EEG WORKSHOP Nonepileptiform Abnormalities

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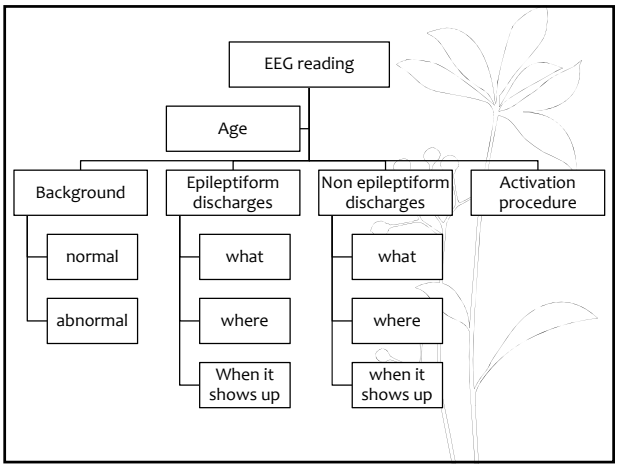
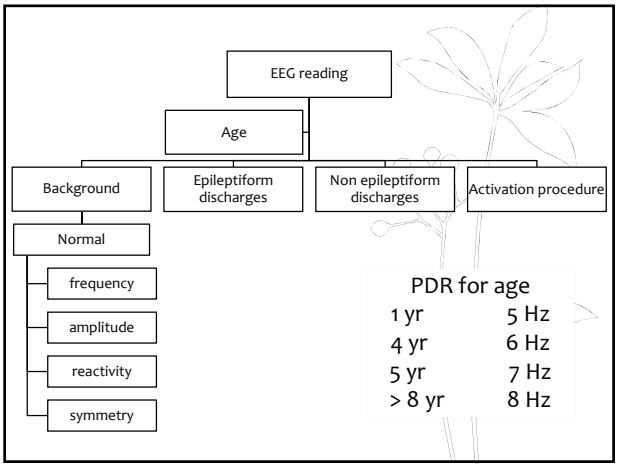
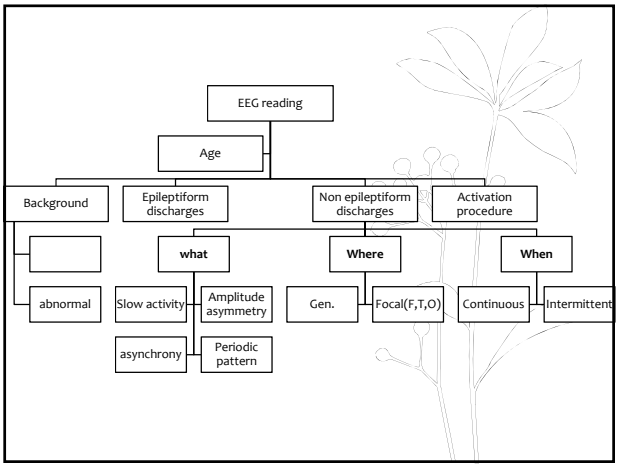
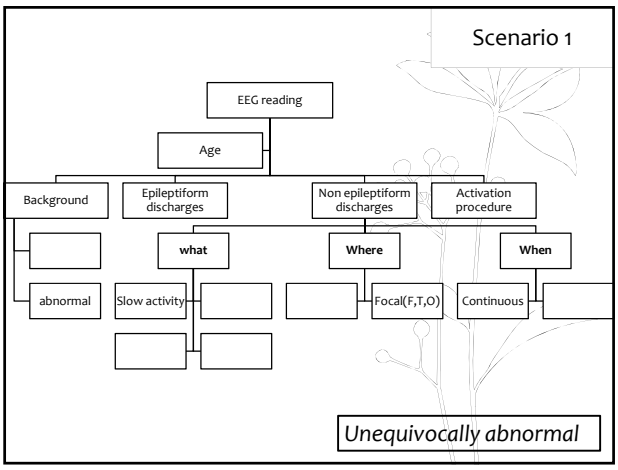




