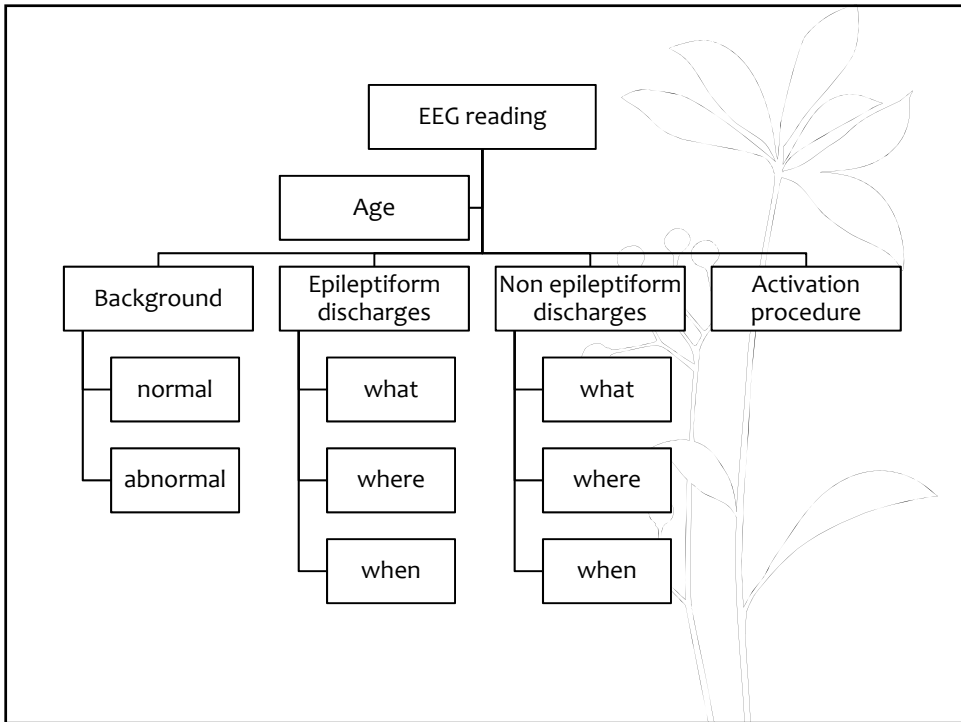
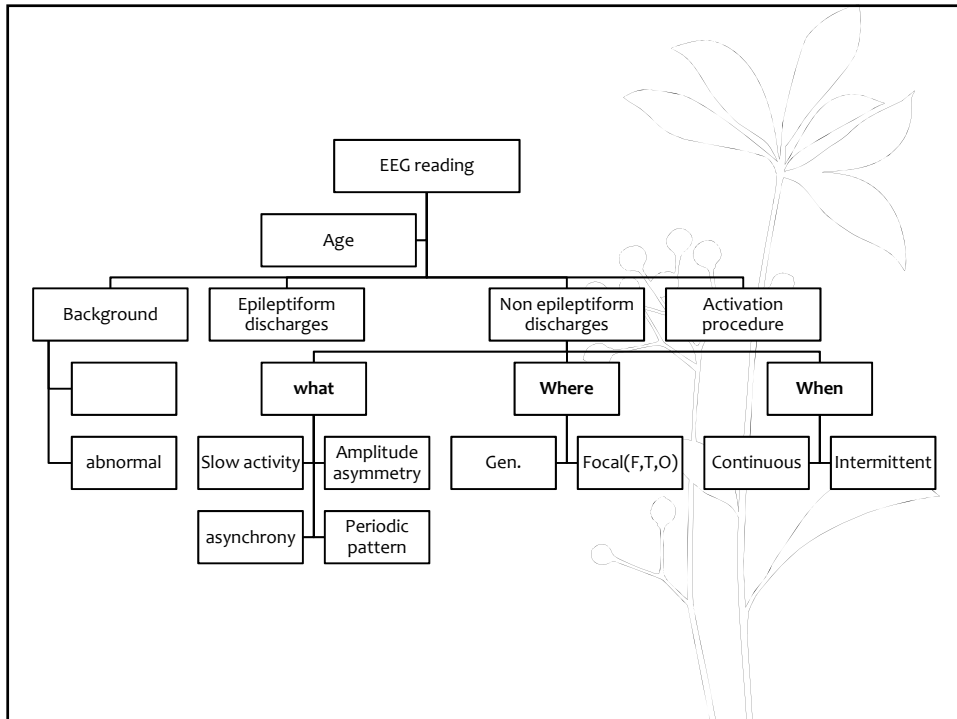
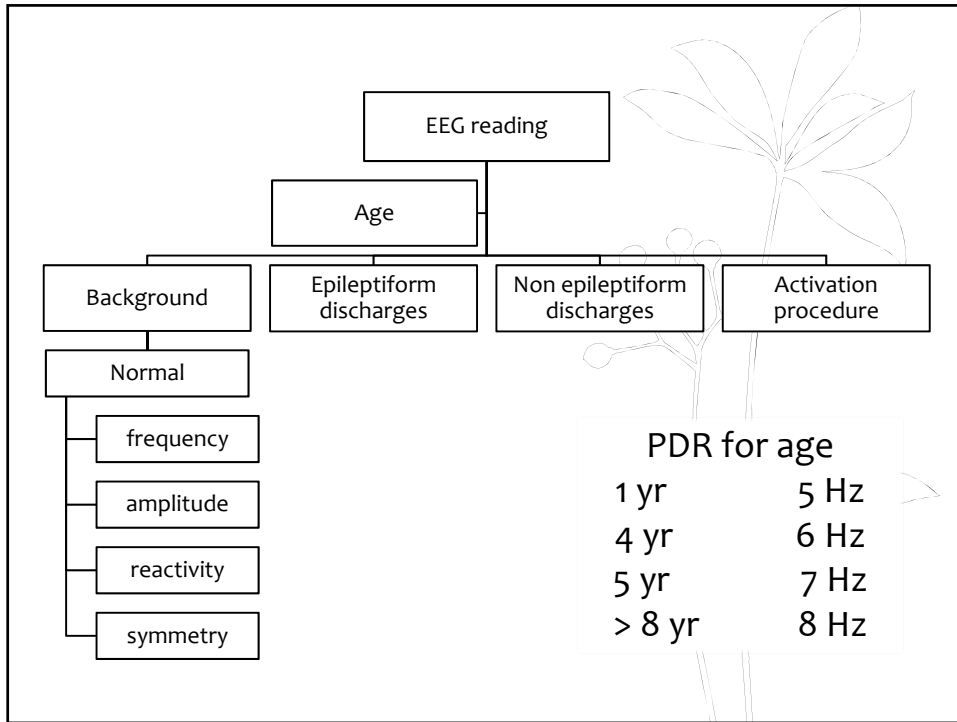


