3.8.8. Akshepaka (Seizures)

3.8.8. Ākṣepaka (Neonatal Seizures)

Learning goals

By the end of this chapter you will be able to: (i) define neonatal seizures in line with the ILAE neonatal framework; (ii) recognise common etiologies and clinical patterns in neonates; (iii) run a step-wise **acute management** (ABC, correct reversible metabolic triggers, antiseizure medication); (iv) outline **investigations** (EEG/aEEG, labs, neuroimaging) and criteria for stopping/continuing ASMs; and (v) relate the condition to **Āyurvedic nosology (Ākṣepaka)** without misapplying adult measures to neonates.

Classical anchor (Āyurveda)

Textual description of Ākṣepaka (convulsions/spasms)—Charaka describes violent, repeated drawing/jerking of the limbs when aggravated **Vāta** contracts the tendons/ligaments:

> "हनुग्रहं च संस्तभ्य हनुं(नू) संवृतवक्त्रताम् ।
> मुहुराक्षिपति क्रुद्धो गात्राण्याक्षेपको ऽनिलः ॥" — *Caraka Saṃhitā*, **Cikitsāsthāna 28/50**.

Relevance: In neonates, such involuntary stereotyped jerks or tonic posturing equate clinically to **seizures**, but management must follow modern neonatal protocols (gentle, physiology-aligned), not adult vātavyādhi measures.

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1) What is a neonatal seizure?

The **ILAE neonatal position** defines neonatal seizures as **electrographic events** with a sudden, paroxysmal abnormal EEG activity; many are **electrographic-only** (no clear clinical sign). Classification is **EEG-anchored**; purely clinical events **without EEG correlate** are not labelled seizures in this framework. Types are grouped by predominant feature: **motor** (automatisms, clonic, tonic, myoclonic, epileptic spasms), **non-motor** (autonomic, behavioral arrest), or **sequential**.

Exam tip: In neonates, most events have **focal onset**; division into focal/generalised is less useful, and **EEG/aEEG** is central to diagnosis.

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2) Why do neonates seize? (etiology & pathophysiology)

- **Hypoxic–ischemic encephalopathy (HIE)**—the commonest cause in term neonates; seizures cluster in the first 24–72 h.
- **Ischaemic stroke** and **intracranial haemorrhage** (especially in preterms).
- **Metabolic**: **hypoglycaemia**, **hypocalcaemia**, **hypomagnesaemia**, **sodium** disturbances.
- **CNS infection** (meningitis/encephalitis), **structural malformations**, **inborn errors of metabolism**, **drug withdrawal/intoxication**.

Neonatal cortex is **hyperexcitable** (high NMDA/low GABA inhibition), predisposing to acute symptomatic seizures after brain insult.

3) How do they present? (recognition & differentials)

Patterns you may see

- **Clonic**: rhythmic jerks of a limb/face; often focal.
- **Tonic**: sustained posturing (focal or axial).
- **Myoclonic**: brief shock-like jerks.
- **Automatisms**: chewing, bicycling, ocular deviation.
- **Autonomic**: apnoea, tachycardia, BP/oxygen changes.

Differentiate from jitteriness/tremor: jitteriness is **stimulus-sensitive**, **suppressed by limb restraint**, and lacks ocular deviation/autonomic change; seizures are **stereotyped**, often **not stimulus-sensitive**, and may have **EEG correlate**. **Electrographic-only** seizures are frequent—hence need for EEG/aEEG.

Generalised Seizure 3D model
Seizure Nerve Impulse 3D model

Causes of Seizures

Cause	Examples
High fever	Heatstroke, Infections
Brain infections	Abscess, HIV, Malaria, Meningitis, Rabies, Syphilis, Tetanus, Toxoplasmosis, Viral encephalitis
Metabolic disorders	High blood levels of glucose (hyperglycemia) or sodium, Low blood levels of glucose (hypoglycemia), calcium, magnesium, or sodium
Other disorders	Kidney failure or liver failure, which can lead to dysfunction of the brain (encephalopathy), Vitamin B6 deficiency (in newborns)
Inadequate oxygen supply to the brain	Abnormal heart rhythms, Cardiac arrest, Carbon monoxide poisoning, Near drowning, Near suffocation, Stroke, Vasculitis
Structural damage to the brain	Brain tumor (noncancerous or cancerous), Head injury, Hydrocephalus, Intracranial hemorrhage (bleeding within the skull), Stroke
Abnormalities present or occurring at birth, including genetic disorders	Birth defect, Hereditary metabolic disorders, such as Tay-Sachs disease or phenyl ketonuria, Injury during birth
Fluid accumulation in the brain (cerebral edema)	Eclampsia, Hypertensive encephalopathy
Prescription medications*	Buspirone (used to treat anxiety disorders), Camphor*, Cefepime (an antibiotic)*, Chlorpromazine (used to treat schizophrenia), Ciprofloxacin (an antibiotic), Chloroquine (used to treat malaria), Clozapine (usually used to treat schizophrenia), Cyclosporine (used to prevent and treat rejection of organ transplants), Imipenem (an antibiotic)*, Indomethacin (used to relieve pain and reduce inflammation)*, Meperidine (used to relieve pain)*, Phenytoin†, Theophylline (used to treat asthma and other airway disorders), Tricyclic antidepressants
Illicit drugs	Amphetamines, Cocaine (overdose)
Withdrawal of a medication or substance after heavy use	Alcohol, General anesthetics (used during surgery), Sedatives, including sleep aids
Exposure to toxins	Lead, Strychnine

