EEG WORKSHOP
Nonepileptiform Abnormalities

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Background
normal
abnormal

Epileptiform discharges
what
where
when

Non epileptiform discharges
what
where
when

Activation procedure
EEG reading

Age

Background
- Normal
  - frequency
  - amplitude
  - reactivity
  - symmetry

Epileptiform discharges

Non epileptiform discharges

Activation procedure

PDR for age
- 1 yr: 5 Hz
- 4 yr: 6 Hz
- 5 yr: 7 Hz
- > 8 yr: 8 Hz

EEG reading

Age

Background

Epileptiform discharges

Non epileptiform discharges

Activation procedure

what
- abnormal
- slow activity
- amplitude asymmetry
- periodic pattern

Where
- gen.
- focal (F, T, O)
- continuous
- intermittent

When
- continuous
- intermittent
**EEG reading**

**Age**

**Background**
- Abnormal

**Epileptiform discharges**

**Non epileptiform discharges**

**Activation procedure**
- Continuous

**Where**
- Focal (F, T, O)

**When**

**Unequivocally**

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**Slow activity**

- Most common EEG manifestation of **focal brain dysfunction**
- More reliable when it is:
  - Continuous
  - Unreactive
  - High amplitude
  - Polymorphic
  - Unilateral
Continuous F-T delta slowing

Background

abnormal

Epileptiform discharges

normal

Non epileptiform discharges

what

Activation procedure

Focal(F,T,O)

Intermittent

EEG reading

Age

Where

When

Slow activity
IRDA: Intermittent rhythmic delta activity

- Varied form
  - non-specific
  - metabolic
  - structural
  - infectious
  - epilepsy / epileptiform pattern

Epilepsy 48(2), 2007

<table>
<thead>
<tr>
<th>IRDA</th>
<th>Adult</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FIRDA</td>
<td>FIRDA (unclear sig)</td>
</tr>
<tr>
<td></td>
<td>TIRDA</td>
<td>TIRDA</td>
</tr>
<tr>
<td></td>
<td>OIRDA (less common)</td>
<td>OIRDA</td>
</tr>
</tbody>
</table>

Epilepsy 48(2), 2007
OIRDA

- Occurs almost exclusively in children
- Associated with 1º generalized syndrome (childhood absence epilepsy)
- Occasionally seen in encephalopathic children (Salmonella infectn, SSPE)

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OIRDA

- Typical absence > atypical absence
- OIRDA in absence sz : 3-4 Hz
- OIRDA in localization-related sz : 2-3 Hz
- Most OIRDA < 5 sec
- When : awake and asleep recording
Absence sz: 3 Hz spike/wave and OIRDA
OIRDA in atypical CNS Samonellosis

FIRDA

- 1.5-4 Hz, Mostly in waking adult EEG

- **Previously** = deep midline structure, post fossa tumor, pituitary tumor, subcortical lesion, HC, cerebral edema, IICP

- **Currently** = ischemic brain injury, hemispheric brain tumor with mild to mod metabolic impairment, postictal
FIRDA

• FIRDA + normal b/g \(\Rightarrow\) structural lesion

• FIRDA + abnormal b/g \(\Rightarrow\) encephalopathy
Case report: Infectious 2001

**FIRDA(+) abn b/g**

1st EEG

2nd EEG

**FIRDA(+) abn b/g in Salmonella encephalopathy in adult**

1st EEG

2nd EEG

1st EEG

2nd EEG
FIRDA + abnormal b/g

- **TIRDA** = Temporal intermittent rhythmic delta activity
- **TIPDA** = Temporal intermittent polymorphic delta activity
- **TIRDA** $\rightarrow$ suggests temporal epileptogenesis
Facts

Temporal lobe epilepsy
- normal awake and asleep EEG
- HV or PS → may or may not induces abnormalities
- sleep deprivation → may induces epileptiform discharges
- TIRDA + epileptiform d/c → temporal lobe epilepsy

TIRDA
TIRDA

**Background**
- Abnormal epileptiform discharges
- Non-epileptiform discharges

**When**
- Continuous activation procedure

**Where**
- Generalized

**What**
- Slow activity

**EEG reading**
- Age
Continuous generalized delta slowing

Background

Abnormal

Epileptiform discharges

Non epileptiform discharges

Activation procedure

Age

What

Amplitude asymmetry

Where

Gen.

Focal (F,T,O)

When

Continuous

Intermittent

Amplitude asymmetry
Amplitude asymmetry

1. May occur as normal finding: isolated finding
e.g. alpha in R-H higher than L-H

2. If amplitude on the Rt is higher than the left for
   1 ½ times = significant asymmetry

3. If amplitude on the Lt is 25% higher than the Rt
   = significant asymmetry

Normal Amplitude Asymmetry
Differences in amplitude

- **Increased** amplitude can be seen in *ipsilateral lesions* (plus epileptiform discharges)

- **Diminished** amplitude
  1. excess fluid between the cortex and electrodes
  2. abnormalities of cortical gray matter
  3. congenital lesions: SWS, porencephalic cyst
  4. transient b/g attenuation = postictal

**Increased amplitude + epileptiform d/c = ipsilateral lesion**
Increased amplitude from the skull defect
Excessive fast activity

Breach rhythm

Diminished amplitude

Diminished amplitude
1. excess fluid between the cortex and electrodes
2. abnormalities of cortical gray matter
3. congenital lesions: SWS, porencephalic cyst
4. transient b/g attenuation = postictal
1. excess fluid between the cortex and electrode

2. Abnormalities of cortical gray matter
3. Congenital lesion + fluid collection

4. Transient b/g attenuation in postictal
Slow activity and amplitude asymmetry

- abnormalities of gray matter c white matter involvement e.g. ischemic stroke
- diminished amplitude b/g + polymorphic delta activity
Asynchrony