Normal Adult EEG

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• No disclosures
Learning Objectives

• To recognize normal EEG patterns in wakefulness and sleep
Electroencephalogram (EEG)
EEG signal source: Dendritic currents

- Apical dendrite acts as a dipole
- Dendritic currents, not action potentials, summate best
- Electrical signal is strongly attenuated at the scalp.
- Inhibitory neuron populations do not produce good dipoles
Referential Montage: absolute voltage

Bipolar Montage: Phase reversal
Awake EEG
Posterior Dominant Rhythm (PDR)/Alpha Rhythm

• 8-12.99 Hz

• Typical amplitude 40-50uV in adults (higher in children)

• Not present up to 10% in healthy individuals

• absent when blindness has been present since early in life

• Morphology: sinusoidal
PDR

• First develops at 3-4 months (4 Hz); 5-6 Hz by 1 year; 8 Hz by 3 years

• Present over the posterior head regions in a state of relaxed wakefulness with eyes closed

• It blocks/attenuated with drowsiness, concentration, stimulation, or visual fixation

• Alpha rhythm blocking – decrease with aging
Abnormal PDR

• PDR with frequencies less than 8.5 Hz is considered as abnormal in adults regardless of age

• Slow PDR is a sign of cerebral dysfunction
Alpha Squeak

• A brief, high frequency 0.5-1 second after eye closure

• Therefore, frequency assessment should not include the first 0.5-1 second after eye closure
Slow Alpha Variant

• Subharmonic of the alpha rhythm (fusion of adjacent waves)
Fast Alpha Variant

• A harmonic of the alpha rhythm and usually has frequency between 16-20 Hz
PDR

• PDR’s distribution always includes the occipital, and commonly extends to posterior temporal and occipital parietal regions. Extension should be symmetric.

• When distribution is asymmetric, the larger field is abnormal

• Frequency asymmetries between the sides should be <1 Hz

• Amplitude asymmetry is common (usually higher on right – related to skull thickness), but rarely >20uV
PDR – During Drowsiness

• PDR disappears during drowsiness
  • Decrease in amplitude
  • Extension of the field anteriorly
  • Loss of clear sinusoidal morphology
  • Inclusion of more theta rhythm

• With arousal from drowsiness, PDR sometimes briefly present with a frontal predominance
PDR – Clinical Significance

• Likely related to the rhythmic cellular interactions between occipital/some parietal cortex and the pulvinar region of the thalamus

• Functions as an active stand-by state

• Fever and hypermetabolic states (hyperthyroidism, amphetamine intoxication) – may increase PDR frequency

• Slowing of PDR commonly seen in encephalopathy and hypoperfusion
The relationship of cerebral blood flow to electroencephalogram (EEG) and pathophysiology. ATP, adenosine triphosphate (CBF).

Data from [2, 4].
PDR – Clinical Significance

• Complete absence of blocking or unilateral block is abnormal (usually due to a structural abnormality – occipital or a pontine lesion, or transiently with migraines)

• Unilateral blocking - “Bancaud’s phenomenon” (the side lacking the blocking response is abnormal)
Absence of PDR blocking
Beta Rhythm

- Frequency: >13 Hz
- Amplitude: <20uV
- Frontocentral beta – sign of drowsiness or sleep onset
- In drowsiness (N1) through stage II (N2) NREM sleep; frontocentral
- Occurs as a burst, amplitude ~60uV
Generalized Beta – Excessive Beta

- Sedative medications: Benzodiazepines, barbiturates are most potent producers
- Others: Chloral hydrate, neuroleptics, phenytoin, cocaine, amphetamine
- Anxiety and hyperthyroidism
Lorazepam

propofol
Theta Rhythm

- Frequency: 4-7.99 Hz
- Morphology: sinusoidal > arciform;
- Drowsiness (N1)
- Location: Midline maximum Cz
Generalized and Focal theta

• Generalized theta – encephalopathy (nonspecific)

• Focal theta – focal cerebral/cortical dysfunction
Polymorphic gen theta - encephalopathy
Delta

• 0-3.99 Hz
• Normal: Polymorphic delta activities – generalized, normal in sleep, mostly stage III (slow wave sleep) > II
• Abnormal
  • Generalized - diffuse encephalopathy
  • Focal – focal cortical/cerebral dysfunction
  • Rhythmic delta activities (RDA) – when focal, may suggest increased risk for seizures
RDAs vs. LPDs

