain disorder
- Cryptogenic – hidden cause, not detectable by current method
<table>
<thead>
<tr>
<th>Localization-related (focal, local partial) epilepsies and epileptic syndromes</th>
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<tbody>
<tr>
<td>Idiopathic (with age-related onset)</td>
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<tr>
<td>Benign childhood epilepsy with centrotemporal spikes</td>
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<td>Childhood epilepsy with occipital paroxysms</td>
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<td>Primary reading epilepsy</td>
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<td>Symptomatic</td>
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<td>Chronic progressive epilepsy partialis continua of childhood (Kojewnikow syndrome)</td>
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<td>Syndromes characterized by seizures with specific modes of precipitation (include partial seizures after acquired lesions, usually involving tactile or proprioceptive stimuli; partial seizures precipitated by sudden arousal or startle epilepsy)</td>
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<tr>
<td>Temporal lobe epilepsy</td>
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<td>Frontal lobe epilepsies</td>
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<td>Occipital lobe epilepsies</td>
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<td>Cryptogenic</td>
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<tr>
<th>Generalized epilepsies and syndromes</th>
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<tr>
<td>Idiopathic (with age-related onset)</td>
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<tr>
<td>Benign neonatal familial convulsions</td>
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<td>Benign myoclonic epilepsy in infancy</td>
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<td>Childhood absence epilepsy (pyknolepsy)</td>
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<td>Juvenile absence epilepsy</td>
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<td>Juvenile myoclonic epilepsy</td>
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<td>Epilepsy with grand mal seizures upon awakening</td>
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<tr>
<td>Other generalized epilepsies (not defined above)</td>
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<td>Epilepsies with seizures precipitated by specific modes of activation</td>
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<td>Cryptogenic or symptomatic</td>
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<td>West syndrome (infantile spasms, Blitz-Nick-Salaam Krämpfe)</td>
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<td>Lennox-Gastaut syndrome</td>
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<td>Epilepsy with myoclonic-astatic seizures</td>
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<td>Epilepsy with myoclonic absences</td>
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<td>Symptomatic</td>
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<tr>
<td>Nonspecific etiology</td>
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<tr>
<td>Early myoclonic encephalopathy</td>
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<tr>
<td>Early infantile epileptic encephalopathy with suppression-burst electroencephalogram other symptomatic generalized epilepsies not defined above</td>
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<tr>
<td>Specific syndromes (including diseases in which seizures are a presenting or predominant feature)</td>
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<tr>
<td>Epilepsies and epileptic syndromes undetermined whether focal or generalized</td>
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<td>With both generalized and focal seizures</td>
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<td>Neonatal seizures</td>
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<td>Severe myoclonic epilepsy in infancy</td>
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<td>Epilepsy with continuous spike-waves during slow-wave sleep</td>
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<td>Acquired epileptic aphasia (Landau-Kleffner syndrome)</td>
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<tr>
<td>Other undetermined epilepsies not defined above</td>
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<tr>
<td>Without unequivocal generalized or focal features</td>
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<td>Special syndromes: situation-related seizures</td>
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<td>Febrile convulsions</td>
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<td>Isolated seizures or isolated status epilepticus</td>
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<td>Seizures occurring only when there is an acute metabolic or toxic event</td>
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Modified with permission.19

Troester M, Rekate HL
Semin Pediatr Neurol
2009; 16 : 16-22
Epileptic syndromes

Age dependency, eg:

Ohtahara → West → Lennox Gastaut syndrome

→ symptomatic frontal lobe epilepsy
   (cortical dysplasia)
Age-dependency

- LKS, CSWS
- MAE
- LGS
- Dravet, WS
- MPSI
- Ohtahara, EME

Age
ILAE Classification of Epilepsies and Epileptic Syndromes, 1989

- Classification of epilepsies - more difficult than seizures
- Multiple classification system possible
- To be accessible and universally used, lower level ie seizure, syndrome
ILAE Commission Report

A Proposed Diagnostic Scheme for People with Epileptic Seizures and with Epilepsy: Report of the ILAE Task Force on Classification and Terminology
ILAE Proposed Diagnostic Scheme 2001

- List of seizure type, syndromes and diseases rather than dichotomistic classification
- Focal replaces partial/localization related
- SPS/CPS – no longer recommended
- ‘Convulsion’ – omitted
- Probably symptomatic replaces cryptogenic
- Epileptic encephalopathy and benign epilepsy introduced
- Reflex epilepsy introduced
ILAE Proposed Diagnostic Scheme 2001