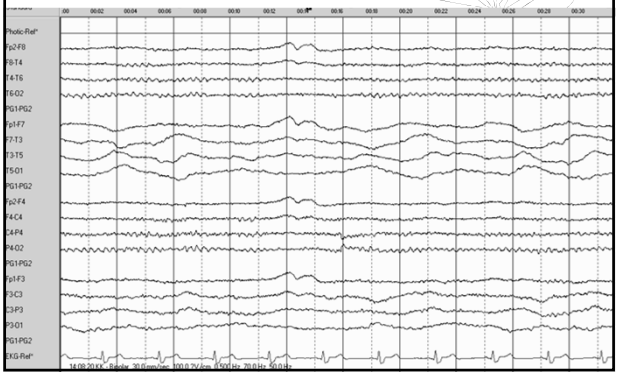




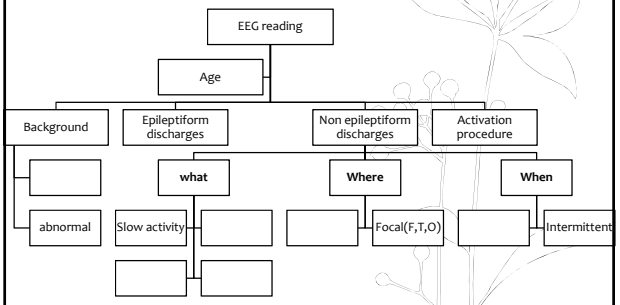
Slow activity

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

Continuous F-T delta slowing



Scenario 2



IRDA, migraine, postictal, TIA

IRDA: Intermittent rhythmic delta activity

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

Epilepsia 48(2), 2007

IRDA

Adult	Children
• FIRDA	• FIRDA (unclear sig.)
• TIRDA	• TIRDA
• OIRDA (less common)	• OIRDA

Epilepsia 48(2), 2007

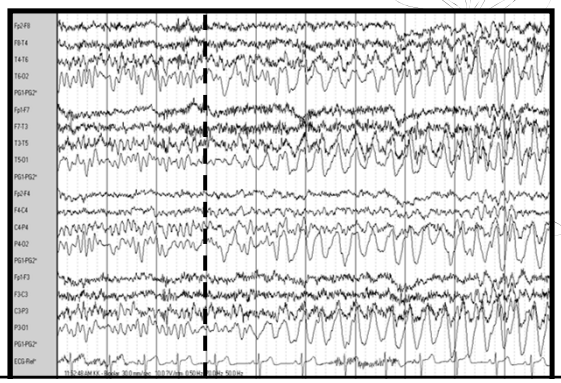
OIRDA

- Occurs almost exclusively in children
- Associated with 1° generalized syndrome (childhood absence epilepsy)
- Occasionally seen in encephalopathic children (Salmonella infect^{n}, SSPE)

OIRDA

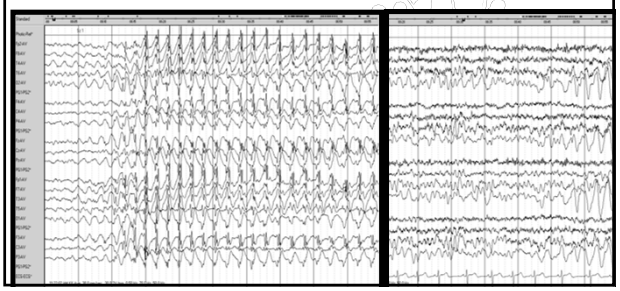
- Typical absence > atypical absence
- OIRDA in absence sz : 3-4 Hz
- OIRDA in localization-related sz : 2-3 Hz
- Most OIRDA < 5 sec
- When : awake and asleep recording

OIRDA



OIRDA

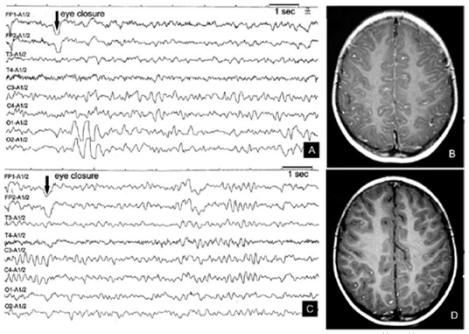
Absence sz : 3 Hz spike/wave and OIRDA



OIRDA

- Occasionally seen in encephalopathic children (Salmonella infect^{n}, SSPE)

