Non-epileptiform EEG abnormalities and the EEG in coma

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I have no financial relationships to disclose that are relative to the content of my presentation.
Self assessment questions
Q1- What is not true about the preceding EEG?

A. Bursts are likely associated with myoclonic jerks
B. Pattern is typical of CJD
C. Interval between discharges decreases with progression
D. Pattern may be associated with PCP intoxication
Q2- what is not true about the pattern above

A. Associated with clinical seizures in most individuals
B. May be seen after stroke
C. Requires barbiturate coma if it persists despite seizure medications
D. May be seen with herpes encephalitis
Q3- Which is true of the EEG above

A. Diagnostic of structural lesion on the right
B. Indicative of subcortical but not cortical abnormality
C. May be a postictal finding
D. Shows a seizure discharge in the left occipital region
Q4- Which is not true of the EEG above?

A. May be seen with hepatic encephalopathy
B. May be seen with renal encephalopathy
C. Indicative of generalized epilepsy
D. Will attenuate with diazepam
Learning Objectives

- To recognize nonepileptiform abnormalities and their pathophysiology
- To recognize the clinical significance of focal and generalized slow abnormalities
- To recognize the clinical significance of focal and generalized amplitude abnormalities
- To recognize periodic patterns and their significance
- To recognize the value of the EEG in coma
EEG ABNORMALITIES

- Epileptiform / ictal activity
  focal/ lateralized
  generalized
- Slow waves
  focal
  generalized asynchronous
  bilaterally synchronous
- Amplitude abnormalities
  focal/ lateralized asymmetry
  generalized changes
- Other deviations from normal patterns
Focal slow wave activity

- Experimentally reproduced by subcortical lesions in white matter, thalamus, hypothalamus, midbrain
- Not seen in purely cortical lesions
- Clinical correlation: Focal structural damage or transient dysfunction in subcortical white matter / thalamus
- Clinical examples: Stroke, tumor, abscess; TIA, migraine, postictal state
Focal slow waves

- lower frequency and more persistent in the center (often lower voltage as well, if cortex is involved)
- less reactive in the center-surround activity may be more reactive
- most conspicuous in the waking state
- underlying structural pathology most likely if slow activity is polymorphic, persistent and unreactive
Focal slow waves
relation to underlying lesion

- temporal relation is best at onset- may precede but usually does not outlast clinical symptoms and signs
- spatial relation varies with location- it is most precise in superficial lesions. EEG changes then are more restricted
Focal slow activity
localizing value

- increased when activity is more discrete and associated with depression of faster frequencies
- slow wave focus may appear lateral and anterior to lesion (central & parietal lesions can give temporal delta)
- Frontal and occipital lesions may give bilateral slow activity
EEG abnormalities associated with focal slow waves

- Widespread asynchronous slow waves in one or both hemispheres
  - more likely in acute lesions
  - may mask focal slow waves

- Bilaterally synchronous slow waves
  - distribution independent from focal slow waves
  - may indicate involvement of deep midline structures
EEG abnormalities associated with focal slow waves

- Focal epileptiform discharges
  - slow waves may be secondary to epileptiform activity
- Asymmetry of alpha rhythm
- Asymmetry of other physiologic activity (beta, mu, vertex, K complexes, sleep spindles)
Generalized asynchronous slow waves

- Reflect widespread structural damage or dysfunction, that includes subcortical white matter

- Clinical examples:
  - Widespread degenerative or cerebrovascular disease
  - Acute anoxia, postictal state
Generalized asynchronous slow waves

- the most common and least specific abnormal EEG pattern
- more reactive than focal slow waves
  - attenuated by eye opening and alerting
  - increased by relaxation and hyperventilation
- normal in drowsiness, normal in childhood
- seen in 10-15% of normal adults
Bilaterally synchronous slow waves

- May be due to abnormal interaction between the thalamus and the cortex (?overactive thalamocortical circuits)

- Clinical settings:
  - Metabolic, toxic and endocrine encephalopathies (ex: hepatic, renal encephalopathies)
  - Diffuse diseases involving subcortical + cortical grey matter (ex: Alzheimer's disease, PSP)
  - Local lesions in deep midline structures (ex: deep masses, hydrocephalus)
Bilaterally synchronous slow waves

- usually rhythmical and intermittent
  - may be sporadic
- may be generalized or restricted
- may be asymmetrical
- may be variable in field
- commonly have a frontal (FIRDA) or occipital maximum (OIRDA)
Bisynchronous slow waves
clinical significance

I normal
  – in drowsiness and sleep at any age
  – in wakefulness under 20
  – with hyperventilation

I abnormal
  – disorders of cerebral function (toxic/metabolic)
  – diffuse lesions of cortical and subcortical gray matter
  – deep midline lesions
## AMPLITUDE ABNORMALITIES