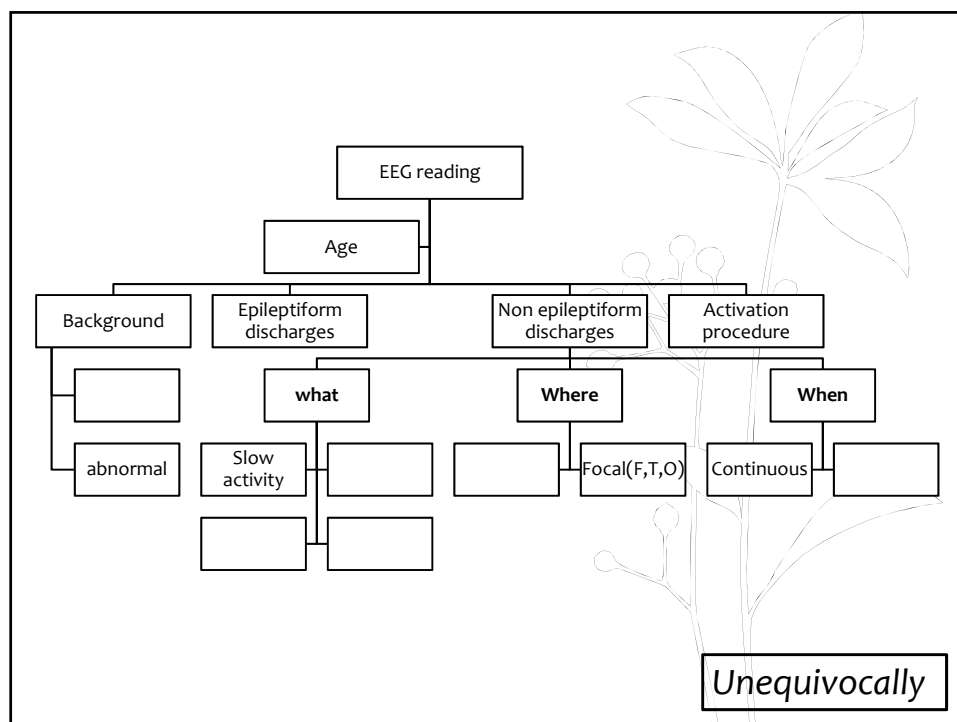
EEG WORKSHOP

Nonepileptiform Abnormalities

Kamornwan Katanyuwong MD
Chiangmai University Hospital
EST: 20th July 2010

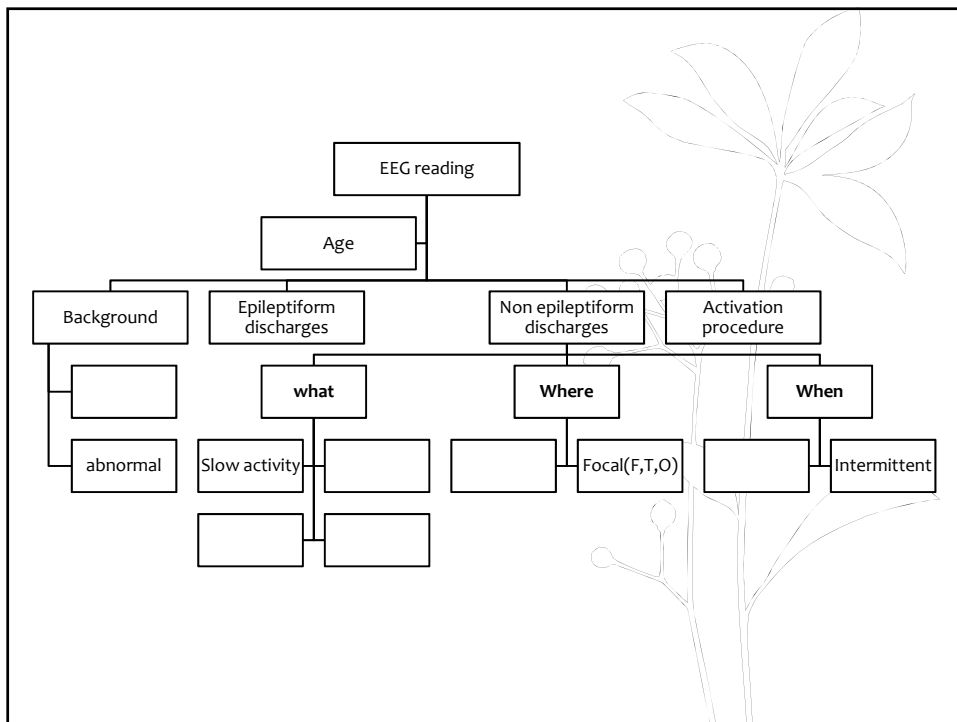
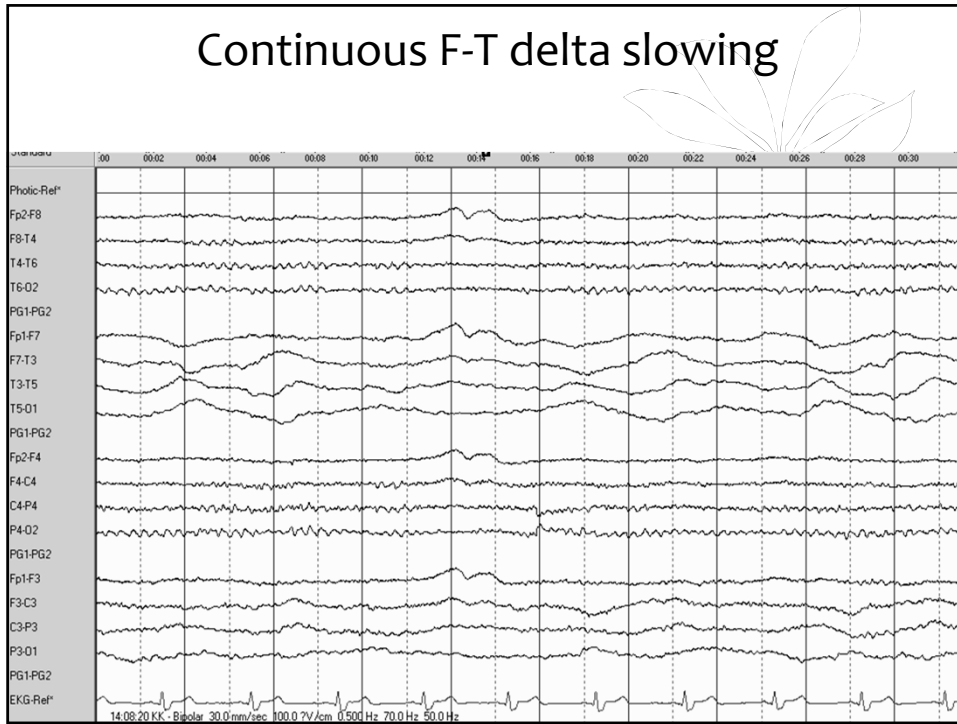