OIRDA in atypical CNS Salmonellosis



Clinical Neurophysiology 2005

FIRDA

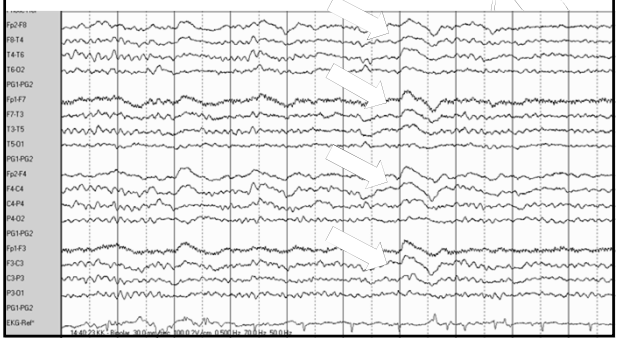
- 1.5-4 Hz, Mostly in waking adult EEG
- Previously = deep midline structure, post^{r} fossa tumor, pituitary tumor, subcortical lesion, HC, cerebral edema, IICP
- Currently = ischemic brain injury, hemispheric brain tumor with mild to mod metabolic impairment, postictal

FIRDA

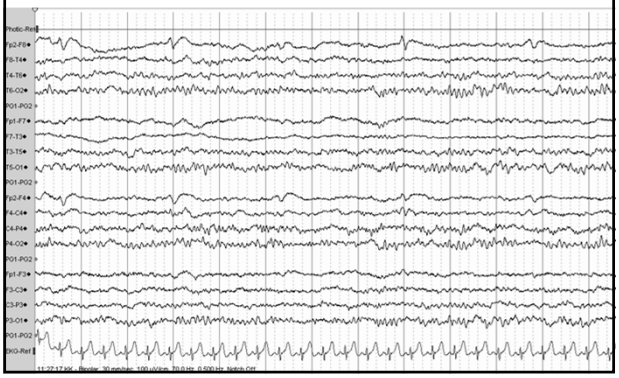
- FIRDA + normal b/g → structural lesion
- FIRDA + abnormal b/g → encephalopathy

Uncommon epilepsy

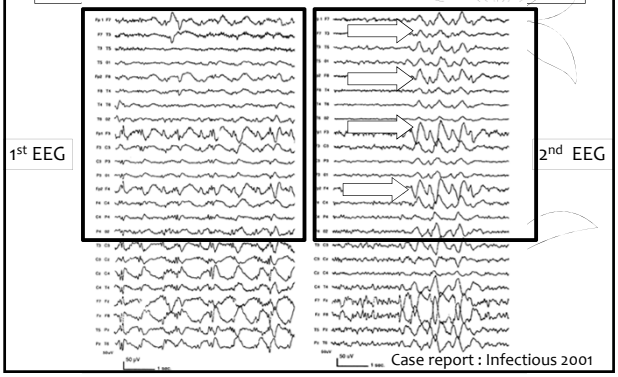
FIRDA (+ normal b/g)



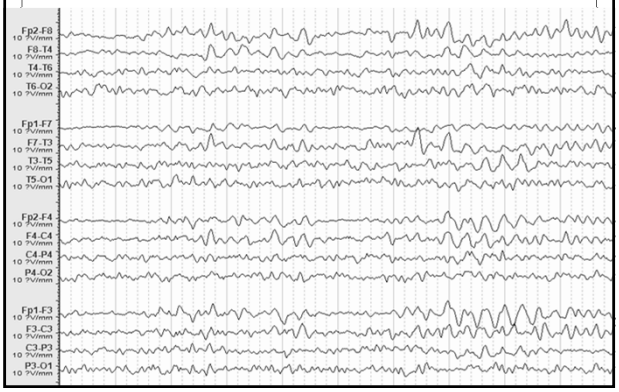
Where is the lesion ?