<table>
<thead>
<tr>
<th>Pattern</th>
<th>General pathologic correlates</th>
<th>Examples of specific conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal asymmetry</td>
<td>focal reduction in electrical activity</td>
<td>cortical infarct, contusion</td>
</tr>
<tr>
<td></td>
<td>- structural cortical damage</td>
<td>cortical ischemia, migraine</td>
</tr>
<tr>
<td></td>
<td>- disorder of cortical function</td>
<td>subdural hematoma</td>
</tr>
<tr>
<td></td>
<td>focal change in media between cortex and recording electrode</td>
<td>skull defect</td>
</tr>
<tr>
<td>Generalized changes</td>
<td>generalized reduction in electrical activity</td>
<td>Postanoxic encephalopathy,</td>
</tr>
<tr>
<td></td>
<td>- structural diseases of cerebral cortex</td>
<td>Huntington's chorea</td>
</tr>
<tr>
<td></td>
<td>- disorders of cortical function</td>
<td>Hypothyroidism, hypoxia, hypothermia, postictal, intoxications, anxiety</td>
</tr>
<tr>
<td></td>
<td>bilateral increase in media between cortex and recording electrode</td>
<td>Subdural hematoma</td>
</tr>
</tbody>
</table>
Amplitude asymmetries

- can be normal
  - photic driving, mu rhythm
  - beta (if < 35%), alpha (if ≤ 50%)

- abnormal
  - decrease (or increase) in all background activity
  - alpha rhythm, beta rhythm, HV response, sleep

- asymmetries may affect normal as well as abnormal EEG activity
Focal attenuation

- May reflect focal reduction in electrical activity
  - structural cortical damage (ex cortical infarct)
  - disorder of cortical function (ex: cortical ischemia, postictal attenuation)
- May reflect increased distance between cortex and recording electrode (ex subdural hematoma)
- Skull defect results in increased sharpness and amplitude of fast activity
Beta activity over a skull defect
Mu activity over a skull defect
Generalized amplitude changes

- May reflect generalized reduction in electrical activity
  - structural diseases of cerebral cortex (ex: postanoxic encephalopathy, Huntington's)
  - disorders of cortical function (ex: hypothyroidism, hypoxia, hypothermia, postictal, intoxications, anxiety)
- May reflect bilateral increase in media between cortex and recording electrode (ex: bilateral subdural hematoma)
Other abnormalities

- Increased fast activity
  - Most commonly seen in patients receiving sedatives.
- Unilateral failure of reactivity
  - Usually reflects parieto-temporal lesion
- Alpha coma pattern
- Spindle coma pattern
- Burst suppression pattern
- Periodic patterns
Unilateral failure of reactivity (Bancaud phenomenon)
Unilateral failure of reactivity

Female, Age: 48 yrs

T3-FP1

T4-FP2

FP1-F3

FP2-F4

C3-P3

C4-P4

P3-O1

P4-O2

Eyes closed: "100-7"

© Parietal astrocytoma
Coma
periodic patterns
The EEG in coma

- Often of great value
- Recordings frequently in the ICU
- Frequently contaminated with artifacts
- Additional electrodes (e.g. physiologic monitoring) may be needed
- Intervention during the EEG may be necessary for diagnostic (or therapeutic) reasons
The EEG in coma - reactivity

- Testing for reactivity essential
  - painful somatosensory & auditory stimuli
- Long recording without stimulation needed to assess spontaneous variability
- The presence of reactivity indicates a lighter level of coma (& favorable prognosis)
- Reactivity of lesser prognostic value in drug-induced encephalopathy
The EEG in coma—clinical value

- May distinguish the following possibilities:
  - Diffuse encephalopathy
  - Focal brain lesion
  - Non-convulsive or subtle convulsive status epilepticus
  - Psychogenic coma
- EEG non-specific in regards to etiology
- May be of prognostic value if the etiology is known, particularly with serial EEGs
The EEG in coma-
metabolic encephalopathy

• A metabolic cause is the most common in “coma of unknown etiology”
• There is progressive diffuse slowing of background rhythms with deepening level (from alpha to theta to delta)
• Generalized asynchronous slow activity
• Intermittent rhythmic delta activity may also be seen
The EEG in coma-hepatic encephalopathy

- Some patterns are suggestive of this specific etiology.
- Triphasic waves are common and are more likely to be associated with severe slowing of the background.
- Reye’s syndrome may have different EEG:
  - Triphasic waves uncommon
  - EEG correlates with level of consciousness
  - High incidence of 14- and 6-Hz positive spikes
The EEG in coma - triphasic waves