Cause	Examples

* Various medications can cause seizures if too much is taken. In some people, certain medications can make seizures more likely to occur by making nerve cells in the brain easier to stimulate. These medications are said to lower the seizure threshold.

† Phenytoin, used to treat seizure disorders, can cause seizures if too much is taken.

Manifestations of Focal-Onset Seizures by Site

Focal Manifestation	Site of Dysfunction
Bilateral tonic posture	Frontal lobe (supplementary motor cortex)
Simple movements (eg, limb twitching, jacksonian march)	Contralateral frontal lobe
Head and eye deviation with posturing	Supplementary motor cortex
Abnormal taste sensation (dysgeusia)	Insula
Visceral or autonomic abnormalities (eg, epigastric aura, salivation)	Insular-orbital-frontal cortex
Olfactory hallucinations	Anteromedial temporal lobe
Chewing movements, salivation, speech arrest	Amygdala, opercular region
Complex behavioral automatisms	Temporal lobe
Unusual behavior suggesting a psychiatric cause or sleep disorder	Frontal lobe
Visual hallucinations (formed images)	Posterior temporal lobe or amygdala-hippocampus
Localized sensory disturbances (eg, tingling or numbness of a limb or half the body)	Parietal lobe (sensory cortex)
Visual hallucinations (unformed images)	Occipital lobe

Clinical Clues to the Causes of Symptomatic Seizures

Finding	Possible Cause
Fever and stiff neck	Meningitis, Subarachnoid hemorrhage, Meningoencephalitis
Papilledema	Increased intracranial pressure
Loss of spontaneous venous pulsations (noted during funduscopy)	Increased intracranial pressure (specificity is 80–90%*)
Focal neurologic defects (eg, asymmetry of reflexes or muscle strength)	Structural brain abnormality (eg, tumor, stroke)Postictal paralysis
Generalized neuromuscular irritability (eg, tremulousness, hyperreflexia)	Medication toxicity (eg, sympathomimetics)Withdrawal syndromes (eg, of alcohol or sedatives)Certain metabolic disorders (eg, hypocalcemia, hypomagnesemia)
Skin lesions (eg, axillary freckling or café-au-lait spots, hypomelanotic skin macules, shagreen patches)	Neurocutaneous disorders (eg, neurofibromatosis, tuberous sclerosis)

* Spontaneous venous pulsations are absent in all patients with increased intracranial pressure; these pulsations are also absent in 10–20% of people with normal intracranial pressure, but sometimes only temporarily.

4) First minutes: Stabilise and search for the reversible

A. ABC + glucose

- Airway, breathing, circulation, temperature.
- **Check glucose immediately**. If **<45 mg/dL**, give **D10W 2 mL/kg IV bolus**, then infusion and recheck; manage per hypoglycaemia protocol (see 3.8.7).

B. Correct electrolytes if abnormal or strongly suspected

- **Hypocalcaemia**: give **10% calcium gluconate 1–2 mL/kg IV slowly** over 5–10 min with ECG monitoring; may continue with infusion depending on severity.
- **Hypomagnesaemia**: **magnesium sulfate 25–50 mg/kg IV** over 2–4 h; some neonatal monographs allow **~100 mg/kg (0.4 mmol/kg)** in severe cases.

- **Sodium** disorders: correct carefully as per unit protocol.
- **Suspected sepsis**: start antibiotics after cultures as indicated.