- Diagnostic scheme rather than fixed classification

5 Axes

Axis 1: Ictal phenomenology
Axis 2: Seizure type
Axis 3: Syndrome
Axis 4: Aetiology
Axis 5: Impairment (optional)
Epileptic encephalopathy

Epileptiform abnormalities themselves are believed to contribute to the progressive disturbance in cerebral function

Ohtahara syndrome
West syndrome
Dravet syndrome
Lennox Gastaut syndrome
Landau Kleffner syndrome
CSWS
Revised terminology and concepts for organization of the epilepsies: Report of the Commission on Classification and Terminology

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Ingrid E. Scheffer, MBBS, PhD, FRACP
2009

- Syndromes: no longer generalized or focal (dichotomy remains useful for seizures)

- Syndromes – restricted to entity reliably identified by a cluster of electro-clinical features

- Does not fit criteria – describe with respect to clinically relevant factors

- Aetiology – grouped as genetic, structural, metabolic, unknown

- Idiopathic/symptomatic abandoned
Problems of classification

- No single all encompassing system to cover all aspects of epilepsy
- Limited knowledge of mechanism of epilepsy
- Empiric classification – limited to a few aspects of the problem, therefore remain at specific level (seizures and syndromes)
Simple classification does not do justice to complexity of epilepsy

Future classification: flexible, multidimensional catalog of key features
Outline

- Classification
- Syndromes - benign/idiopathic
  - epileptic encephalopathy
- Approach
Epileptic Syndromes

- **Idiopathic**
  - Generalized
    - **Infantile onset:**
      - Benign neonatal convulsions
      - Benign neonatal familial convulsions
      - Benign myoclonic epilepsy
    - **Childhood onset:**
      - Childhood absence epilepsy
      - Myoclonic astatic epilepsy
      - Epilepsy with myoclonic absences
      - Generalized epilepsy with febrile seizures plus
    - **Adolescent onset:**
      - Juvenile myoclonic epilepsy
      - Juvenile absence epilepsy
      - Epilepsy with grand mal on awakening
  - Localization-related
    - **Infantile onset:**
      - Benign partial epilepsy in infancy
      - Benign infantile familial convulsions
    - **Childhood onset:**
      - Benign occipital epilepsy—early onset
      - Benign childhood epilepsy with centrotemporal spikes
      - Benign occipital epilepsy—late onset
      - Autosomal dominant frontal lobe epilepsy
      - Familial temporal lobe epilepsy

- **Symptomatic**
  - **Infantile onset:**
    - Early myoclonic encephalopathy
    - Early infantile epileptic encephalopathy
    - Malignant migrating partial seizures
    - West syndrome
    - Dravet syndrome
  - **Childhood onset:**
    - Lennox-Gastaut syndrome
    - Landau-Kleffner syndrome
    - Continuous spike-wave in sleep
    - Rasmussen encephalitis
    - Devastating encephalopathy in school-aged children
  - **Varying ages:**
    - Progressive myoclonic epilepsy
Benign childhood epilepsy with centrotemporal spikes

- Commonest idiopathic focal epilepsy in childhood
- Male, 2-13y (median 7y)
- Simple focal onset - sensory symptoms: tongue, lips, cheek, followed by hemifacial twitching, speech arrest, drooling, and preservation of consciousness, ± secondary generalization
- Occur during sleep 2/3 cases
Benign childhood epilepsy with centrotemporal spikes

- Low seizure frequency, 1 sz: 10-13%
- Most seizure free after 5 years of onset
- 95% seizure free by age of 14
- However – language, behavioural and cognitive problem
- No treatment or excellent response to VPA/CBZ
West Syndrome

...The first sign of any problem with James was at the age of 4 months when he showed strange casting of the eyes towards the ceiling for several days, twice or thrice each day...... slight bobbings of the head forward..... increased in frequency, and at length became so frequent and powerful, as to cause a complete heaving of the head forward towards his knees, and then immediately relaxing into the upright position...... bowings and relaxings would be repeated alternately at intervals of a few seconds, and repeated from 10 to 20 or more times at each attack, which attack would not continue more than 2 or 3 minutes.....

