



ASHFAQ ACADEMY OF CLINICAL MEDICINE

Neonatology Mastery

*Sample notes · Day 4: Neurology, Cardiology & Surgical
Emergencies*

This is a short preview of the illustrated student notes you receive with the course. The full Day 4 notes cover neonatal seizures, IVH and PHVD, congenital heart disease, the preterm PDA, two surgical-emergency chapters, hypotonia and SVT, closing with a master recap and dosing reference.

Shown here: how to use the notes, the contents, and the opening pages of Chapter 1.

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PREVIEW EDITION

STUDY GUIDE

HOW TO USE THESE NOTES

Every chapter follows the same journey, so once you know the rhythm, any new disease slots into a place you already know.



Each chapter opens with **four key numbers** and a one-line promise, and ends with a **scenario** (answer it before reading the answer) and a **recap** — if time is short, read those two first.

The Box Key

DEFINITION	The wording to learn exactly.	EXAM ALERT	What examiners ask again and again.
CLINICAL PEARL	Bedside technique and judgement textbooks rarely spell out.	RED FLAG	A sign, trap or error that changes management or causes harm.
BEYOND THE TEXTBOOK	Mechanism, landmark trials and molecular depth.	NHS / NICE PROTOCOL	Guideline protocols as written — thresholds, volumes, steps.
PAKISTAN NOTE	Local availability, brands and practical realities.	CLINICAL SCENARIO	A short case with model answers.
FROM THE NICU FLOOR	Ward-earned judgement, in the author's voice.	QUICK RECAP	The end-of-chapter revision summary.

Contents

Ch.	Topic
1	Neonatal Seizures vs Jitteriness — hold the limb · fix glucose, Ca and Mg first · the staged anticonvulsant ladder
2	Intraventricular Haemorrhage & Post-Haemorrhagic Ventricular Dilatation — grade the bleed · measure the ventricles, don't wait for symptoms · therapeutic LP → VAD → shunt
3	Congenital Heart Disease: Classification, Duct Physiology & the Major Lesions — the plumbing once, then every lesion · prostaglandin · tet spells
4	Patent Ductus Arteriosus in the Preterm Infant — the duct that won't close · echo · ibuprofen / paracetamol
5	Surgical Emergencies I — Oesophageal Atresia / TEF & Congenital Diaphragmatic Hernia — never reach for the mask · Replogle · PPHN
6	Surgical Emergencies II — Abdominal Wall Defects, Intestinal Obstruction & Anorectal Malformation — sac or no sac · bile = surgical · HAEC · ARM · stoma care
7	Neonatal Hypotonia — The Floppy Infant — weak or just loose? · central vs peripheral · SMA, myotonic dystrophy, Pompe
8	Neonatal Arrhythmias — Supraventricular Tachycardia — ECG · ice, then adenosine · unstable = DC cardioversion
M	Master Recap, Q&A & Bridge to Day 5 — the 'do NOT' wall · every Day 4 dose in one table · fluid strategies

Figures are schematic teaching diagrams, not to scale. Day 4 builds directly on Days 1–3: sepsis, hypoglycaemia, jaundice and IEM work-ups are cross-referenced rather than repeated.

CHAPTER 1

NEONATAL SEIZURES VS JITTERINESS

A seizure is a **SIGN**, not a diagnosis — recognise it accurately, hunt the cause with urgency, and fix the reversible causes first.

1-3% of TERM newborns seize (more in preterm)	≈ 50% of seizures are HIE — onset within 24 h	> 2-3 min • > 2-3/h treat if prolonged, frequent or cardiorespiratory compromise	20 mg/kg phenobarbital load (max total 40 mg/kg)
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1.1 Definition & Epidemiology

DEFINITION

Neonatal seizures are a manifestation of neurological dysfunction — a paroxysmal, stereotyped alteration in neurological function (motor, autonomic or behavioural) caused by abnormal, excessive, synchronous electrical discharge in the brain. They occur in **1-3% of TERM newborns**, and in a **GREATER** proportion of preterm babies.

Neonatal seizures are almost never a diagnosis in themselves — they are a **sign**, and your job is twofold and simultaneous: **(1) recognise the seizure accurately** (neonatal seizures often look nothing like the seizures of an older child) and **(2) hunt the cause with real urgency**, because several of the commonest causes are immediately treatable and time-sensitive — **hypoglycaemia, hypocalcaemia and infection** chief among them.

Motor types: **subtle • clonic • myoclonic • tonic**. *Subtle* seizures are what make this topic genuinely hard — which is why recognition gets the most time below.

Causes by frequency — know the proportions

- **HIE ≈ HALF of all cases** — the single most common cause; onset classically within the first 24 hours of life.
- **Intracranial haemorrhage, ischaemic stroke and other structural causes** — a further substantial proportion.
- **Infection** (meningitis, encephalitis) and **metabolic derangement** — each a smaller fraction, but disproportionately important because they are the most immediately and completely **treatable**.