No interdischarge interval

Interdischarge interval
Figure. Model of Pattern Characteristics and Seizure Risk

Seizure Risk vs. Pattern Frequency, Hz

- LPD
- LPD+
- LRDA+, GPD+
- LRDA, GPD
- GRDA, GRDA+

Significant Risk
Hyperventilation
Hyperventilation (HV)

• Cerebral hypocapnia → vasoconstriction/cerebral hypoxia

• HV response depends on several factors: Effort, age, posture, blood sugar

• Younger individuals – largest response

• Response – generalized rhythmic delta slowing
8 yo boy misdiagnosed with absence epilepsy
Prominent Hyperventilation Response in an Adult

F 28

[Graph showing EEG recordings with labeled channels: Fp1-F3, F3-C3, C3-P3, P3-O1, Fp2-F4, F4-C4, C4-P4, P4-O2. Each channel shows waveforms over a 1 second period, with a scale of 100 µV.]
Abnormal HV Response

- Epileptiform discharges
- Seizures
- Focal slowing
- Asymmetry
- Prolonged slowing beyond 90 seconds post-HV
Photic Stimulation Response

• Visual stimulation with a flashing strobe produces sharply contoured, positive, monophasic transients in bilateral occipital (may extend to posterior temporal)

• Best elicited with the eyes closed or with stimulation using red light

• Photic driving response amplitude is maximum with stimulation frequencies close to PDR

• Maturation: 3 months
Photoparoxysmal Response (Abnormal)
Normal Sleep
Drowsiness (N1)
Drowsiness

- Decrease mini blinks
- Slowing of PDR
- Increase slow roving eye movements
- Low amplitude, mixed frequencies
Drowsiness – mixed frequencies

- Dropout of alpha
- Theta slowing
- Bursts of theta
- Temporal slow waves
- Increase in beta activity
Slowing of the Background with Drowsiness

F 21

Fp1-F7
F7-T3
T3-T5
T5-O1
Fp2-F8
F8-T4
T4-T6
T6-O2

Drowsy

1 sec 50 µV
Drowsiness

F 50

F_{P1}-F_3
F_3-C_3
C_3-P_3
P_3-O_1
F_{P2}-F_4
F_4-C_4
C_4-P_4
P_4-O_2

1 sec
30 μV
Increased Beta with Drowsiness
Positive Occipital Sharp Transients of Sleep (POSTS)

- Mostly stage I (N1), but may persist into stage III (N3)
- Most frequently seen during the first 30 minutes after sleep onset
- Positive polarity sharp waves with phase reversal at either O1 or O2
- Morphology and phase: Triangular, monophasic or diphasic
- Amplitude: 20-75uV
- Maturation: 3 years
Vertex Sharp Transients (Vertex Waves)

- Phase reversal at or near vertex/midline, central
- Drowsiness and NREM sleep
- Often triphasic
- 100ms
- Singlets or in trains (rarely longer than few seconds)
- Maturation: 5-6 months
Stage II (N2) Sleep: Spindles, K complexes

Signs of N2:

- **K complex**: well-delineated, negative, sharp waves immediately followed by a positive component, ≥0.5s, max in amp in frontal; Often immediately precedes a sleep spindle; maturation - 5 months

- **Sleep spindles**: 11-16 Hz, ≥0.5s, max in central regions/parasagittal midline; first appear at 2 months
Sleep Spindles
K complexes
Rapid Eye Movement (REM) Sleep – Stage R

- REM: eye movements with conjugate, irregular, sharply peaked eye movements with an initial deflection usually <500 ms
- Sawtooth waves: Trains of sharply contoured triangular, 2-6 Hz waves maximum in amplitude over central head, often, but not always, preceded a burst of rapid eye movements
- Low amplitude, mixed frequencies
- No spindles or K complexes
FIGURE 15. REM and sawtooth waves.
REM sleep with Sawtooth Waves
Stage III (N3) – Slow Wave Sleep

- In AASM staging:
  
  Slow wave activity: 0.5-2 Hz and peak-to-peak amplitude >75 uV
  
  Score N3 when ≥20% epoch consists of slow wave activity
EEG

• Identify normal patterns

• Not to misinterpret them as abnormal activity
Questions?