C. If still seizing or high suspicion of electrographic seizures → move to ASM (below), **do not delay** EEG placement.

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5) Antiseizure medication (ASM): evidence-based steps

First-line (ILAE 2023): Phenobarbital

- **Loading**: **20 mg/kg IV**.
- If seizures persist (EEG/clinical), give **additional 10–20 mg/kg IV** (up to ~**40 mg/kg** cumulative).
- **Maintenance**: ~**5 mg/kg/day**. Phenobarbital was **more effective** than levetiracetam as initial therapy (EEG-verified control **80% vs 28%** in a key RCT). Monitor for **respiratory depression, hypotension**.

Second-line options (choose one; consider comorbidities):

- **Fosphenytoin/Phenytoin**: **20 mg/kg PE IV**; maintenance ~**5 mg/kg/day**, cardiac monitoring.
- **Levetiracetam**: **40 mg/kg IV load**, then an **extra 20 mg/kg** if needed; maintenance **40–60 mg/kg/day** in 3 doses; generally well-tolerated.

Refractory (after two ASMs):

- **Lidocaine infusion** (avoid if phenytoin used or cardiac disease).
- **Midazolam**: **0.05–0.15 mg/kg bolus → infusion 1 µg/kg/min**, titrate up to **5 µg/kg/min**; watch for hypotension/respiratory depression.

Vitamin-responsive epilepsies: don't miss

- If seizures remain refractory or phenotype suggests metabolic/early-onset epilepsy, consider supervised **pyridoxine** trial (**100 mg IV once**, ventilation ready), then **30 mg/kg/day** for 3–5 days if responsive; consider **pyridoxal-5′-phosphate** for suspected **PNPO deficiency**.

Stopping ASMs: For **acute provoked** neonatal seizures with no neonatal-onset epilepsy, many centres stop ASMs after **~72 h seizure-freedom** and normalising evaluation prior to discharge—individualise with neurology and EEG.

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6) Investigations: what to send and when

Bedside/urgent labs (start while treating): **Glucose**, **electrolytes** (Na/K/Cl/HCO_3^-), **ionised Ca**, **Mg**, **blood gas**, **CBC/CRP**, **blood/CSF cultures** if infection suspected. **Ammonia**, **lactate**, ± **urine organic acids/serum amino acids** if no obvious cause.

Neurophysiology:

- **EEG** is the **gold standard** to confirm electrographic seizures and guide titration; **aEEG** provides bedside screening/monitoring if full EEG is not immediately available.

Imaging:

- **Cranial ultrasound** in preterms; **MRI** (ideal) to characterise HIE, stroke, malformations; **CT** if haemorrhage is suspected and MRI unavailable/unstable infant.

Metabolic/genetic work-up in refractory/unexplained cases (consider **pyridoxine/PLP** pathway defects, **KCNQ2/3** channelopathies).

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7) Putting it together: a practical algorithm

1. **Stabilise** (airway, breathing, circulation, temperature).
2. **Check glucose**; if low → **D10W 2 mL/kg IV** + infusion; recheck.
3. **Draw labs** and **correct electrolytes** (Ca, Mg, Na) as indicated.
4. If ongoing seizures or high suspicion: **Phenobarbital 20 mg/kg IV**, repeat **10–20 mg/kg** if needed (max ~40 mg/kg).
5. If persistent: choose **fosphenytoin/phenytoin 20 mg/kg PE** *or* **levetiracetam 40 (+20) mg/kg**.
6. **Refractory**: consider **midazolam infusion** or **lidocaine** (avoid with phenytoin), and **pyridoxine 100 mg IV** trial under monitoring.
7. **EEG/aEEG-guided** monitoring throughout; treat the **underlying etiology** (HIE protocols including TH where appropriate, antibiotics for sepsis, etc.).
8. **Plan for ASM discontinuation** in acute symptomatic cases once safe, with follow-up.

8) Documentation, counselling, and follow-up

- Record **time-locked events**, vitals, **EEG changes**, doses and responses, lab values, imaging findings, and conversations with family.
- **Family counselling** before discharge: cause, recurrence risk, first aid, red flags, and follow-up plan (neurology/developmental clinic).

9) Kaumārabhṛtya alignment

- Correlate neonatal seizures with **Ākṣepaka** (Vāta-pradhāna movement disorder) as per Charaka's description of repeated jerks due to **snāyu/kaṇḍarā saṅkocha** (tendon/ligament contraction) by provoked Vāta—**but** state that neonates require **mṛdu** (gentle), evidence-based measures: warmth, euglycaemia, electrolyte balance, oxygenation, and modern ASMs/EEG.
- Emphasise **stanya-poshana** and thermal protection in preventing metabolic triggers (e.g., hypoglycaemia, cold stress)—these themes align with classical bāla-rakṣaṇa, while actual seizure therapy follows contemporary neonatal guidelines.