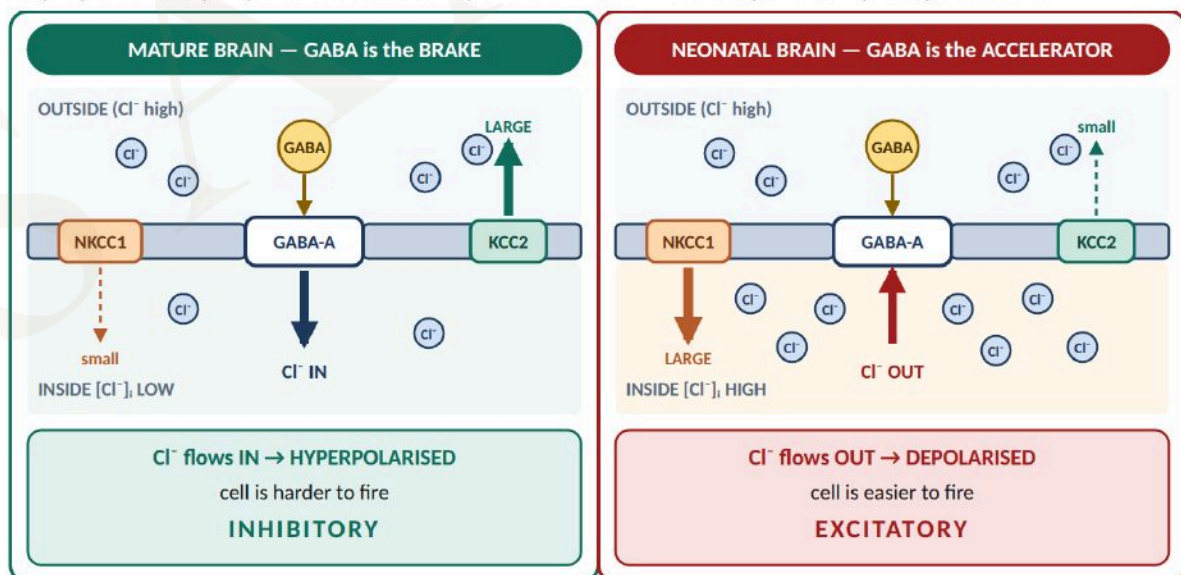


Figure 1.1 GABA flips from brake to accelerator in the immature neuron — NKCC1 high / KCC2 low drives Cl^- efflux.



BEYOND THE TEXTBOOK

The GABA paradox — why the immature brain seizes so readily. In the MATURE brain GABA opens Cl^- channels, Cl^- flows IN and the cell hyperpolarises (harder to fire). In the IMMATURE brain neurons express HIGH NKCC1 (Cl^- importer) and LOW KCC2 (Cl^- exporter) → intracellular Cl^- is HIGH → when GABA opens the same channel Cl^- flows OUT and the cell **depolarises**: GABA is functionally **excitatory**.

The ratio reverses over the first months as KCC2 rises — which is why seizure threshold changes so much between the neonatal period and later infancy, and why GABA-enhancing drugs (**phenobarbital, benzodiazepines**) are less reliably effective in neonates. **Bumetanide** (loop diuretic; NKCC1 inhibitor) has been trialled as an adjunct on exactly this rationale but is **not standard therapy** — mixed trial results and ototoxicity concerns.



HIGH-YIELD FOR EXAMS

GABA is EXCITATORY in the immature brain — high-yield precisely because it is counter-intuitive (most learners assume GABA is 'calming' everywhere). It also explains why the globally less-mature, less-excitabile neonatal brain is paradoxically **seizure-prone**.

1.2 Clinical Features — Recognition and the Jitteriness Trap



RECOGNIZING IT — Clinical Presentation

Beyond obvious convulsive movements, look deliberately at **four regions** — subtle seizures show themselves here far more often than as a dramatic generalised convulsion.

Region	What to look for
Eyes	Staring, blinking, horizontal deviation
Oral	Mouthing, chewing, sucking, tongue thrusting, lip smacking
Limbs	Boxing, cycling, pedalling movements
Autonomic	Apnoea, tachycardia, unstable blood pressure

Note whether the movement is **FOCAL** (one extremity) or **MULTIFOCAL** (several body parts), and always perform a full physical and neurological examination alongside.

The exact jitteriness-vs-seizure discriminator — you must be able to reproduce this

Sign	Jitteriness	Seizure
Stimulus provoked	YES	NO
Predominant movement	Rapid, oscillatory, tremor	Clonic, tonic
Movements cease when limb is held	YES	NO
Conscious state	Awake or asleep, normal	Altered
Eye deviation	NO	YES



HIGH-YIELD FOR EXAMS

The fastest bedside test: HOLD THE LIMB. Jitteriness stops when you gently restrain the limb with passive flexion; a true seizure continues regardless. Add whether the movement was stimulus-provoked (jitteriness typically is; seizures typically are not) and you can make a confident call before any investigation returns.



