STEM-S

Last Minute Revision

LMR NOTES

Sample Notes Pediatric

PRESENTED BY
Stem-S

Growth, development

1. OFC = Occipito-Frontal Circumference (Head Circumference)

Category	Details
Definition	OFC = Occipito-Frontal Circumference (Head Circumference)
Normal OFC at 2 years	47–48 cm
Mean OFC at 2 years	~47.5 cm
Acceptable normal range	46–49 cm
Microcephaly at 2 years	OFC < 45 cm
Macrocephaly at 2 years	OFC > 50 cm
Growth pattern memory trick	“Birth 35 → 1 year 46 → 2 years 48”
Brain growth milestone	~80% of adult brain size reached by 2 years
HC growth rate (0–3 months)	~1 cm/month
HC growth rate (3–12 months)	~0.5 cm/month
HC growth rate after 1 year	~1 cm/year
HC growth pattern after 3 years	Growth nearly plateaus

2. Development milestones

Age	Gross Motor	Fine Motor	Language	Social
6 weeks	—	—	—	Social smile
2 months	—	—	Cooing	—
3 months	Head control	Hands open	Laughs / coos	Smiles responsively
4–6 months	Rolls; sits with support (6 mo)	Reach & transfer	Babbling	Stranger anxiety begins
6 months	Sit with support	Transfers	Babble	Stranger anxiety
9 months	Sit without support; pull to stand	Inferior pincer	Mama/Dada (nonspecific)	Separation anxiety; peek-a-boo
10–12 months	Cruising (holds furniture while walking)	—	—	—
12 months	Walk with one hand held	Mature pincer grasp	1–2 meaningful words	Waves bye-bye; follows simple command
15 months	Walk alone	Scribbles	3–5 words	Indicates wants
18 months	Run; walk upstairs with help	Tower of 3 blocks	10–20 words; points to body parts	Feeds self with spoon
2 years	Climbs stairs (2 feet/step); jumps	Tower of 6 blocks	2-word phrases; 50-word vocab	Parallel play
3 years	Tricycle; stands on one foot	Tower of 9 blocks; copies circle	3-word sentences; 75% intelligible	Group play, shares
4 years	Hops on one foot	Copies cross	Tells stories; 100% intelligible	Cooperative + pretend play
5 years	Skips	Copies triangle	Fluent speech	Follows rules

- MMR vaccine timing in child on chemotherapy

Topic	NEET SS–Relevant Information
Use of MMR during chemotherapy	**Contraindicated** (live vaccine; risk of disseminated infection)
When to give MMR after completing chemotherapy?	**3–6 months after completion of chemotherapy** (most guidelines use 6 months)
Reason for delay	Immune reconstitution of T-cells & B-cells required; earlier vaccination gives poor immunogenicity and risk of vaccine-associated disease
If MMR was given before chemotherapy	Immunity may wane → Revaccinate 6 months after chemotherapy
Measles exposure during chemotherapy	**Give IVIG (NOT MMR) for post-exposure prophylaxis**
During low-dose maintenance chemo (e.g., ALL maintenance)	Live vaccines still not recommended; avoid MMR
Household contacts	MMR allowed in contacts (it does NOT shed) Avoid OPV in contacts
General rule for live vaccines in immunosuppression	Avoid during chemo; resume ≥3–6 months post-therapy depending on immune recovery

- AEFI (based on MMR in ALL child)

AEFI Type	Relevance to ALL Child Receiving MMR (Exam Hint)
Vaccine-associated measles (VAM)	A rare but serious AEFI — can occur if MMR is mistakenly given during immunosuppression (e.g., acute lymphoblastic leukemia on chemotherapy)
Mechanism	Inability to control the live attenuated virus → can cause pneumonitis, severe rash, encephalitis, marrow suppression
AEFI Classification	Vaccine product-related reaction (live attenuated virus replicates excessively in an immunocompromised host)
NEET SS Implication	If a child with ALL inadvertently receives MMR → suspect VAM, manage as severe AEFI, report, and give IVIG if measles-like illness develops
Prevention	Strict contraindication; verify immunosuppression status before live vaccines

11. Miscellaneous General Pediatrics Topics

Allowed decibel level in NICU = 45 dB

- Maximum permitted sound level in NICU = 45 dB.
- Exceeding this causes apnea, bradycardia, increased oxygen use, and hearing damage, especially in preterm infants.
- Control measures: alarm optimization, soft door closure, quiet zones.