FIRDA(+ abn b/g)



FIRDA + abnormal b/g



TIRDA

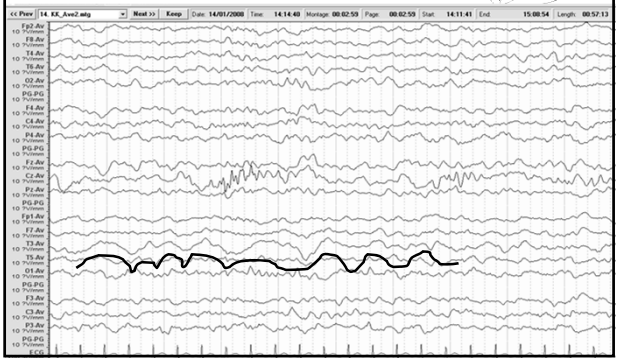
- TIRDA = Temporal intermittent rhythmic delta activity
- TIPDA = Temporal intermittent polymorphic delta activity
- TIRDA → suggests temporal epileptogenesis

Facts

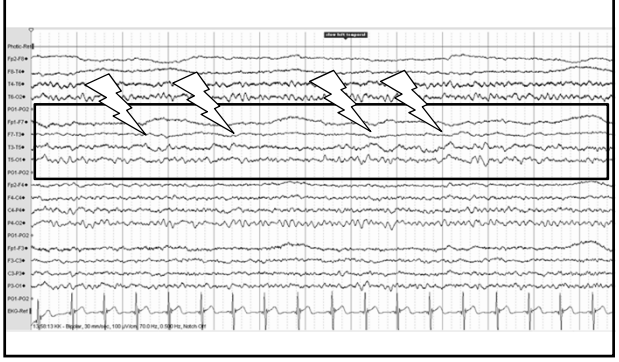
Temporal lobe epilepsy

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

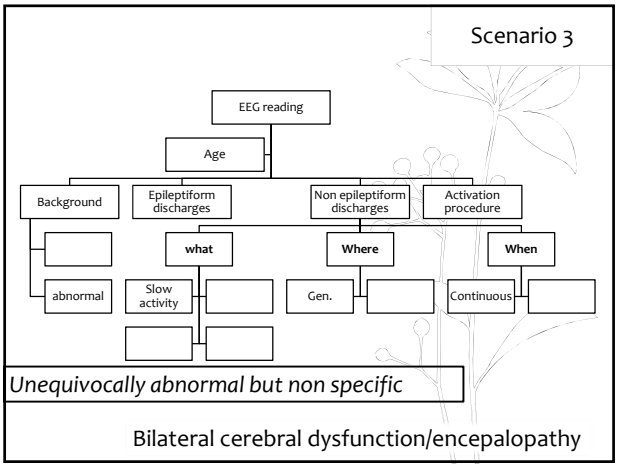
TIRDA



TIRDA



Scenario 3