CLINICAL PEARL

Benign sleep myoclonus — a distinct entity: focal or generalised myoclonic limb jerks that do **NOT involve the face**, occurring as the baby is going to sleep or waking. EEG is normal; resolves by **4–6 months**. Naming it prevents an unnecessary work-up in a neurologically normal baby.

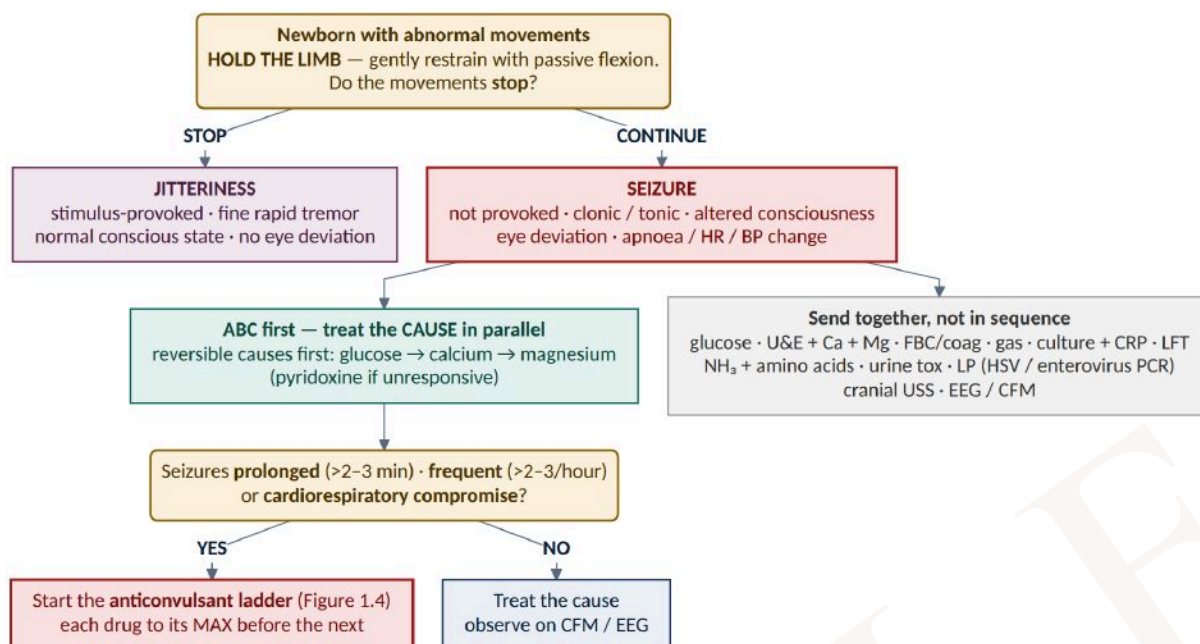


Figure 1.2 From 'is this jittery?' to the anticonvulsant ladder — the whole chapter on one page.

1.3 Causes — Organised By Category

Organise the differential exactly as the investigation panel is organised — **metabolic first (fastest to fix)**, then infective, then structural, then rarer causes.

Category	Causes
Metabolic	Hypoglycaemia · hypocalcaemia · hypomagnesaemia · hyponatraemia / hypernatraemia · inborn errors of metabolism (Day 3 IEM framework applies directly)
Hypoxic-ischaemic / structural	HIE (Day 1) · intracranial haemorrhage (Chapter 2) · stroke · congenital brain malformation
Infective	Bacterial or viral meningitis / encephalitis — specifically herpes simplex and enterovirus (Day 3 sepsis and TORCH frameworks apply directly)
Drug-related	Maternal substance use / withdrawal · local anaesthetic toxicity
Genetic / rare	Pyridoxine-dependent epilepsy · other inborn errors · genetic epilepsy syndromes

★ HIGH-YIELD FOR EXAMS

A seizing baby is where everything already built across the course must be available at once: hypoglycaemia and hypocalcaemia (Day 2), HIE (Day 1), meningitis and HSV (Day 3) — not retrieved topic by topic.

1.4 Investigations & EEG Correlation

CONFIRMING IT — Diagnosis & Work-Up

Category	FIRST-LINE — send essentially simultaneously, not sequentially	SECOND-LINE — if first-line is unrevealing
Metabolic	- Blood glucose - Serum electrolytes including calcium and magnesium - Blood gas - LFTs - Serum ammonia and amino acids - Urine amino acids and organic acids	Serum acylcarnitine, biotinidase, VLCFA, uric acid, sulphocysteine, total and free homocysteine