Slow activity

- Most common EEG manifestation of focal brain dysfunction
- More reliable when it is :
 - : unreactive
 - : high amplitude
 - : polymorphic
 - : unilateral



IRDA: Intermittent rhythmic delta activity

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



Epilepsia 48(2), 2007

IRDA

Adult	Children
<ul style="list-style-type: none"> • FIRDA • TIRDA • OIRDA (less common) 	<ul style="list-style-type: none"> • FIRDA (unclear sig) • TIRDA • OIRDA

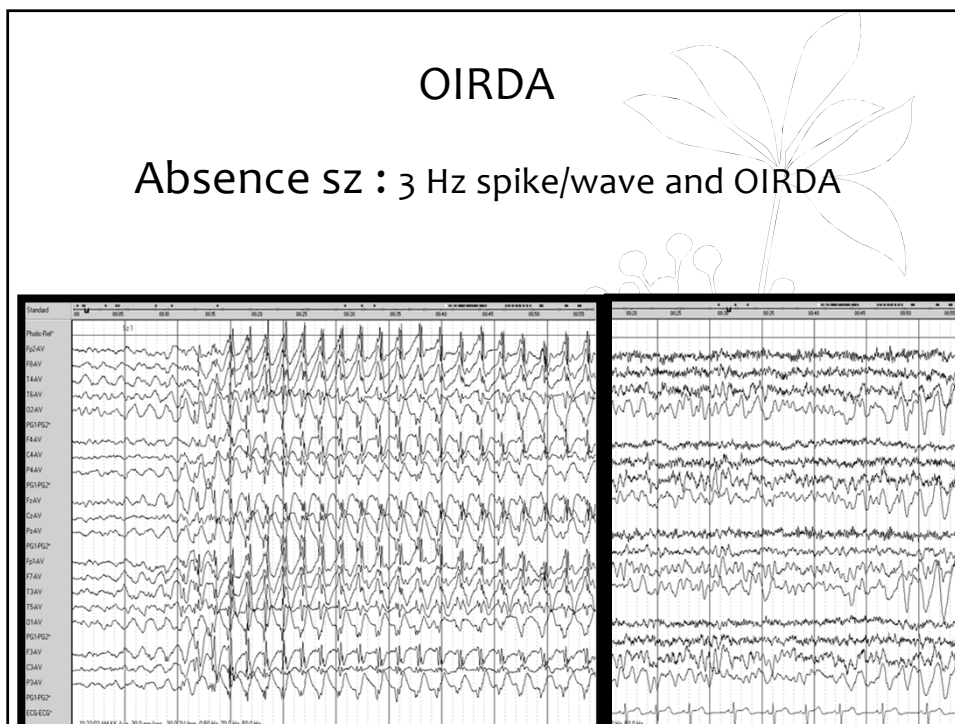
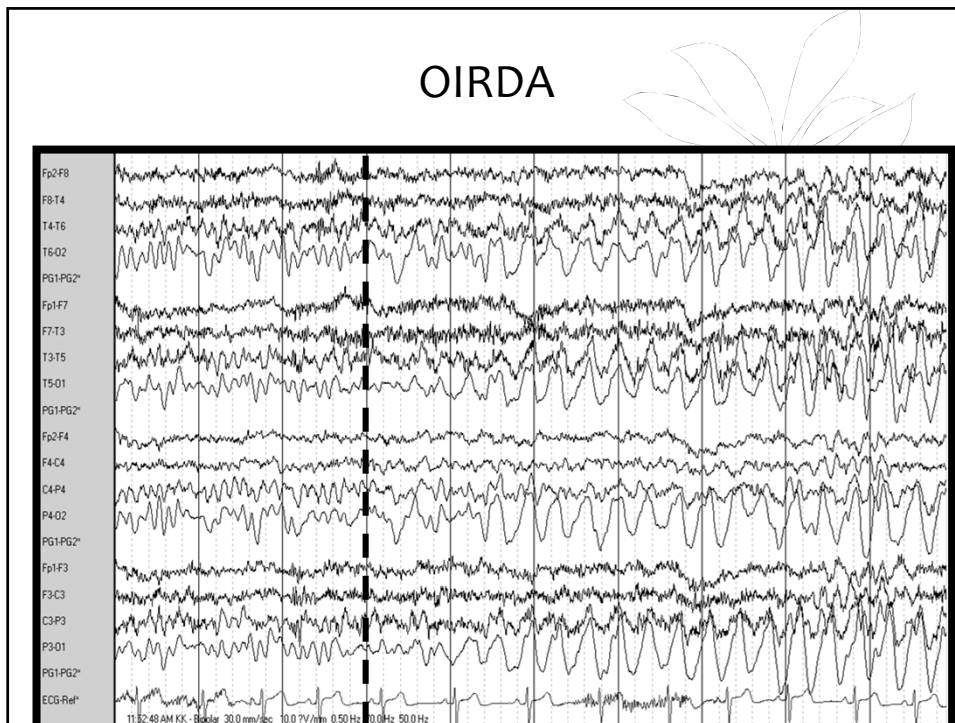
Epilepsia 48(2), 2007

OIRDA

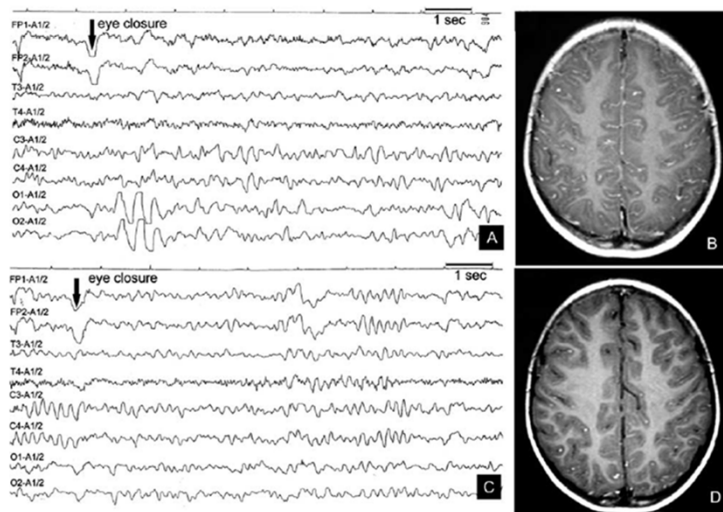
- Occurs almost exclusively in children
- Associated with 1^o generalized syndrome (childhood absence epilepsy)
- Occasionally seen in encephalopathic children (Salmonella infectⁿ, SSPE)