Self-assessment

A. MCQs (one best answer)

1. According to **ILAE (2021)**, which statement is **true** for neonatal seizures?
   A. Clinical events without EEG correlate are seizures
   B. **Electrographic confirmation is central; many seizures have no clinical correlate**
   C. All neonatal seizures are generalised
   D. EEG is optional for diagnosis
   Answer: B.
2. A term neonate with suspected seizures has **glucose 32 mg/dL**. Best **first** step?
   A. Phenobarbital 20 mg/kg IV immediately
   B. **D10W 2 mL/kg IV bolus, then infusion and recheck**
   C. Levetiracetam 40 mg/kg
   D. CT brain
   Answer: B.

3. Which initial ASM is **first-line** by current consensus?
   A. Levetiracetam 40 mg/kg
   B. **Phenobarbital 20 mg/kg IV load (repeat if needed)**
   C. Midazolam infusion
   D. Lidocaine infusion
   Answer: B.
4. For **symptomatic hypocalcaemia** with seizures, an appropriate acute dose is:
   A. Calcium chloride 10% 5 mL/kg peripherally
   B. **Calcium gluconate 10% 1–2 mL/kg IV slowly with ECG**
   C. Oral calcium only
   D. No calcium; magnesium first
   Answer: B.
5. A baby continues to seize despite phenobarbital 40 mg/kg. The **next reasonable** step is:
   A. Increase phenobarbital to 60 mg/kg
   B. **Add fosphenytoin 20 mg/kg PE or levetiracetam 40 (+20) mg/kg**
   C. Stop all ASMs and observe
   D. Give lidocaine after phenytoin
   Answer: B. (Avoid lidocaine if phenytoin used.)

B. Short notes (3–5 lines each)

1. **Differentiate seizure vs jitteriness** in a neonate.
2. Outline the **vitamin-responsive** seizure trial in NICU (drug, dose, monitoring).
3. Write the **investigation panel** for a neonate with first seizure.
4. Explain when and how you consider **stopping ASMs** before discharge.

References

Classical sources

- **Caraka Saṃhitā**, *Cikitsāsthāna 28/50*—Ākṣepaka description (Vāta provocations causing recurrent limb jerks).

Modern guidelines & key reviews

- **ILAE Task Force** (2021). *The ILAE classification of seizures and the epilepsies: Modification for seizures in the neonate.* Epilepsia — EEG-based definition/classification.
- **WHO** (2011). *Guidelines on Neonatal Seizures* — practical global guidance.
- **Pressler RM et al.** (2023). *Treatment of seizures in the neonate: Guidelines and consensus-based recommendations* — dosing tables (phenobarbital, levetiracetam, phenytoin, midazolam, lidocaine) and pathway.
- **StatPearls** (2023–2024). *Neonatal Seizure*; *Neonatal Seizures & Neonatal Epilepsy* — evaluation (EEG-guided care; ASM discontinuation in acute provoked).
- **Merck Manual Professional** (2025). *Neonatal Seizure Disorders* — immediate lab panel and LP indications.
- **Cappellari AM et al.** (2023). *Questions and Controversies in Neonatal Seizures* — diagnostic/management overview.
- **Calcium/Magnesium protocols**: Starship (2018) **Calcium gluconate** neonatal guidance; LHSC NICU **Magnesium sulfate** dosing; Davis Drug Guide neonatal **magnesium** dosing; Vuralli (2019) hypocalcaemia review.
- **Vitamin-responsive epilepsies**: ILAE notes on **pyridoxine/PLP** dosing and PNPO deficiency resources.

60-second last-minute revision

- **Definition:** Neonatal seizures are **EEG-confirmed** paroxysms; many are electrographic-only. **EEG/aEEG is pivotal**.
- **Causes:** HIE, stroke/ICH, **hypoglycaemia**, **Ca/Mg/Na** derangements, infection, metabolic/genetic.

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- **First steps: ABC → Glucose** (D10W 2 mL/kg), correct **Ca/Mg/Na**, cultures/antibiotics if sepsis suspected.
- **ASMs: Phenobarbital 20 mg/kg** (±10–20 mg/kg) first-line → **fosphenytoin 20 mg/kg PE** or **levetiracetam 40(+20) mg/kg → midazolam/lidocaine** if refractory; consider **pyridoxine 100 mg IV** trial.
- **Stop ASMs** after ~**72 h seizure-freedom** in acute symptomatic cases when safe. **Counsel & follow up**.

You now have a clean, exam-ready script linking Ākṣepaka with neonatal seizure science—quote Charaka 28/50 for definition, then score marks with ILAE-aligned diagnostics and dosing.