NeuroTrace Academy Study Guide

Category: Domain III - Pattern Recognition
Topic: Encephalopathy Differentials & EEG Patterns
Style: Differential diagnosis, pattern-based, clinical correlation

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1. Core Principle

Encephalopathy = Diffuse Brain Dysfunction

EEG patterns in encephalopathy reflect the **severity** and **underlying cause** of diffuse cerebral dysfunction. Understanding the differential helps technologists recognize patterns and guide clinical interpretation.

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2. Encephalopathy Classification by Etiology

A. Metabolic Encephalopathies

Hepatic Encephalopathy

- **EEG Pattern:**
  - **Early:** Diffuse slowing (theta range)
  - **Moderate:** Triphasic waves (classic finding)
  - **Severe:** Burst suppression, alpha coma pattern
- **Key Features:**
  - Triphasic waves are **highly characteristic** (but not pathognomonic)
  - Frontal predominance
  - Reactivity may be preserved early
- **Clinical Context:** Liver failure, elevated ammonia
- 🔑 **Exam Pearl:** Triphasic waves in hepatic encephalopathy are typically frontal-predominant and may be reactive to stimulation

Uremic Encephalopathy

- **EEG Pattern:**
  - Diffuse slowing (theta/delta)
  - May show triphasic waves (less common than hepatic)
  - Background disorganization
- **Key Features:**
  - Less specific than hepatic encephalopathy
  - Slowing correlates with BUN/creatinine
- **Clinical Context:** Renal failure, elevated BUN/creatinine

Hypoglycemic Encephalopathy

- **EEG Pattern:**
  - Diffuse slowing
  - May show epileptiform activity
  - Background attenuation
- **Key Features:**
  - Rapid onset
  - May be reversible with glucose
- **Clinical Context:** Low blood glucose, diabetic patients

Hyperglycemic Encephalopathy

- **EEG Pattern:**
  - Diffuse slowing
  - May show triphasic waves
- **Key Features:**
  - Less specific pattern
- **Clinical Context:** Diabetic ketoacidosis, hyperosmolar state

Hyponatremia/Hypernatremia

- **EEG Pattern:**
  - Diffuse slowing
  - Background disorganization
- **Key Features:**
  - Severity correlates with degree of electrolyte imbalance
- **Clinical Context:** Electrolyte disturbances

B. Toxic Encephalopathies

Alcohol Withdrawal

- **EEG Pattern:**
  - Diffuse slowing
  - May show beta activity (enhanced)
  - Photoparoxysmal response
- **Key Features:**
  - Enhanced beta is common
  - May show withdrawal seizures
- **Clinical Context:** Recent alcohol cessation, delirium tremens

Benzodiazepine Withdrawal

- **EEG Pattern:**
  - Diffuse slowing
  - Enhanced beta (during use)
  - Beta withdrawal (during withdrawal)
- **Key Features:**
  - Beta enhancement during use
  - Slowing during withdrawal
- **Clinical Context:** Benzodiazepine dependence, withdrawal

Anticonvulsant Toxicity

- **EEG Pattern:**
  - Diffuse slowing
  - Background suppression
  - May show paradoxical increase in epileptiform activity
- **Key Features:**
  - Phenytoin: cerebellar signs + slowing
  - Valproate: hepatic effects + slowing
- **Clinical Context:** High drug levels, toxicity symptoms

Lithium Toxicity

- **EEG Pattern:**
  - Diffuse slowing
  - Triphasic waves (may occur)
- **Key Features:**
  - Less specific pattern
- **Clinical Context:** High lithium levels, psychiatric patients

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C. Hypoxic-Ischemic Encephalopathy (HIE)

Mild HIE

- **EEG Pattern:**
  - Diffuse slowing (theta)
  - Background reactivity preserved
- **Key Features:**
  - Reversible if treated early
  - Good prognosis if reactivity maintained
- **Clinical Context:** Brief hypoxia, early intervention

Moderate HIE

- **EEG Pattern:**
  - Diffuse slowing (delta)
  - Loss of reactivity
  - Background disorganization
- **Key Features:**
  - Poorer prognosis
  - May progress to severe
- **Clinical Context:** Prolonged hypoxia, cardiac arrest

Severe HIE

- **EEG Pattern:**
  - **Burst suppression** (classic)
  - **Alpha coma pattern** (paradoxical)
  - **Isoelectric/flat EEG** (worst)
- **Key Features:**
  - Burst suppression: alternating bursts and suppression
  - Alpha coma: alpha frequency but non-reactive (poor prognosis)
  - Isoelectric: minimal/no activity
- **Clinical Context:** Severe anoxia, cardiac arrest, poor prognosis
- 🔑 **Exam Pearl:** Alpha coma pattern shows alpha frequency but is non-reactive and indicates severe brainstem dysfunction with poor prognosis

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D. Infectious Encephalopathies