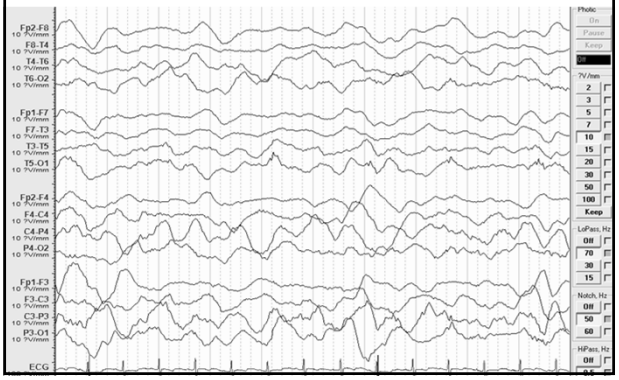


Unequivocally abnormal but non specific

Bilateral cerebral dysfunction/encepalopathy

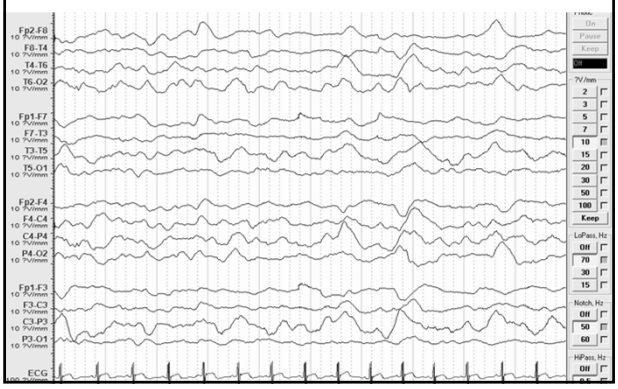
Continuous generalized delta slowing

Ex 1

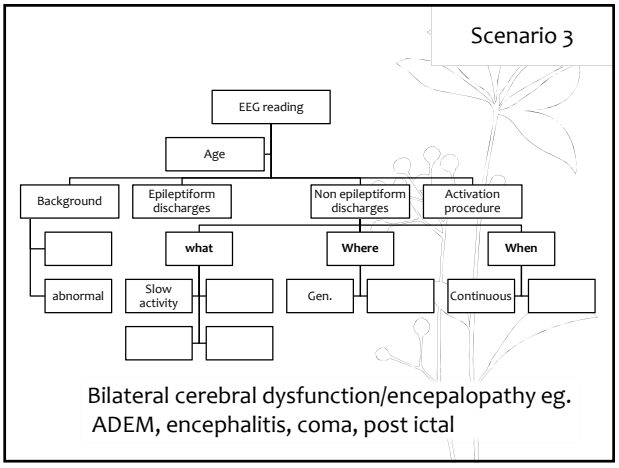


Continuous generalized delta slowing

Ex 2

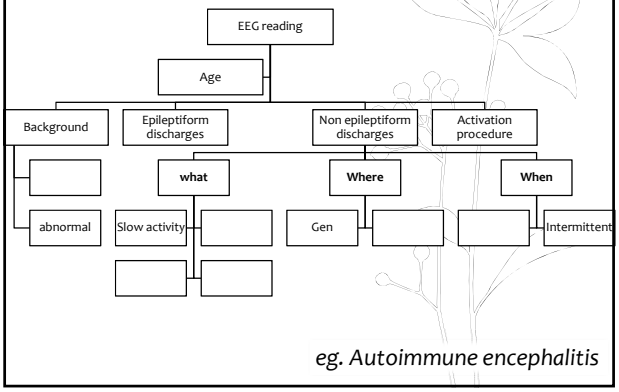


Scenario 3

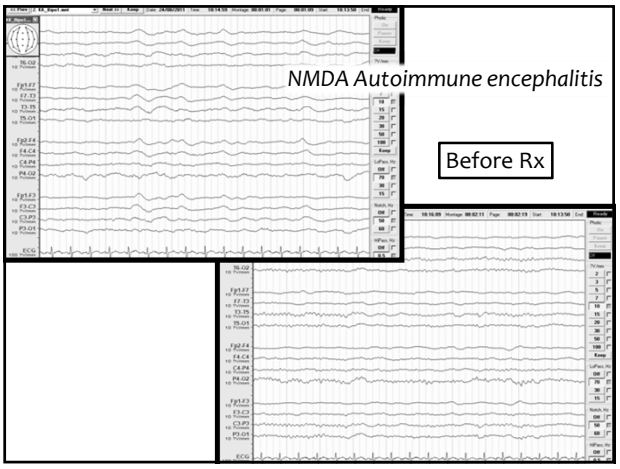


Bilateral cerebral dysfunction/encepalopathy eg. ADEM, encephalitis, coma, post ictal