William West 1841
West Syndrome

- Cluster of spasms - symmetrical/ asymmetrical
  - hemispasm
  - flexor / extensor
- Onset 3-7 m
- Developmental arrest/regression
- EEG : Hypsarrhythmia (interictal)
  Diffuse fast activity, then high voltage positive followed electro-decremental response
- 75-80% - mental retardation
  - autistic spectrum disorder
  - continuing seizure
Epileptic spasm

Muscular contraction lasting 1-2s, then decreases equal duration

- Slower than myoclonic jerk
- More rapid than tonic seizure

myoclonic jerk           spasm               tonic spasm
(rhombos/tadpole)
Dravet syndrome

Dravet (1982) – severe myoclonic epilepsy

Severe myoclonic epilepsy of infants

Myoclonic seizures may be absent

2001 – Dravet syndrome
Dravet syndrome

2m – 1y
- complex febrile seizures
- normal development and EEG

1y +
- polymorphic seizures, hemiclonic (switching sides)
- abnormal EEG – NCSE/CSE
- developmental arrest
- ataxia
Dravet syndrome

Family history of FC or epilepsy > 25%
Fever sensitivity
Photo and pattern sensitivity

In infant with recurrent prolonged febrile convulsions, think of DS…diagnosis not sure until appearance of
- other seizure types: myoclonias, focal, atypical absences
- psychomotor delay
- photosensitivity
Dravet syndrome

‘channelepsy’ – alpha-1 subunit of sodium channel

Mutation *SCN1A* gene in 80%
Mutation *SCN2A*, *GABRG2* in others

Spectrum

GEFS+\(\overset{\text{missense mutation}}{\leftrightarrow}\) DS\(\overset{\text{truncation mutation}}{\leftrightarrow}\)
GEFS+

- Heterogenous epilepsy phenotypes within families
- Diagnosis in a family (vs FS+ diagnosis in an individual)

FS
FS/FS+
FS/FS+ and absences
FS/FS+ and myoclonic seizures
FS/FS+ and atonic seizures
FS/FS+ and partial epilepsy
Myoclonic astatic epilepsy
Dravet syndrome

Only 20% has mutation: 
SCN1A
SCN1B
SCN2A
GABRG2
<table>
<thead>
<tr>
<th>Gene</th>
<th>Syndrome</th>
<th>Phenotype</th>
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<tbody>
<tr>
<td>SCN1A</td>
<td>Dravet synd</td>
<td>FS, FS+, MAE, BFIS</td>
</tr>
<tr>
<td>SCN1B</td>
<td>GEFS+</td>
<td>MAE, Focal epilepsy</td>
</tr>
<tr>
<td>SCN2A</td>
<td>GEFS+</td>
<td>MAE, Focal epilepsy</td>
</tr>
<tr>
<td>GABRG2</td>
<td>BFIS</td>
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De-novo mutations of the sodium channel gene SCN1A in alleged vaccine encephalopathy: a retrospective study

Samuel F Berkovic, Louise Harkin, Jacinta M McMahon, James T Pelekanos, Sameer M Zuberi, Elaine C Wirrell, Deepak S Gill, Xenia Iona, John C Mulley, Ingrid E Scheffer

Summary

Background Vaccination, particularly for pertussis, has been implicated as a direct cause of an encephalopathy with refractory seizures and intellectual impairment. We postulated that cases of so-called vaccine encephalopathy could have mutations in the neuronal sodium channel α1 subunit gene (SCN1A) because of a clinical resemblance to severe myoclonic epilepsy of infancy (SMEI) for which such mutations have been identified.

Methods We retrospectively studied 14 patients with alleged vaccine encephalopathy in whom the first seizure occurred within 72 h of vaccination. We reviewed the relation to vaccination from source records and assessed the specific epilepsy phenotype. Mutations in SCN1A were identified by PCR amplification and denaturing high performance liquid chromatography analysis, with subsequent sequencing. Parental DNA was examined to ascertain the origin of the mutation.
SCN 1A and Vaccine encephalopathy

Retrospective study (part of larger study of unexplained encephalopathy, N = 96)
First seizure within 72h of vaccination (n=14)

11/14 mutation SCN1A
5 - truncation
6 - missense

All had specific epilepsy syndrome diagnosis
12/14 – SMEI/SMEB
2/14 – Lennox-Gastaut (no mutation)
Outline

- Classification
- Syndromes
- Approach
Diagnosis

History, history, history!

*JB Stephenson ("Fits and Faints")*

Need to distinguish acute symptomatic seizures from epilepsy
The commonest reason for mismanagement of epilepsy is related to the diagnosis and classification of the epilepsy.

Eg absence epilepsy diagnosed erroneously as focal epilepsy and treated with carbamazepine may precipitate absence status epilepticus.
All you need to know….