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



OIRDA in atypical CNS Salmonellosis



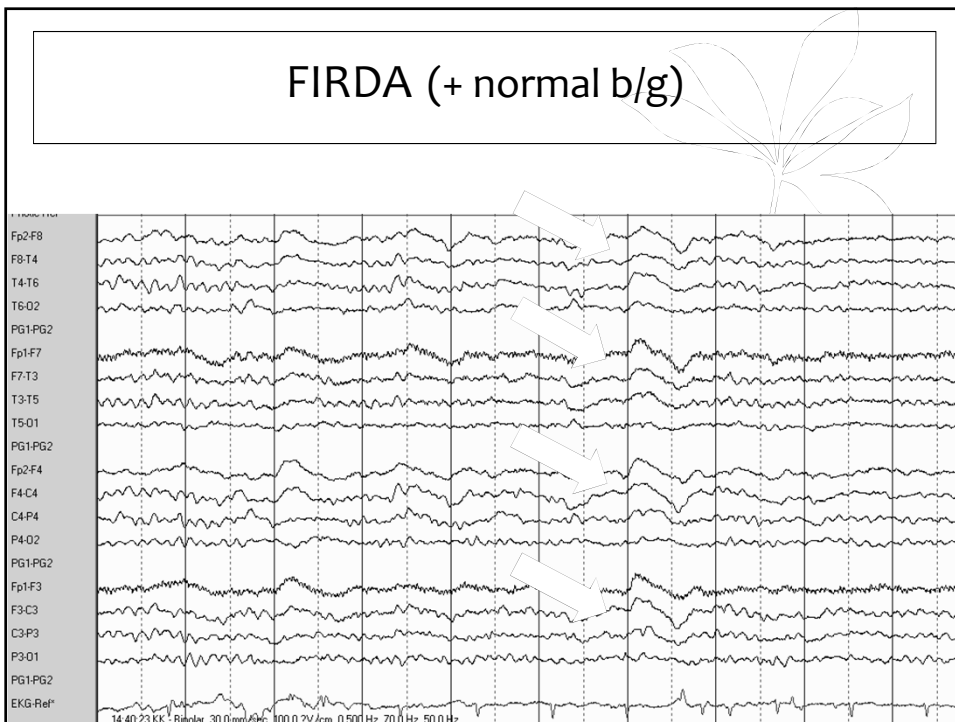
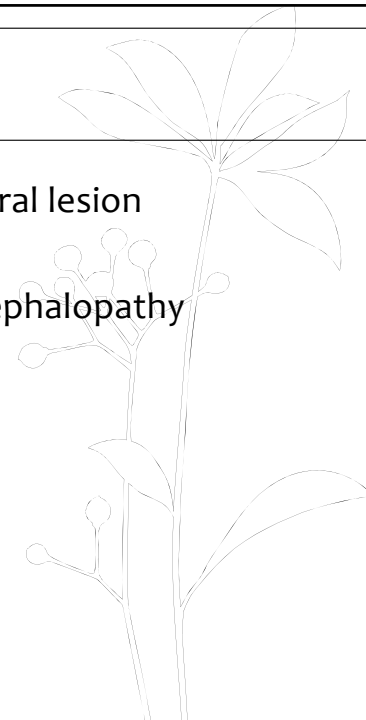
Clinical Neurophysiology 2005

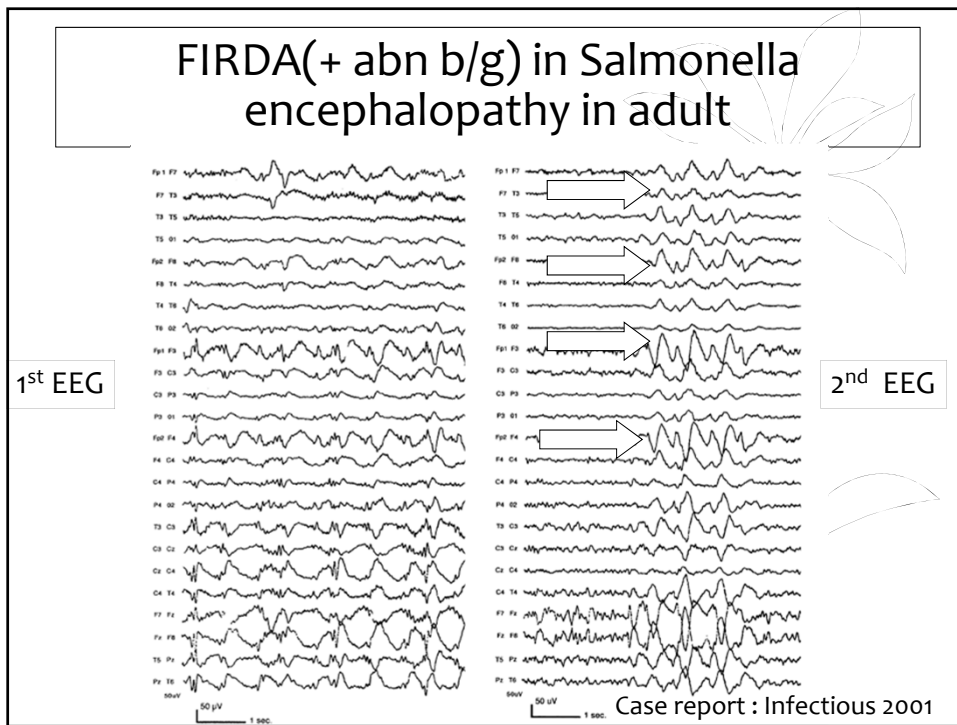
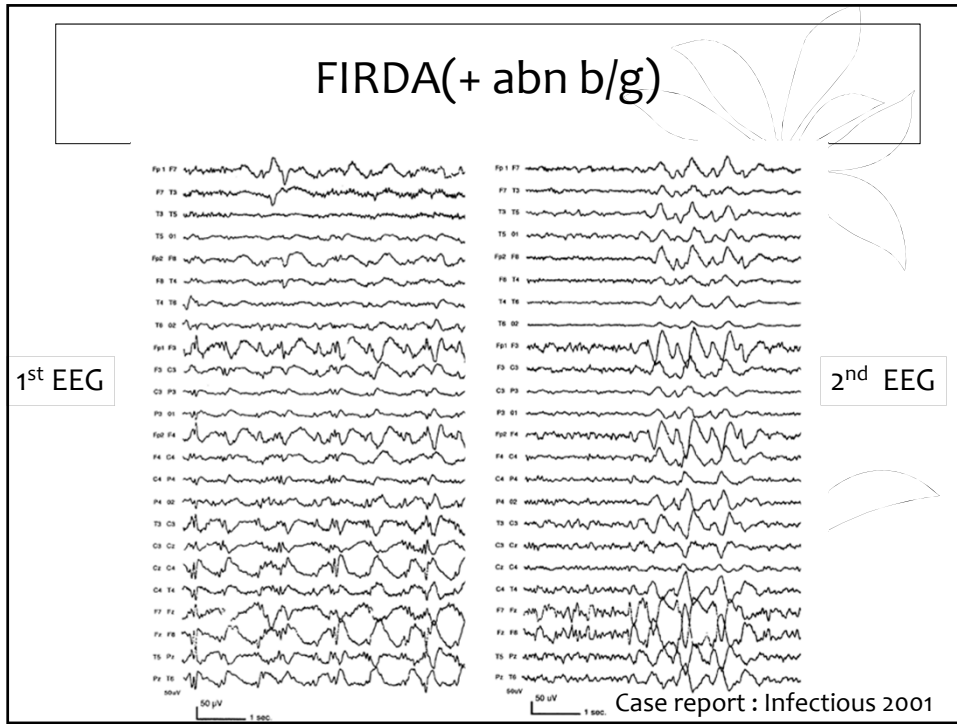
FIRDA