Scenario 4

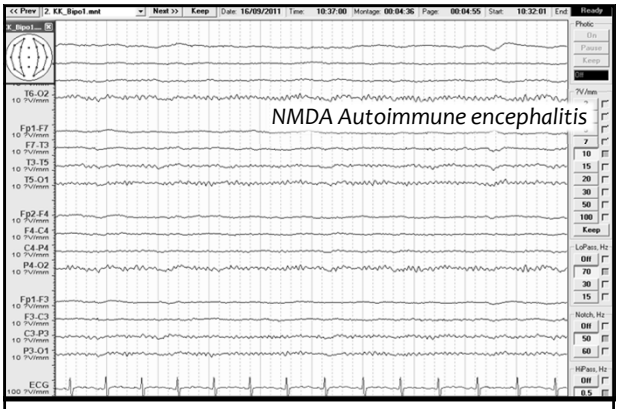


eg. Autoimmune encephalitis



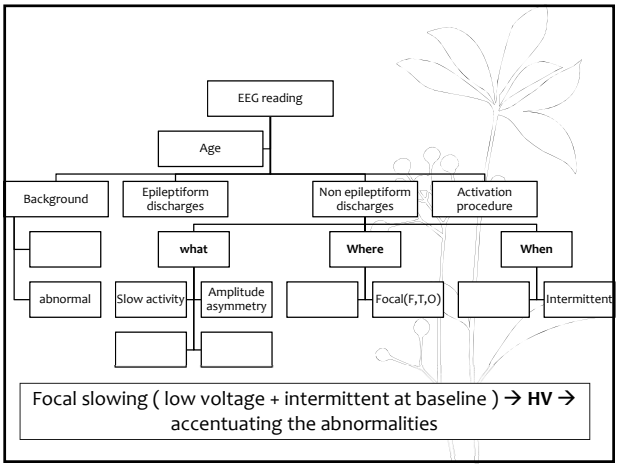
NMDA Autoimmune encephalitis

Before Rx

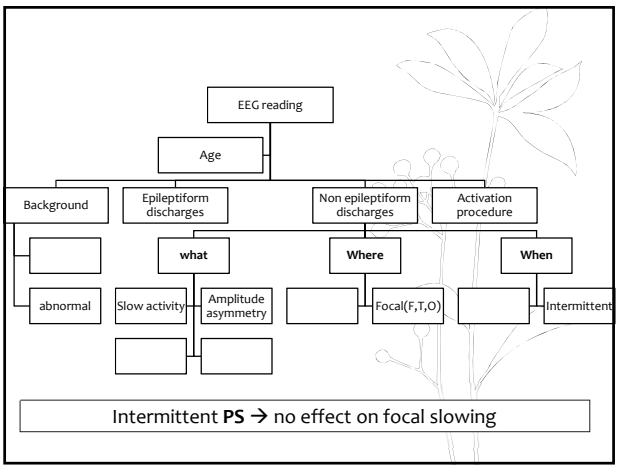


NMDA Autoimmune encephalitis

9 days after IVIG treatment

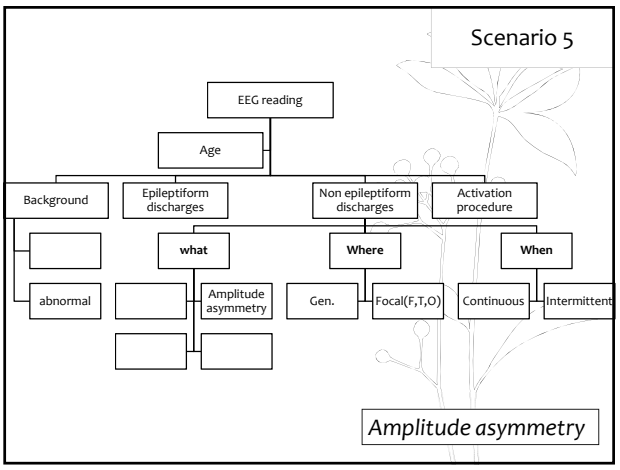


Focal slowing (low voltage + intermittent at baseline) → **HV** → accentuating the abnormalities