- NICU = ≤45 dB (WHO/AAP).
- Operating Room = ≤45–50 dB.
- Incubator ≤60 dB.
- Hospital wards = 45 dB day, 35 dB night.

Leucokoria = abnormal finding

- Leukocoria = white pupillary reflex, an abnormal and urgent finding in any child.
- Associated with life-threatening or vision-threatening diseases.
- Leukocoria = retinoblastoma until proven otherwise.
- Any white reflex → urgent evaluation.

Category	Cause	Key Clue
Malignant	Retinoblastoma	MCC; calcification on US/CT
Congenital	Congenital cataract	TORCH infections, metabolic disease
Retinal	Retinopathy of prematurity (ROP)	Preterm, oxygen exposure
Persistent fetal vasculature (PFV/PHPV)	Microphthalmia	
Infectious	Toxocariasis	Unilateral granuloma
Other	Coat's disease	Lipid exudates; unilateral

SAAG interpretation (general hepatology)

- Formula SAAG = Serum Albumin – Ascitic Albumin
- Accuracy ~97% for detecting portal hypertension

- **Phoenix score (Prediction of Mortality in Pediatric Septic Shock)**
- **(Used to stratify mortality risk & identify refractory shock)**

Component	What It Measures
P – Pupillary Abnormality	Fixed / dilated pupils
H – Hypotension (persistent)	MAP < age norm despite fluids
O – Oxygenation Deficit	PaO_2/FiO_2 < 200
E – Elevated Lactate	Lactate ≥ 4 mmol/L
N – Neurologic Dysfunction	GCS < 8 or coma
I – Inotropic Requirement	High-dose vasoactive infusion Epinephrine ≥0.3 µg/kg/min or equivalent
X – eXtreme Tachycardia / Bradycardia	Out-of-range HR for age

- Higher PHOENIX score → higher mortality risk.
- Helps identify catecholamine–refractory shock early.

- **Raised ICP**

Category	High-Yield Points
Definition	**ICP > 20–25 mmHg (sustained).**
Causes	Mass lesions: tumor, abscess, hemorrhage. Hydrocephalus Cerebral edema (trauma, hypoxia, meningitis, DKA cerebral edema) Venous sinus thrombosis Idiopathic intracranial hypertension (IIH).
Classic Triad (Cushing's Reflex)	Hypertension + Bradycardia + Irregular respiration → impending herniation.
Red-Flag Exam Findings	• Papilledema (rare in infants). • Sunset sign (hydrocephalus). • Sixth nerve palsy. • Bulging fontanelle + widened sutures (infants).
ICP Targets	ICP < 20 mmHg; CPP > 50–60 mmHg in children.
Initial Medical Management	• Elevate head 30°, midline. • Maintain airway; avoid hypoxia/hypercarbia ($PaCO_2$ 35–40 mmHg). • Adequate sedation + analgesia. • Avoid hypotension (MAP support essential).
Osmotherapy	• Mannitol 0.25–1 g/kg (avoid if hypotensive). • Hypertonic saline 3% (bolus 3–5 mL/kg) — preferred in trauma & DKA cerebral edema.
Hyperventilation	**Not routine; only for acute herniation. Goal $PaCO_2$: 30–35 mmHg temporarily.**
Steroids	**Only for tumors / vasogenic edema.** **NOT used in traumatic brain injury or infections.**
Definitive Measures	• External ventricular drain (EVD) for hydrocephalus. • Surgical decompression for mass lesions.
Avoid	**Hypotonic fluids, aggressive hyperventilation, repeated boluses of mannitol without monitoring osmolality.**