- Is it a seizure?
- What is the seizure type?
- Is it epilepsy?
- What is the epileptic syndrome?
- What is the aetiology?
- What are the co-morbidities/impairment?
History

Antenatal

- seizures in utero – “increased foetal movement”
- infection

Birth history - AS, asphyxia

Neonatal history

Developmental history

Seizure – onset, “before, during, after” – home video

Family pedigree

Other associated problems
Clues to focal seizure

- Nocturnal
- Headache, vision disturbances
- Aura ( = simple focal seizure)
- Ictal - version - eye, head
  - asymmetric tonic/clonic
  - posturing/dystonic
- Post ictal - automatism
  - nose rubbing
  - headache, vision
Examination

Dysmorphism
Head circumference
Neurocutaneous stigmata
Hair and nail
Eyes
Organomegaly
Developmental milestones
EEG

Awake and sleep recording

Natural sleep preferred – written and verbal instructions
Sedated sleep – fast beta activity interferes interpretation

Activation
sleep – increase yield from 60% to 90%
hyperventilation
photic stimulation – slow stimulation 1-3Hz
Idiopathic

85%

Symptomatic

Vascular disease
Tumour
MTS
Trauma
Infection

ADULT

15%

CD

CHILDREN

45%

Trauma
Infection
Metabolic
Genetic
Structural

55%
Neuroimaging

All except:

- myoclonic epilepsy of infancy
- juvenile myoclonic epilepsy
- childhood absence epilepsy
- juvenile absence epilepsy
- BECTS
Neuroimaging

- Epilepsy protocol – thin slices T1, T2, flair in 3 planes

- First 6 months – T1- maturational changes
  T2- cortical abnormalities

- After 6 month – reverse
  - FLAIR – eliminate CSF distortion
Idiopathic epilepsy: single gene defect

- **IGE**
  - 5q  \( \text{GABA}_A\gamma 2 \) subunit: GABRG2
  - 5q  \( \text{GABA}_A\alpha 1 \) subunit: GABRG1
  - 3q  voltage gated Cl channel: CLCN2

- **GEFS+**
  - 19q  Na channel \( \alpha 1 \) subunit: SCN1A
  - 2q  Na channel \( \beta 1 \) subunit: SCN1B
  - 2q  Na channel \( \alpha 2 \) subunit: SCN2A
  - 5q  \( \text{GABA}_A\gamma 2 \) subunit: GABRG2

- **Benign familial neonatal seizures**
  - 20q  K channel: KCNQ2
  - 8q   K channel: KCNQ3

- **Benign familial infantile seizures**
  - 2q   Na channel \( \alpha 2 \) subunit: SCN1A
Chromosome and epilepsy

Wolf-Hirschhorn syndrome           4p-
Miller-Dieker syndrome              del 17p13.3
Angelman syndrome                  del 15q11-q13
Inversion duplication 15 syndrome
Terminal deletion 1q
Terminal deletion 1p
*Ring chromosome 14
*Ring chromosome 20

*subtle dysmorphism, recurrent status epilepticus, may miss on karyotyping
Genetic investigation

2-3% of all epilepsy

Conventional karyotyping *may* miss diagnosis (microdeletion, subtelomeric deletion, ring chromosome)

Other techniques:
- Methylation PCR
- FISH
- DNA sequencing
Metabolic investigation

- Increasing recognition of metabolic cause
- Some potentially treatable:

  - Pyridoxine dependent epilepsy
  - Folinic acid deficiency
  - Biotinidase deficiency
  - Mitochondrial disorder
  - GLUT 1 transporter defect
  - Neurotransmitter disease
Metabolic – when to investigate

Unexplained refractory seizure
Unexplained white matter changes (MRI brain)
Early myoclonic epilepsy of infancy
Progressive myoclonic epilepsy
Neuroregression
Other unexplained findings – eg optic atrophy, retinitis pigmentosa, multiorgan involvement
Consider co-morbidities

- Cerebral palsy - GORD, oromotor dysfunction
- Failure to thrive – appetite
- Obesity
- Learning difficulty, mental retardation
- Hyperactivity
- Autism
- Migraine
- Mitochondrial disorder – avoid PB, PHT, VPA
Conclusions

- Classification: evolving, to incorporate recent advances

- Epilepsy syndrome remains useful concept

- Reliable history cornerstone to accurate diagnosis

- Increasing recognition of genetic, structural and metabolic causes of epilepsy