Intermittent **PS** → no effect on focal slowing

Scenario 5

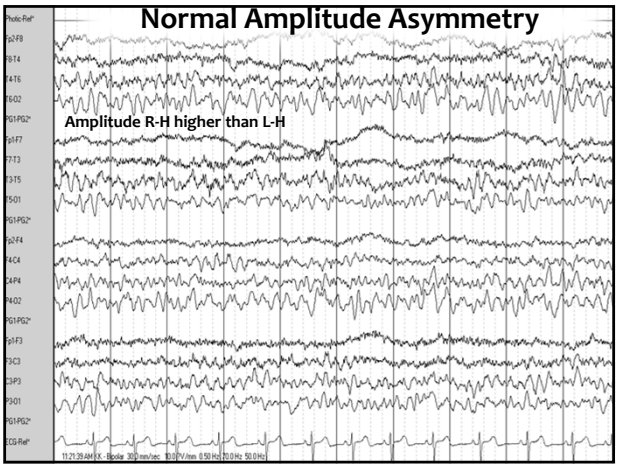


Amplitude asymmetry

Amplitude asymmetry

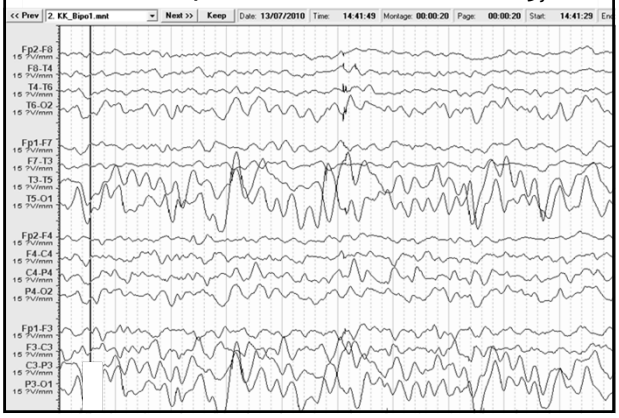
1. May occur as normal finding: isolated finding eg. alpha in R-H higher than L-H
2. If amplitude on the Rt is higher than the left for 1 ½ times = significant asymmetry
3. If amplitude on the Lt is 25% higher than the Rt = significant asymmetry

Normal Amplitude Asymmetry



Which hemisphere is abnormal ?

3y6m

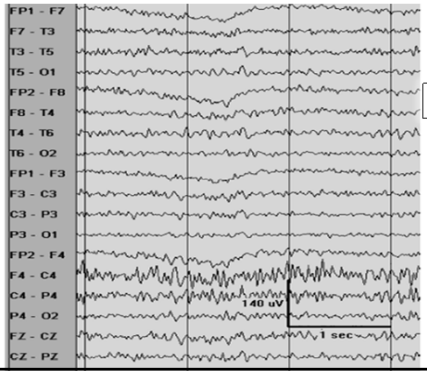


Differences in amplitude

- 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

Increased amplitude from the skull defect
Excessive fast activity

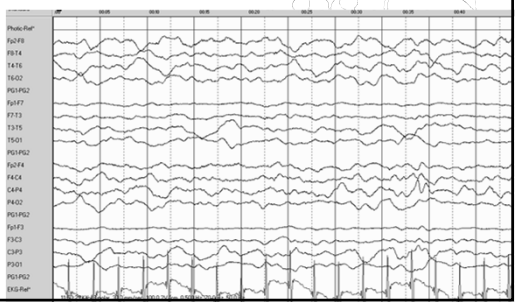


Breach rhythm

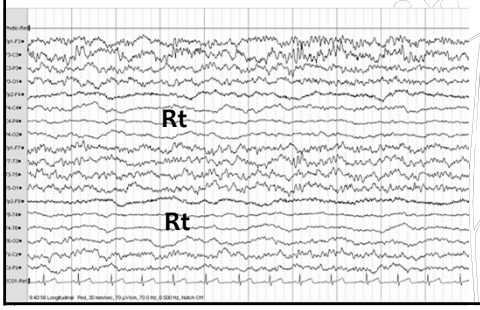
Diminished amplitude

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

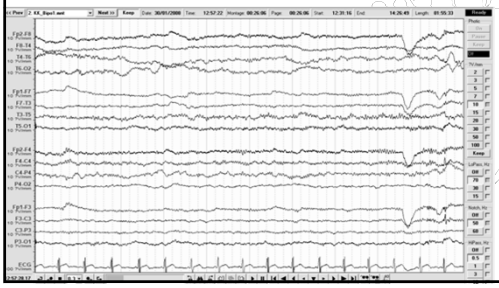
1. excess fluid between the cortex and electrode



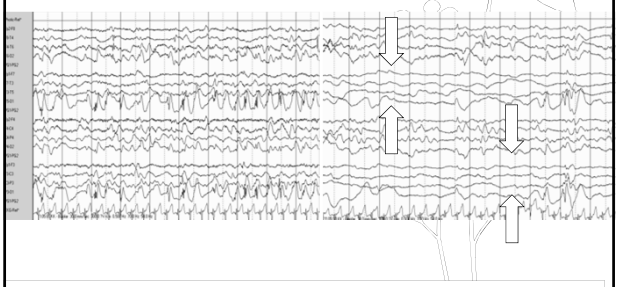
2. Abnormalities of cortical gray matter