- EEGs

EEG Pattern	Description	Clinical Association / Diagnosis
Generalized 3-Hz spike–wave	Bilateral, symmetric, synchronous	Childhood Absence Epilepsy
2.5–4 Hz spike-wave	Faster irregular spike–wave	Juvenile Absence / JME
Generalized polyspike–wave	Multiple spikes + slow wave	JME (Juvenile Myoclonic Epilepsy)
Hypsarrhythmia	Chaotic, high amplitude, multifocal spikes	Infantile Spasms (West syndrome)
Burst–Suppression Pattern	Bursts of high voltage alternating with flat periods	Ohtahara syndrome, severe encephalopathy, deep anesthesia
Slow Spike–Wave (<2.5 Hz)	Generalized slow spike-wave	Lennox–Gastaut Syndrome (LGS)
Periodic Lateralized Epileptiform Discharges (PLEDs / LPDs)	Repetitive focal spikes at regular intervals	Acute focal structural lesions (HSV encephalitis, stroke)
Delta Brush	Slow delta with superimposed fast beta	Preterm EEG; also anti-NMDA encephalitis
Triphasic Waves	Generalized high amplitude triphasic morphology	Hepatic encephalopathy

Genes for Common Pediatric Epilepsies

- Na^+ channel disorders → SCN family

→ SCN1A (Dravet, GEFS+)
→ SCN1B (GEFS+)
→ SCN2A (LGS, infantile epileptic encephalopathy)

- K^+ channel neonatal epilepsy → KCNQ2/Q3

(Think "K for Kute Newborn Seizures)

- Absence epilepsies → GABA receptor genes (GABRA1, GABRG2, CACNA1H)

- Infantile spasms genes

ARX, CDKL5, STXBP1→ "A-C-S = ARX–CDKL5–STXBP1 → ACTH syndrome babies."

- BECTS / ESES (Electrical Status Epilepticus in Sleep)

GRIN2A → "GRIN = speech grinning difficulty"

- Severe migrating seizures (Epilepsy with Migrating Focal Seizures of Infancy)

KCNT1 → "Can't stop seizures."

- JME → EFHC1
- Ohtahara →ARX, STXBP1 (Early Infantile Epileptic Encephalopathy)
- TSC epilepsy → TSC1/2
- Angelman → UBE3A

- **NNJ**

Category	High-Yield Points for NEET SS
Physiologic Jaundice	Appears after 24 h, peaks 3–5 days, resolves by 7–10 days; unconjugated; TSB usually < 12–14 mg/dL in term.
Pathologic Jaundice — Red Flags	<24 h onset, **rising > 0.3 mg/dL/h, TSB > 95th percentile,** duration > 2 weeks (term)**, conjugated > 2 mg/dL or >20% of total.**
Most Common Causes (Unconjugated)	• Hemolysis: ABO incompatibility (MC), Rh, G6PD deficiency, spherocytosis. • Infection: Sepsis, UTI.
Conjugated/Direct Hyperbilirubinemia Causes	Biliary atresia (BA), neonatal hepatitis, CMV, PFIC, metabolic (galactosemia, tyrosinemia), sepsis, TPN cholestasis
Direct Hyperbilirubinemia — AAP Red Flag	**Any direct bilirubin ≥1 mg/dL if total ≤5, or ≥20% of total = cholestasis → urgent evaluation.**
Breastfeeding vs. Breastmilk Jaundice	Breastfeeding jaundice: day 2–4, due to **dehydration/poor intake** → treat with improved feeding. Breastmilk jaundice: day 5–7 to 12 weeks, normal thriving infant,
ABO vs Rh Hemolysis	ABO: MC cause; can occur in 1st pregnancy; DAT may be weak/negative. Rh: more severe; hydrops; positive DAT; profound anemia.
G6PD Deficiency	Sudden severe jaundice especially after stress/drugs; DAT negative; risk of kernicterus even at “moderate” bilirubin.
Biliary Atresia (BA)	Persistent jaundice >2 weeks, **acholic stools**, high GGT, hepatomegaly; USG: absent/triangular cord; HIDA non-excreting
Kernicterus Signs	Early: poor feeding, hypotonia, high-pitched cry. Late: opisthotonus, seizures, deafness, choreoathetoid CP.
2022 AAP Phototherapy Threshold (Updated)	**Higher treatment thresholds than previous AAP. Based on GA, risk factors (hemolysis, sepsis, asphyxia, hypoalbuminemia <3 g/dL).**
Phototherapy	Irradiance = therapeutic (Amount of light energy delivered per unit area) vs luminance = brightness only (lux) **Intensive phototherapy = ≥30 μW/cm²/nm Standard = 8–12**
Double–Volume Exchange Transfusion (DVET)	Indications: • TSB ≥ exchange line on AAP chart despite intensive phototherapy.
Medications	IVIG (1 g/kg) for isoimmune hemolysis when **bilirubin rising >0.5 mg/dL/h.** Phenobarbital rarely used (induces conjugation).
Evaluation in Prolonged Jaundice (>2 weeks)	Fractionated bilirubin, LFTs, GGT, thyroid profile, urine for reducing substances, sepsis screen, USG hepatobiliary, stool color check.
Latest Updates (2023–2024)	• Pulse-ox screening improves detection of duct-dependent CHD but also picks up cholestatic infants. • Routine newborn screening for G6PD deficiency recommended