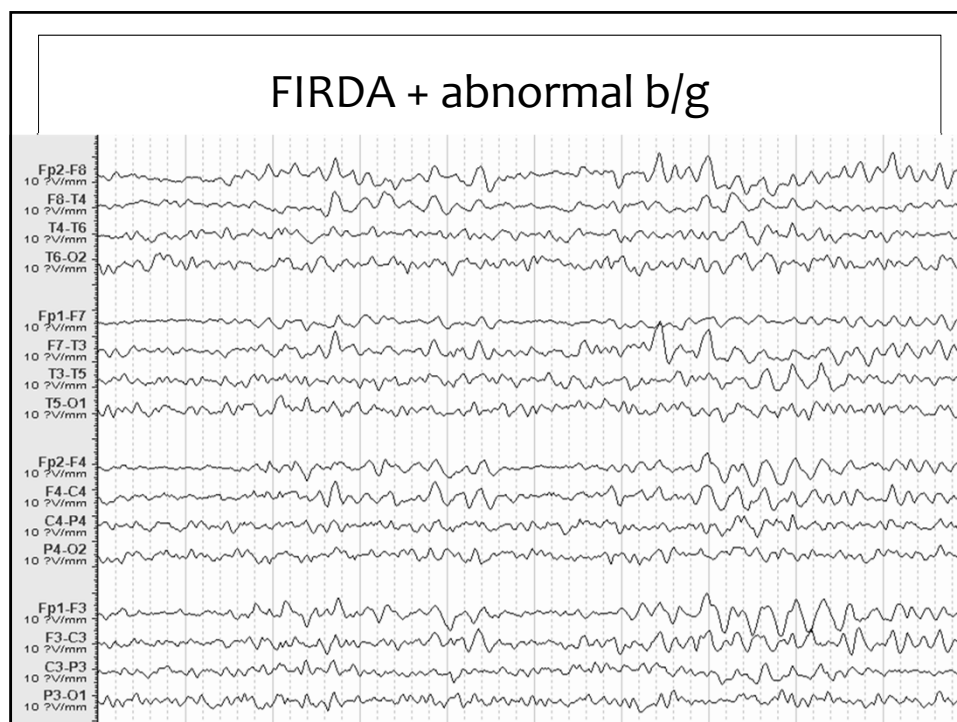
- 1.5-4 Hz, Mostly in waking adult EEG
- Previously = deep midline structure, post^r fossa tumor, pituitary tumor, subcortical lesion, HC, cerebral edema, IICP
- Currently = ischemic brain injury, hemispheric brain tumor with mild to mod metabolic impairment, postictal

FIRDA

- FIRDA + normal b/g → structural lesion
- FIRDA + abnormal b/g → encephalopathy







TIRDA

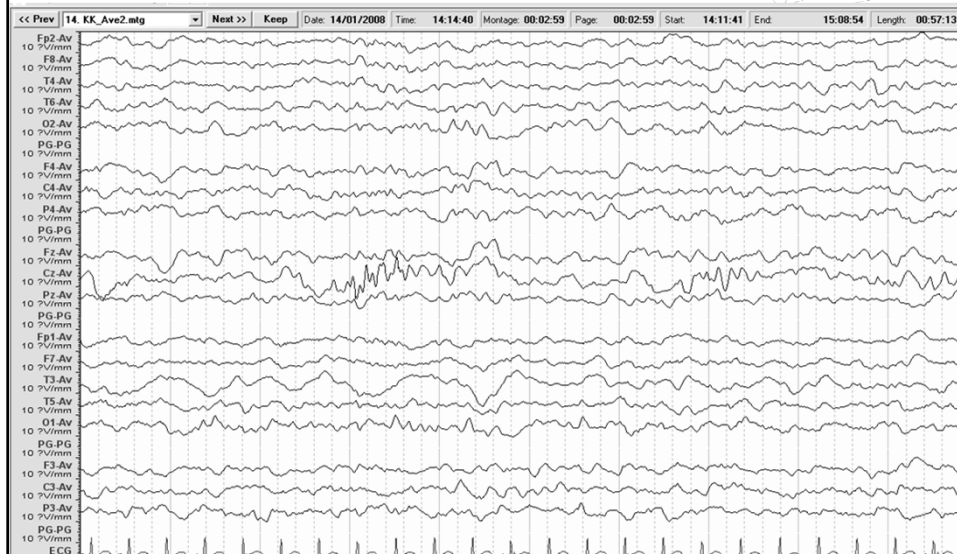
- TIRDA = Temporal intermittent rhythmic delta activity
- TIPDA = Temporal intermittent polymorphic delta activity
- TIRDA → suggests temporal epileptogenesis