3. Congenital lesion + fluid collection



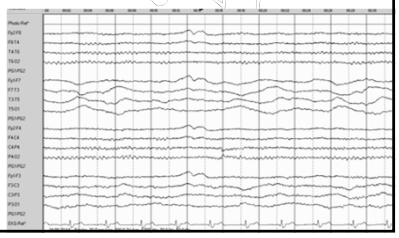
4. Transient b/g attenuation in postictal

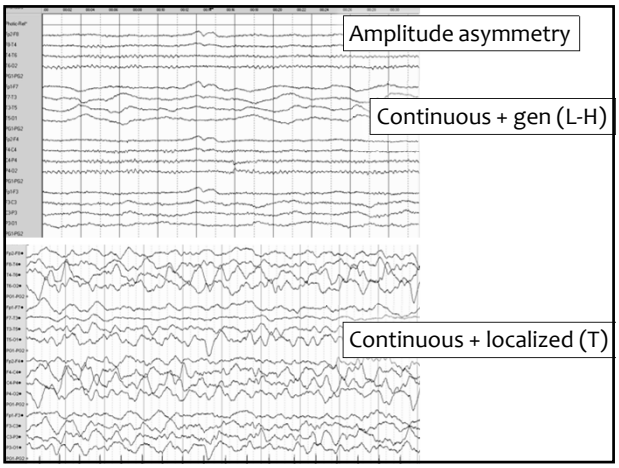


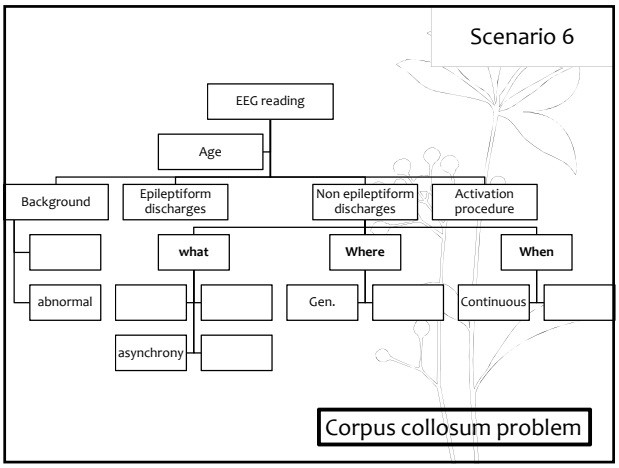
Post ictal state can be seen with generalized slow activity

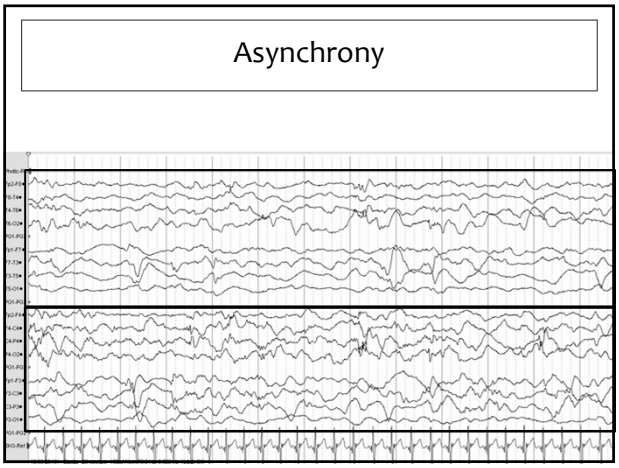
Slow activity and amplitude asymmetry

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




Amplitude asymmetry
Continuous + gen (L-H)
Continuous + localized (T)


Scenario 6
EEG reading
Age
Background
Epileptiform discharges
Non epileptiform discharges
Activation procedure
what
Where
When
abnormal
Gen.
Continuous
asynchrony
Corpus collosum problem


Asynchrony