Nomogram Component	High-Yield Summary (Latest AAP 2022–23 + IAP)
Age Axis	Use postnatal age in hours (mandatory). Accurate plotting = key.
Gestational Age Bands (AAP 2022)	≥38 wks, 35–37+6 wks <35 wks (premature use of IAP/NICE charts).
Phototherapy Thresholds (Approx.)	≥38 wks: ~15–18 mg/dL after day 2. 35–37 wks: ~12–15 mg/dL. Lower thresholds if hemolysis/sepsis.
Exchange Transfusion Thresholds	≥38 wks: ~22–25 mg/dL 35–37 wks: ~18–22 mg/dL.
Risk Factors That Lower Threshold by 2–3 mg/dL	**Hemolysis (Rh/ABO)** **G6PD deficiency** **sepsis, asphyxia, acidosis, temperature instability**
What AAP 2022 Removed	**No Bhutani risk zones (no low/intermediate/high risk).**
TcB (Transcutaneous) Rules	Valid only up to 15 mg/dL. If TcB within 3 mg/dL of phototherapy line → get serum bilirubin (TSB) NOT reliable during or 24 h after phototherapy.
Preterm Nomograms (<35 wks)	IAP/NICE charts: thresholds lower → Start PT at 5–12 mg/dL depending on GA.
Key AAP 2022 Principles	**Higher PT thresholds** **GA-driven; separate PT & exchange charts** **consider rate of rise (>0.3 mg/dL/hr).**

Rheumatology

- **Interferonopathies**

Disease	Key Features	Imaging	Gene	Diagnostic Clue
Aicardi–Goutières Syndrome (AGS)	Encephalopathy, spasticity, chilblains, regression	**Basal ganglia + periventricular calcifications,**	TREX1, RNASEH2, SAMHD1, ADAR1, IFIH1	**↑ IFN-α, CSF lymphocytosis, high ISG**
STING-associated Vasculopathy of Infancy (SAVI)	Neonatal vasculitis, acral necrosis, ILD	**Interstitial lung disease**	TMEM173	**Vasculitis + ILD + high IFN signature**
COPA Syndrome	Arthritis + alveolar hemorrhage, ILD	**Diffuse ILD**	COPA	**Autoimmune phenotype + IFN signature**
ISG15 Deficiency	Early inflammatory crisis, seizures, TORCH-like	Intracranial calcifications	ISG15	High IFN activity
USP18 Deficiency	Severe neonatal inflammation, sepsis-like picture	Variable	USP18	Profound interferon activation