Facts

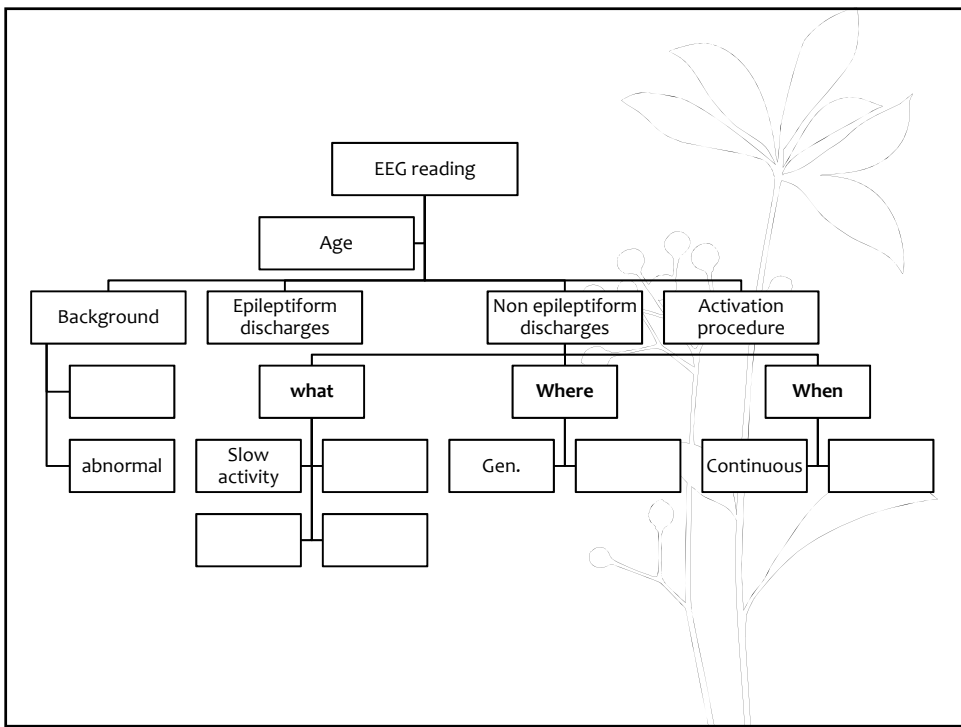
Temporal lobe epilepsy

- 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

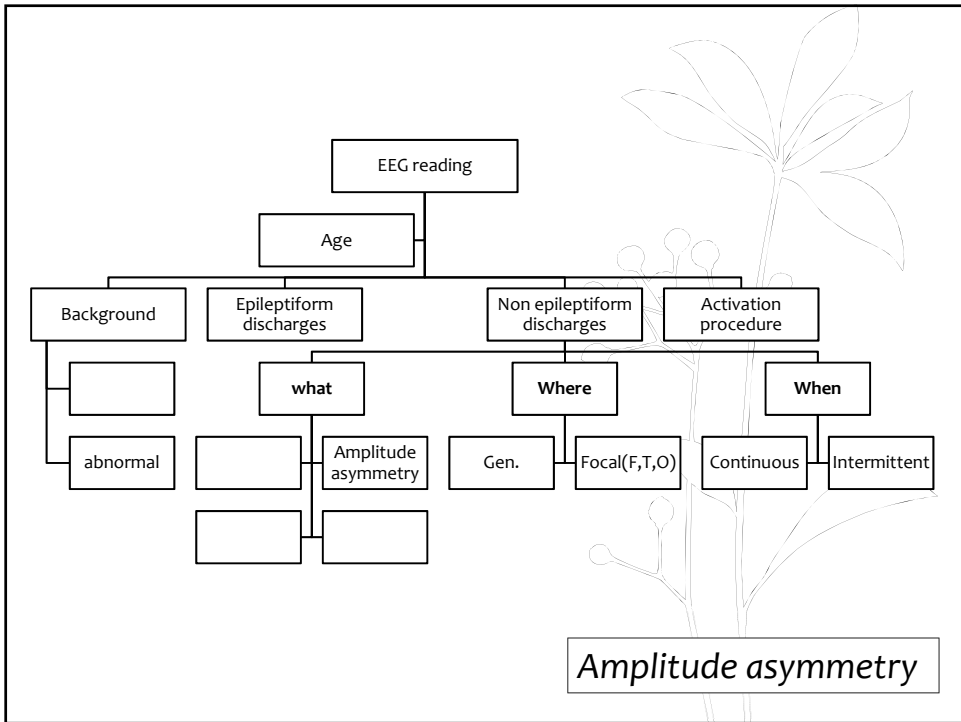
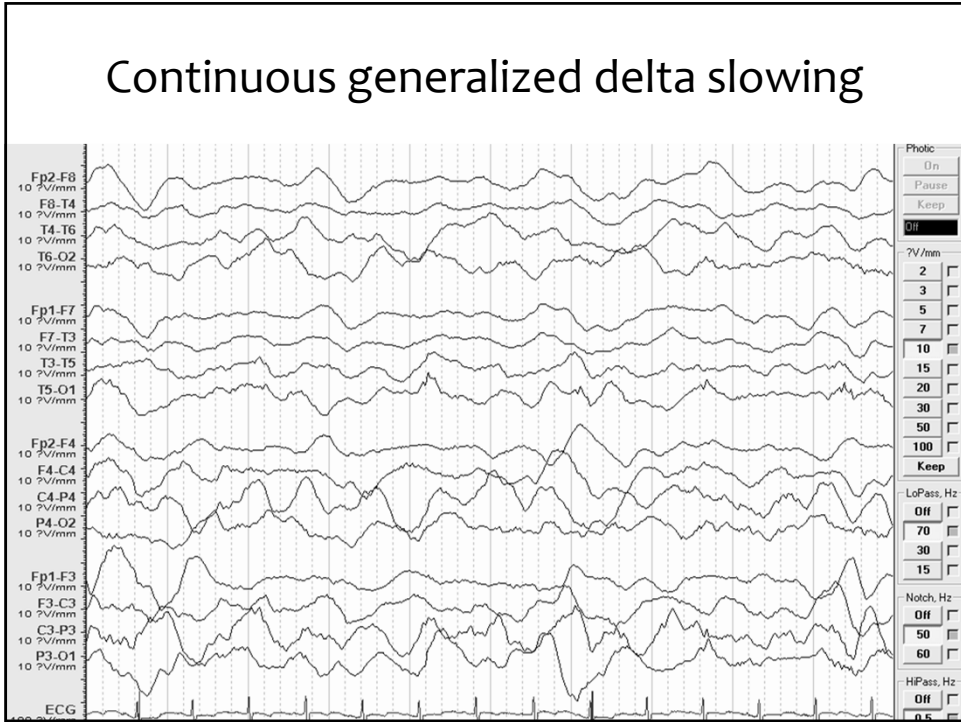
TIRDA