Viral Encephalitis

- **EEG Pattern:**
  - Diffuse slowing
  - Focal or multifocal slowing (if focal involvement)

    - May show periodic patterns (e.g., HSV: PLEDs)
- **Key Features:**
    - HSV: Periodic lateralized epileptiform discharges (PLEDs)
    - Diffuse involvement: generalized slowing
- **Clinical Context:** Fever, altered mental status, viral infection

Bacterial Meningitis/Encephalitis

- **EEG Pattern:**
    - Diffuse slowing
    - Background suppression
    - May show epileptiform activity
- **Key Features:**
    - Severity correlates with clinical state
- **Clinical Context:** Fever, meningeal signs, infection

Prion Disease (CJD)

- **EEG Pattern:**
    - **Periodic sharp wave complexes** (PSWCs) - classic
    - 1-2 Hz periodic discharges
    - Background slowing
- **Key Features:**
    - PSWCs are **highly characteristic** (but not pathognomonic)
    - Bilateral, synchronous
    - 1-2 Hz frequency
- **Clinical Context:** Rapidly progressive dementia, myoclonus
- 🔑 **Exam Pearl:** Periodic sharp wave complexes (PSWCs) at 1-2 Hz are highly characteristic of CJD but can occur in other conditions

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E. Autoimmune/Inflammatory Encephalopathies

Autoimmune Encephalitis (e.g., Anti-NMDA)

- **EEG Pattern:**
    - Diffuse slowing
    - Extreme delta brush (characteristic of anti-NMDA)
    - May show epileptiform activity
- **Key Features:**
    - Extreme delta brush: rhythmic delta with superimposed beta
    - May be focal or generalized
- **Clinical Context:** Psychiatric symptoms, movement disorders, seizures

Limbic Encephalitis

- **EEG Pattern:**
    - Temporal slowing (focal)
    - May show temporal epileptiform activity
- **Key Features:**
    - Focal temporal involvement
    - May be unilateral or bilateral
- **Clinical Context:** Memory loss, temporal lobe symptoms

F. Structural/Traumatic Encephalopathies

Traumatic Brain Injury (TBI)

- **EEG Pattern:**
  - Diffuse slowing
  - Focal slowing (if focal injury)
  - May show epileptiform activity
- **Key Features:**
  - Pattern depends on injury location and severity
- **Clinical Context:** Head trauma, altered mental status

Subarachnoid Hemorrhage (SAH)

- **EEG Pattern:**
  - Diffuse slowing
  - May show focal slowing (if vasospasm)
- **Key Features:**
  - Severity correlates with clinical state
- **Clinical Context:** Sudden headache, altered mental status

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3. EEG Pattern-Based Differential Table

EEG Pattern	Primary Associations	Secondary Associations	Key Distinguishing Features
Triphasic Waves	Hepatic encephalopathy	Uremic, hyperglycemic, lithium toxicity	Frontal-predominant, may be reactive
Burst Suppression	Severe HIE, deep anesthesia	Severe metabolic, toxic	Alternating bursts and suppression
Alpha Coma	Severe HIE (brainstem)	Severe anoxia	Alpha frequency but non-reactive (poor prognosis)
Periodic Sharp Wave Complexes (PSWCs)	CJD (Creutzfeldt-Jakob)	Other prion diseases	1-2 Hz, bilateral, synchronous
Extreme Delta Brush	Anti-NMDA encephalitis	Other autoimmune encephalitis	Rhythmic delta with superimposed beta
Diffuse Slowing	All encephalopathies (early)	Metabolic, toxic, infectious	Non-specific, severity-dependent
FIRDA	Encephalopathy (adults)	Deep midline lesions	Rhythmic frontal delta
OIRDA	Encephalopathy (children)	Posterior dysfunction	Rhythmic occipital delta
PLEDs	HSV encephalitis	Focal structural lesions	Periodic lateralized discharges

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4. Severity-Based Pattern Progression

Mild Encephalopathy

- **Pattern:** Diffuse theta slowing
- **Reactivity:** Preserved
- **Background:** Organized
- **Prognosis:** Good (if reversible)

Moderate Encephalopathy

- **Pattern:** Diffuse delta slowing
- **Reactivity:** Reduced or absent
- **Background:** Disorganized
- **Prognosis:** Variable

Severe Encephalopathy

- **Pattern:** Burst suppression, alpha coma, or isoelectric
- **Reactivity:** Absent
- **Background:** Severely abnormal
- **Prognosis:** Poor

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5. High-Yield Exam Patterns

Triphasic Waves

- **Classic Association:** Hepatic encephalopathy
- **Morphology:** Three-phase waveform (negative-positive-negative)
- **Location:** Frontal-predominant
- **Frequency:** 1.5-2.5 Hz
- **Reactivity:** May be reactive to stimulation
- 🔑 **Exam Pearl:** Triphasic waves are highly characteristic of hepatic encephalopathy but can occur in other metabolic encephalopathies