- Treatment- JAK inhibitors (Ruxolitinib/Baricitinib)

- **PIRDS including HLH**

Feature	HLH	MAS (Macrophage Activation Syndrome)
Ferritin	**>10,000 typical**	≥684 (diagnostic cutoff) Often 1,000–10,000
Triglycerides	**>265 mg/dL**	>156 mg/dL
Fibrinogen	**<150 mg/dL**	≤360 mg/dL
Soluble IL-2 Receptor (sCD25)	**Very high (>2,400 U/mL)**	Mild–moderate elevation
ESR	Normal/low	**Low ESR (key clue)**
CRP	Mild–moderate elevation	**Very high CRP (50–200 mg/L)**
LFTs	AST/ALT elevated	**AST disproportionately high (key clue)**
D-dimer	High	**Very high**
Triggers	**EBV, malignancy, primary HLH genes**	**SJIA, SLE flare**
Bone Marrow	Hemophagocytosis (not mandatory)	Hemophagocytosis (often later finding)
Classic Clinical Clue	Hepatosplenomegaly + cytopenias + extremely high ferritin	Drop in ESR, falling platelets during SJIA flare
Treatment	Steroids + Etoposide + Cyclosporine; HSCT in primary HLH	High-dose IV steroids, then Cyclosporine / Anakinra

- Because MAS is part of SJIA-driven cytokine activation → fibrinogen falls later and is not as severely depressed as in HLH.

Cardiology

CMP

1. Most common pediatric cardiomyopathy = Dilated CM (DCM).
2. Reversible DCM → Primary carnitine deficiency (SLC22A5 mutation).
3. HCM in infants → think POMPE until proven otherwise (acid α-glucosidase deficiency).
4. Syncope during exertion = HCM until proven otherwise.
5. Arrhythmias > HF symptoms → Restrictive CM (RCM).
6. Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) clue =

- Adolescent + VT + epsilon waves
- Gene: Desmosomal proteins (PKP2).

7. Left Ventricular Non-Compaction (LVNC)

- Strongly linked with mitochondrial disorders and congenital myopathies
- Look for skeletal muscle weakness + arrhythmias.

8. Anthracycline cardiotoxicity = cumulative dose–dependent

- check Ejection fraction every cycle.
- Max cumulative dose: 450–550 mg/m^2.

9. Noonan syndrome → HCM (Mutations in RAS/MAPK pathway genes- PTPN11, RAF1)
10. Giant Cell Myocarditis does NOT cause cardiomyopathy in infants (exam trap)

• Typically affects older children/adults.