- Generally medium to high amplitude (100 to 300 μV) and 1.5-2.5 Hz, often in clusters
- Frequently frontally predominant
- Fronto-occipital lag may be present
- Bisynchronous, but may show shifting asymmetries
- N1 small and sharp, P1 largest component
- Not specific with respect to etiology
  - if patient awake, more likely non-metabolic
<table>
<thead>
<tr>
<th>Condition</th>
<th>Somnolent /confused</th>
<th>Stupor</th>
<th>Light coma</th>
<th>Deep Coma</th>
<th>Total pts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatic</td>
<td>18</td>
<td>0</td>
<td>8</td>
<td>2</td>
<td>28</td>
</tr>
<tr>
<td>Renal</td>
<td>7</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>Anoxic-hypoglycemic</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Hyperosmolar</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>
Age: 54 Yr

- FP1 - F3
- F3 - C3
- C3 - P3
- P3 - O1
- FP2 - F4
- F4 - C4
- C4 - P4
- P4 - O2
GENERALIZED MEDIUM AMPLITUDE DELTA
Encephalopathy (Reye's syndrome)—Deep Coma—Death

Fp1-A1

Fp2-A2

C3-A1

C4-A2

O1-A1

O2-A2

T3-A1

Face

20μV

1 sec.
The EEG in coma-renal encephalopathy

- Progressive increase in slow activity with superimposed slow bursts- BUN is blood parameter that best correlates with the EEG
- Triphasic waves seen in ~20%
- Paroxysmal/ epileptiform abnormalities may occur
- There may be a photoparoxysmal or photomyogenic response
- Dialysis disequilibrium syndrome and progressive dialysis encephalopathy may show specific changes
♀ Age: 30 Yr

Dialysis begins

On dialysis 3½ hrs

FP1-F3
F3-C3
C3-P3
P3-O1
FP2-F4
F4-C4
C4-P4
P4-O2
Age: 59 yr

Chronic renal failure
Confused and disoriented

100 µV
1 sec
♀ Age: 1 Yr

Water intoxication syndrome
Na = 112 Meq/L
The EEG in coma-drug intoxication

- Generalized fast activity suggests barbiturate or benzodiazepine intoxication
  - Beta activity slower than in awake patients (10-16 Hz)
  - Superimposed on a diffusely slow background
- A burst suppression pattern then electrocerebral silence are seen with deeper levels of coma
- Periodic discharges may be seen with baclofen and with lithium toxicity
- Spontaneous epileptiform discharges and PPR may be seen in delirium due to EtOH, barbiturate or BZD withdrawal (EEG may be near normal)
Progression of drug-induced coma
The EEG in coma—Anoxia

- Wide variety of abnormal EEG patterns
- Periodic patterns (particularly burst-suppression) carry a poor prognosis (96% die)
- Bursts/periodic discharges may correlate with myoclonic jerks or vertical eye movements
- Electrographic status epilepticus patterns may occur
- Triphasic waves seen in generally deeper level of coma than in hepatic encephalopathy
- Theta pattern coma (intermittent theta >anteriorly), alpha coma, spindle coma may be seen- the patterns are non-specific, but prognosis is worse with anoxia
GENERALIZED LOW AMPLITUDE DELTA
Ruptured Appendix—Septicemia and Anemia—Cardiorespiratory Arrest—Deep Coma—Death

Jaw

resp.

resp.

EKG

Fp2 — C4

C4 — O2

FP1 — C3

C3 — O1

T4 — Cz

Cz — T3

10μV

1sec.
Age: 77 Yr

T3-C3
C3-C2
C2-C4
C4-T4
T5-P3
P3-Pz
Pz-P4
P4-T6

Cardiac arrest 1 day ago
Patient died 3 days later
♂ Age: 24 Yr

Comatose
Cardiac arrest three days ago

Pinch left arm no response

50 μV
1 sec
The EEG in coma-supratentorial lesions

- EEG usually markedly abnormal
  - Abnormalities greater with rapidly expanding lesions
- Continuous focal polymorphic delta activity (PDA)
- FIRDA appears with involvement of deeper structures
- Periodic lateralized epileptiform discharges (PLEDS)
  - stroke, mass lesion, infection (herpes), anoxia
  - commonly associated with seizures (may be ictal)
- BIPLEDs
Several day history of fever, headache, and progressive obtundation
LATERALIZED PAROXYSMAL ABNORMALITY
Cerebral Vascular Accident—Focal Seizures—Respiratory Insufficiency—Coma—Temporary Improvement—Death