TIRDA

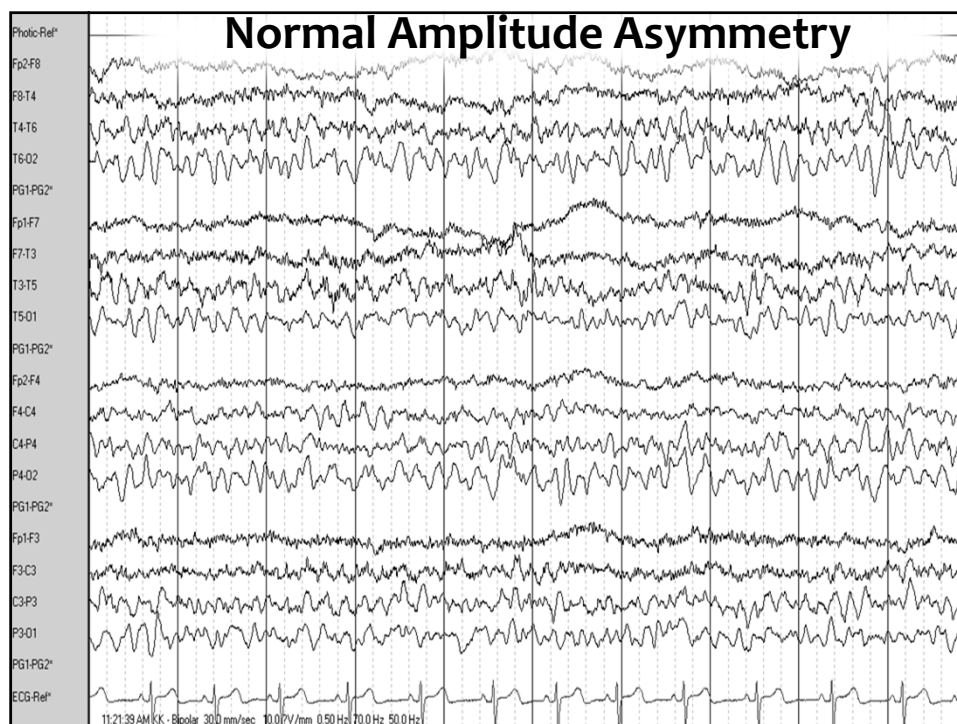


Continuous generalized delta slowing



Amplitude asymmetry

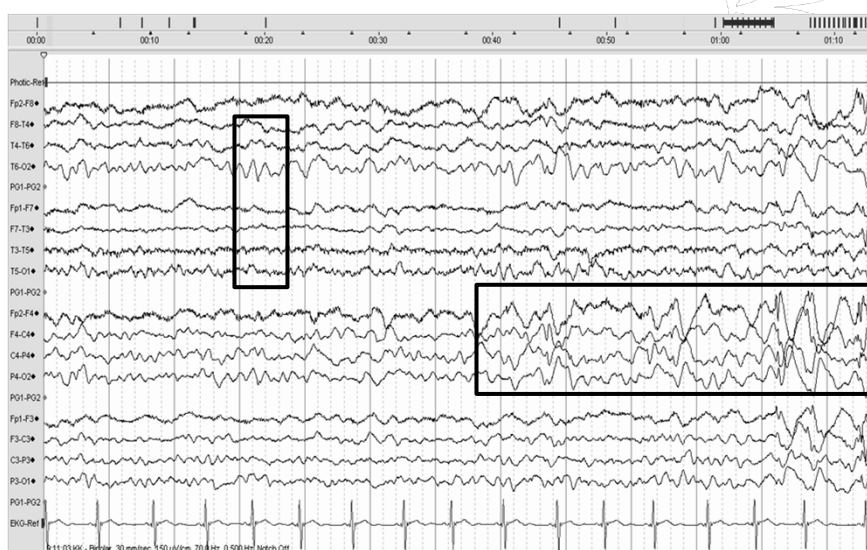
1. May occur as normal finding: isolated finding
e.g. alpha in R-H higher than L-H
2. If amplitude on the Rt is higher than the left for
 $1\frac{1}{2}$ times = significant asymmetry
3. If amplitude on the Lt is 25% higher than the Rt
= significant asymmetry