Burst Suppression

- **Classic Association:** Severe HIE, deep anesthesia
- **Morphology:** Alternating bursts of activity and suppression
- **Prognosis:** Poor (if persistent)
- 🔑 **Exam Pearl:** Burst suppression indicates severe cortical dysfunction; persistent pattern has poor prognosis

Alpha Coma Pattern

- **Classic Association:** Severe HIE with brainstem involvement
- **Morphology:** Alpha frequency (8-13 Hz) but non-reactive
- **Prognosis:** Very poor
- 🔑 **Exam Pearl:** Alpha coma pattern shows alpha frequency but is non-reactive, indicating severe brainstem dysfunction with poor prognosis

Periodic Sharp Wave Complexes (PSWCs)

- **Classic Association:** CJD
- **Morphology:** Periodic sharp waves at 1-2 Hz
- **Location:** Bilateral, synchronous
- 🔑 **Exam Pearl:** PSWCs at 1-2 Hz are highly characteristic of CJD but can occur in other prion diseases

Extreme Delta Brush

- **Classic Association:** Anti-NMDA receptor encephalitis
- **Morphology:** Rhythmic delta with superimposed beta
- **Location:** May be focal or generalized

- 🔑 **Exam Pearl:** Extreme delta brush is characteristic of anti-NMDA receptor encephalitis

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6. Clinical Correlation Guide

When You See Triphasic Waves

1. **First Consider:** Hepatic encephalopathy
2. **Check:** Liver function tests, ammonia level
3. **Also Consider:** Uremic, hyperglycemic, lithium toxicity
4. **Key:** Frontal-predominant, may be reactive

When You See Burst Suppression

1. **First Consider:** Severe HIE, deep anesthesia
2. **Check:** History of cardiac arrest, anoxia, anesthesia
3. **Also Consider:** Severe metabolic or toxic encephalopathy
4. **Key:** Alternating bursts and suppression, poor prognosis if persistent

When You See Alpha Coma Pattern

1. **First Consider:** Severe HIE with brainstem involvement
2. **Check:** History of anoxia, cardiac arrest
3. **Key:** Alpha frequency but non-reactive = poor prognosis

When You See PSWCs

1. **First Consider:** CJD
2. **Check:** Rapidly progressive dementia, myoclonus
3. **Key:** 1-2 Hz periodic sharp waves, bilateral

When You See Extreme Delta Brush

1. **First Consider:** Anti-NMDA receptor encephalitis
2. **Check:** Psychiatric symptoms, movement disorders
3. **Key:** Rhythmic delta with superimposed beta

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7. ABRET Exam Pearls

1. **Triphasic waves** are highly characteristic of hepatic encephalopathy but can occur in other metabolic encephalopathies.
2. **Burst suppression** indicates severe cortical dysfunction; persistent pattern has poor prognosis.
3. **Alpha coma pattern** shows alpha frequency but is non-reactive, indicating severe brainstem dysfunction with poor prognosis.
4. **PSWCs at 1-2 Hz** are highly characteristic of CJD but can occur in other prion diseases.
5. **Extreme delta brush** is characteristic of anti-NMDA receptor encephalitis.
6. **FIRDA** is more common in adults with encephalopathy; **OIRDA** is more common in children.
7. **Diffuse slowing** is non-specific and occurs in all types of encephalopathy; severity correlates with clinical state.

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8. Quick Reference Summary

Metabolic Encephalopathies

- **Hepatic:** Triphasic waves (classic), diffuse slowing
- **Uremic:** Diffuse slowing, less specific
- **Hypoglycemic:** Diffuse slowing, may show epileptiform
- **Hyperglycemic:** Diffuse slowing, triphasic waves (less common)

Toxic Encephalopathies

- **Alcohol withdrawal:** Enhanced beta, diffuse slowing
- **Benzodiazepine:** Beta enhancement (use) or slowing (withdrawal)
- **Anticonvulsant toxicity:** Diffuse slowing, background suppression

Hypoxic-Ischemic Encephalopathy

- **Mild:** Theta slowing, preserved reactivity
- **Moderate:** Delta slowing, lost reactivity
- **Severe:** Burst suppression, alpha coma, isoelectric

Infectious Encephalopathies

- **Viral (HSV):** PLEDs, diffuse slowing
- **Bacterial:** Diffuse slowing, background suppression
- **CJD:** PSWCs (1-2 Hz), highly characteristic

Autoimmune Encephalopathies

- **Anti-NMDA:** Extreme delta brush (characteristic)
- **Limbic:** Temporal slowing, temporal epileptiform

Next Steps:

- Memorize classic pattern associations (triphasic waves = hepatic, PSWCs = CJD, etc.)
- Understand pattern progression (mild → moderate → severe)
- Learn distinguishing features for each pattern
- Practice clinical correlation with EEG findings