$F_p - F_8$

$F_8 - T_4$

$T_4 - T_6$

$T_6 - O_2$

$F_p - F_7$

$F_7 - T_3$

$T_3 - T_5$

$T_5 - O_1$

[50μV, 1 sec]
The EEG in coma-
subtentorial lesions

- EEG of lesser value
- alpha pattern coma with reactivity may be seen with lesions at or below the pontomesencephalic junction
  - more posterior, more variable, and more reactive than alpha coma in anoxic injury
  - should be distinguished from locked-in state (pontine tegmentum spared)
- Involvement of midbrain or diencephalon results in generalized delta activity (continuous or intermittent rhythmic)
The EEG in coma-
nonconvulsive status epilepticus

- EEG extremely useful- may be the only means of
  making the diagnosis
- A variety of patterns may be seen
- May be difficult to distinguish from periodic patterns of
  encephalopathy
- May be generalized or partial- a partial onset is often
difficult to identify
- IV administration of a benzodiazepine during EEG may
  be important for diagnosis
## Periodic EEG patterns

<table>
<thead>
<tr>
<th></th>
<th>PLEDs (LPDs)</th>
<th>PSI DDs</th>
<th>PLI DDs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intervals</strong></td>
<td>0.5-4 s</td>
<td>0.5-4 s</td>
<td>4-30 s</td>
</tr>
<tr>
<td><strong>Topography</strong></td>
<td>Unilateral or bil independent</td>
<td>Diffuse</td>
<td>Diffuse</td>
</tr>
<tr>
<td><strong>Etiology</strong></td>
<td>Varied</td>
<td>Metabolic; anoxia; toxic; CJD; NGSE</td>
<td>SSPE; toxic; anoxia</td>
</tr>
</tbody>
</table>
## Periodic EEG patterns

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Wave duration (seconds)</th>
<th>Interval duration (seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PLEDs</td>
<td>0.06-0.5</td>
<td>1-2</td>
</tr>
<tr>
<td>CJD</td>
<td>0.15-0.6</td>
<td>0.5-2</td>
</tr>
<tr>
<td>SSPE</td>
<td>0.5-3</td>
<td>3-20</td>
</tr>
<tr>
<td>Triphasic waves</td>
<td>0.2-0.5</td>
<td>0.5-2</td>
</tr>
<tr>
<td>Burst suppression</td>
<td>1-3</td>
<td>2-∞</td>
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### Table 1.—Causative Disorders

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of Patients</th>
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<tbody>
<tr>
<td></td>
<td>PLEDs*</td>
</tr>
<tr>
<td>Stroke (recent)</td>
<td>15</td>
</tr>
<tr>
<td>Seizure disorder</td>
<td></td>
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<tr>
<td>Chronic</td>
<td>10</td>
</tr>
<tr>
<td>Recent onset</td>
<td>4</td>
</tr>
<tr>
<td>Anoxic encephalopathy</td>
<td>3</td>
</tr>
<tr>
<td>CNS infection</td>
<td>2</td>
</tr>
<tr>
<td>Tumor</td>
<td>5</td>
</tr>
<tr>
<td>Craniotomy (recent)</td>
<td>3</td>
</tr>
<tr>
<td>Hepatic encephalopathy</td>
<td>1</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>1</td>
</tr>
<tr>
<td>Hypertensive encephalopathy</td>
<td>0</td>
</tr>
<tr>
<td>Hypoglycemic encephalopathy</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>45</td>
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### Table 2.—Clinical Features

<table>
<thead>
<tr>
<th></th>
<th>No. of Patients</th>
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<tr>
<td></td>
<td>PLEDs*</td>
</tr>
<tr>
<td></td>
<td>(N = 45)</td>
</tr>
<tr>
<td>Seizures</td>
<td></td>
</tr>
<tr>
<td>Focal</td>
<td>26</td>
</tr>
<tr>
<td>Generalized</td>
<td>6</td>
</tr>
<tr>
<td>Both</td>
<td>5</td>
</tr>
<tr>
<td>Focal neurologic deficits</td>
<td>37</td>
</tr>
<tr>
<td>Coma</td>
<td>11</td>
</tr>
</tbody>
</table>
Creutzfeld-Jacob disease
Bilateral jerking of the arms (1-4-73)

1 sec  40 µv
Discharges provoked by clapping

9-27
Creutzfeld-Jacob disease
Discharge Evolution - 4 weeks after first EEG - 3 days before death
SSPE
SSPE- compressed time base
SSPE- jerks associated with complexes
SSPE
SSPE- improvement with AED
Periodic complexes - Ketamine anesthesia
<table>
<thead>
<tr>
<th>Old</th>
<th>New</th>
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<tbody>
<tr>
<td>PLEDs</td>
<td>LPDs</td>
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<tr>
<td>BiPLEDs</td>
<td>BIPDs</td>
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<tr>
<td>PLEDs plus</td>
<td>LPDs + F</td>
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<tr>
<td>GPEDs</td>
<td>GPDs</td>
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<tr>
<td>Triphasic waves</td>
<td>GPDs, triphasic morphology</td>
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