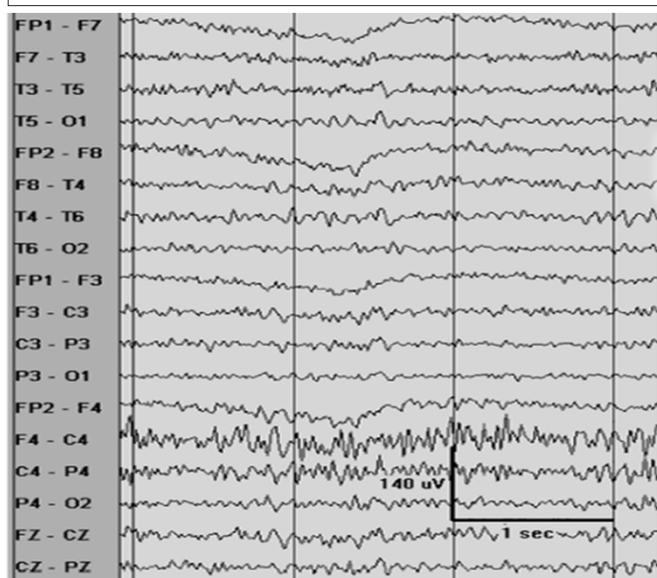
Differences in amplitude

- ▶ 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

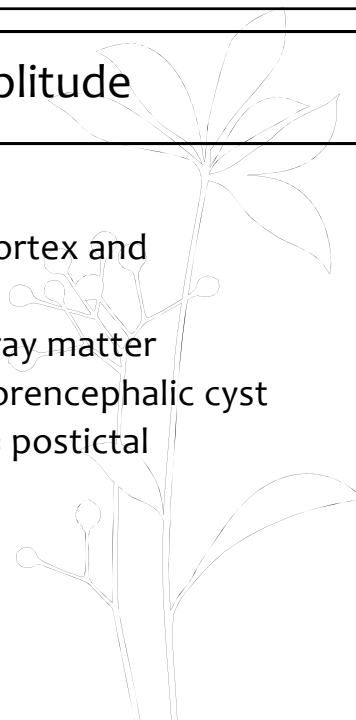


Breach rhythm

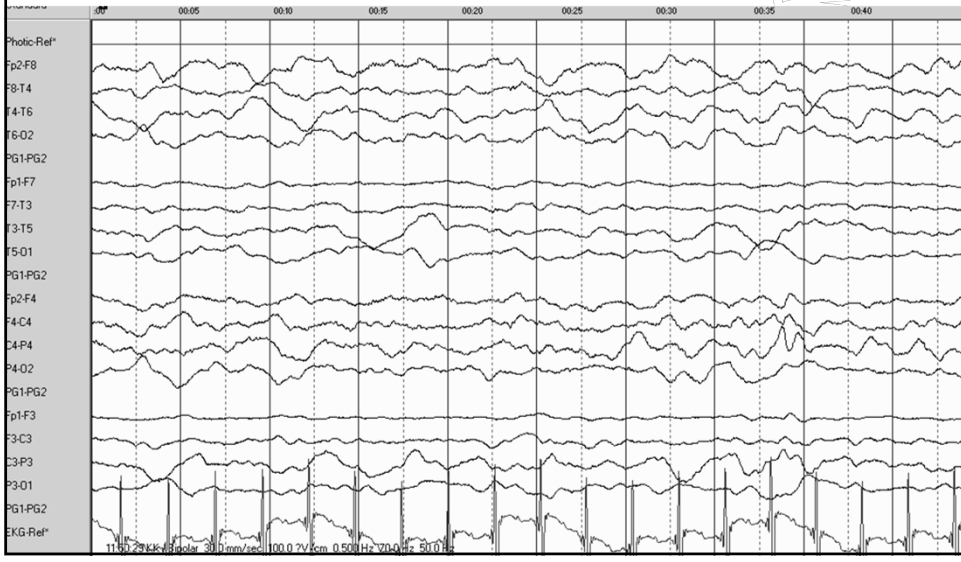
Diminished amplitude

Diminished amplitude

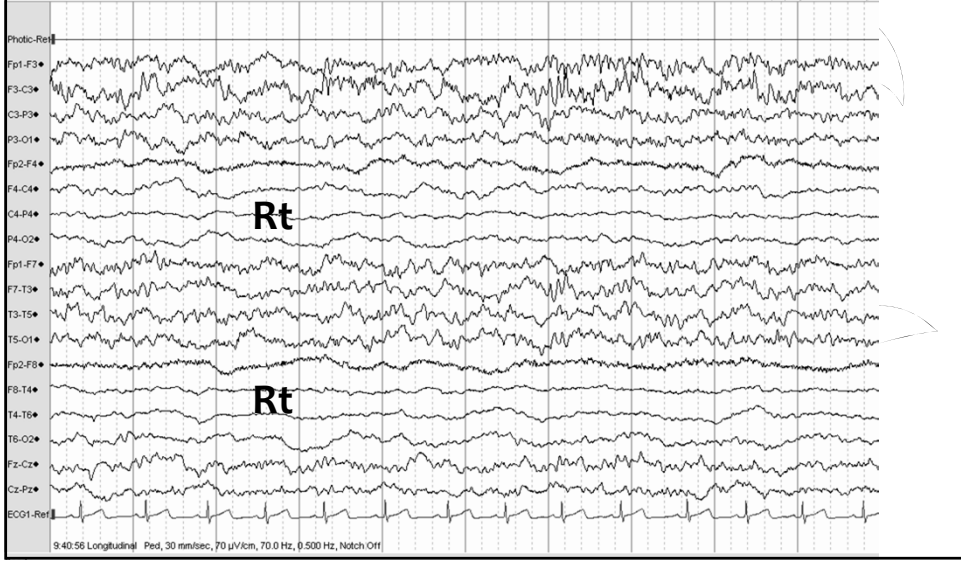
1. excess fluid between the cortex and electrodes
2. abnormalities of cortical gray matter
3. congenital lesions: SWS, porencephalic cyst
4. transient b/g attenuation = postictal



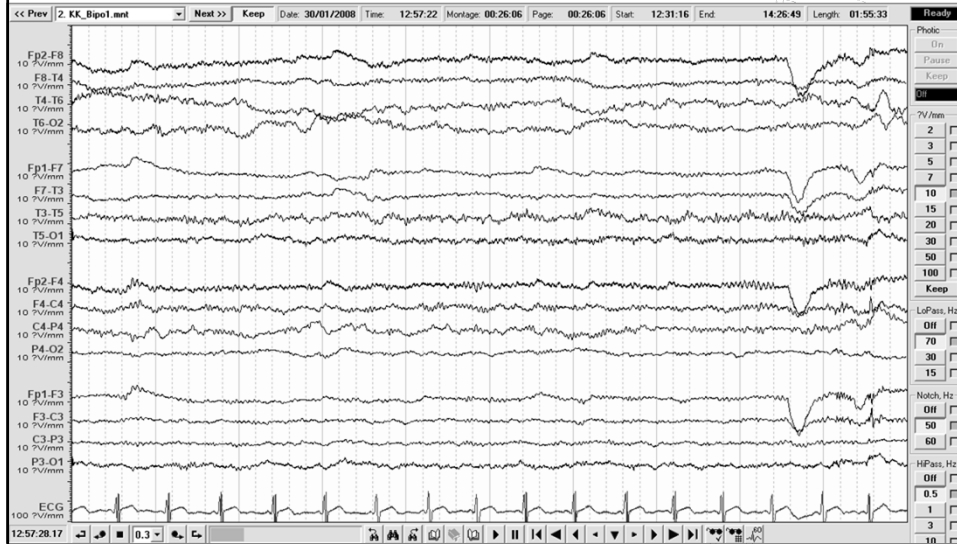
1. excess fluid between the cortex and electrode



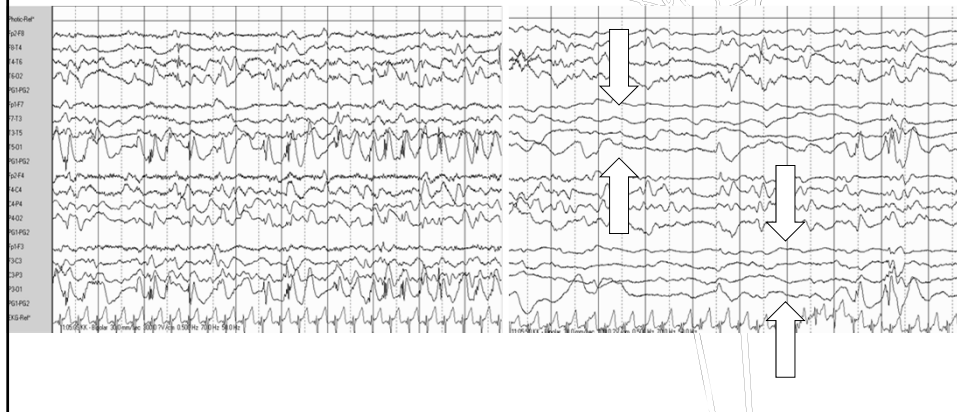
2. Abnormalities of cortical gray matter



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