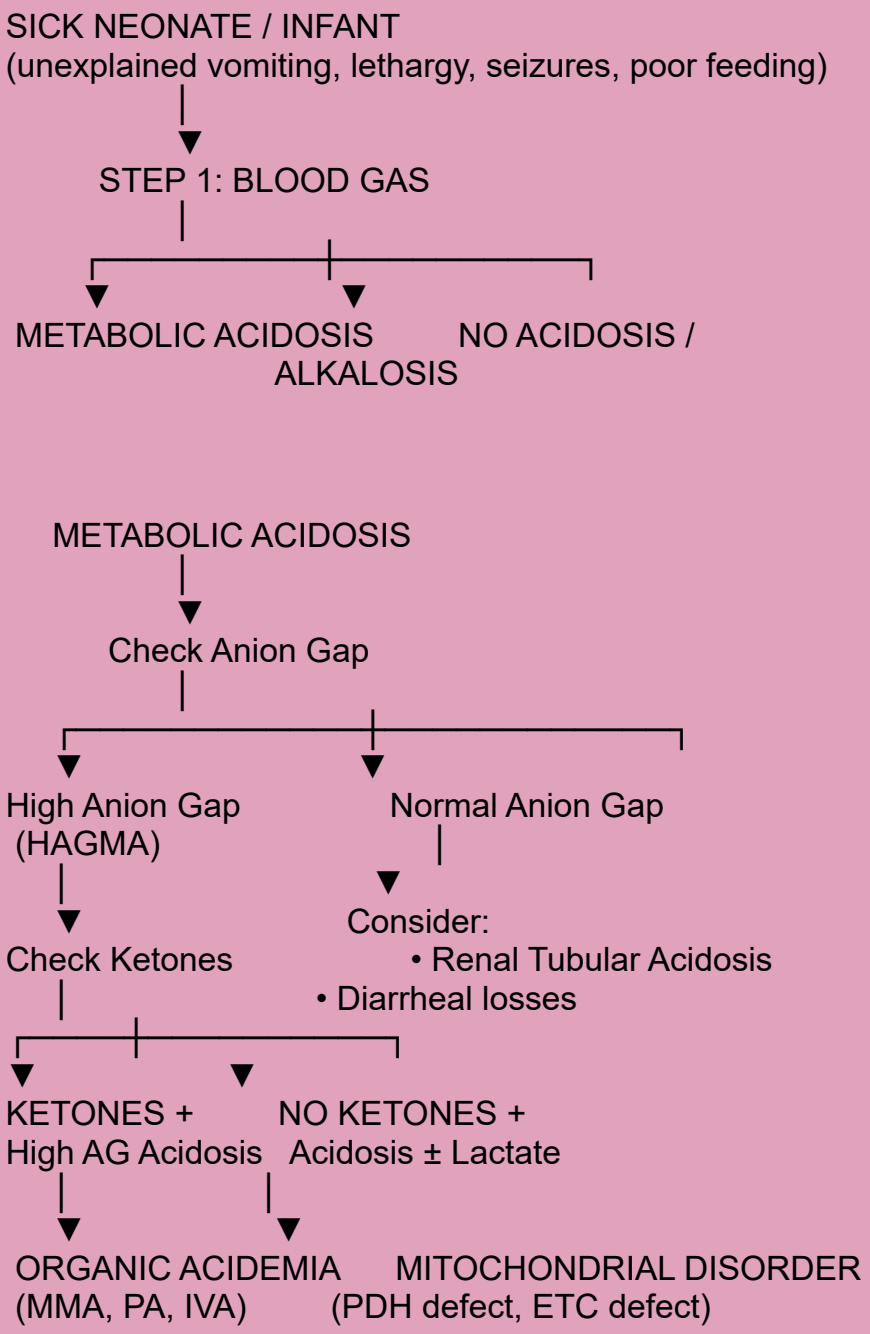
Named Surgeries

- HLHS- Norwood f/by Glenn f/by Fontan

Named Procedure	Indication (Cardiac Condition)	Distinguishing Clues
Norwood Procedure	Hypoplastic Left Heart Syndrome (HLHS)	Stage I palliation → create neo-aorta + BT shunt
Bidirectional Glenn (Cavopulmonary Shunt)	Single ventricle lesions (post-Norwood)	**SVC** → pulmonary artery; reduces ventricular volume load
Fontan Procedure	Final stage palliation for single ventricle	**IVC** → pulmonary artery; non-pulsatile pulmonary flow
Blalock–Taussig (BT) Shunt temporary palliation	TOF / Cyanotic CHD with ↓ PBF	**Subclavian artery** → pulmonary artery
Potts Shunt	Severe pulmonary hypertension	**Descending aorta** → left PA; used in suprasystemic PAH
Jatene Arterial Switch Operation (ASO)	d-TGA	Switch arteries + reimplant coronaries; **surgery of choice in neonates**
Rashkind Balloon Atrial Septostomy	d-TGA with restrictive atrial septum	**Emergency** to improve mixing; bedside cath procedure
Rastelli Procedure	d-TGA + VSD + PS	VSD closure + conduit from RV → PA
Nikaidoh Procedure	d-TGA + LVOTO	Translocation of aortic root + VSD closure
Ross Procedure	Aortic stenosis in children	Replace aortic valve with pulmonary autograft
Konno Procedure	Severe aortic stenosis with small LVOT	Augment LVOT with VSD enlargement

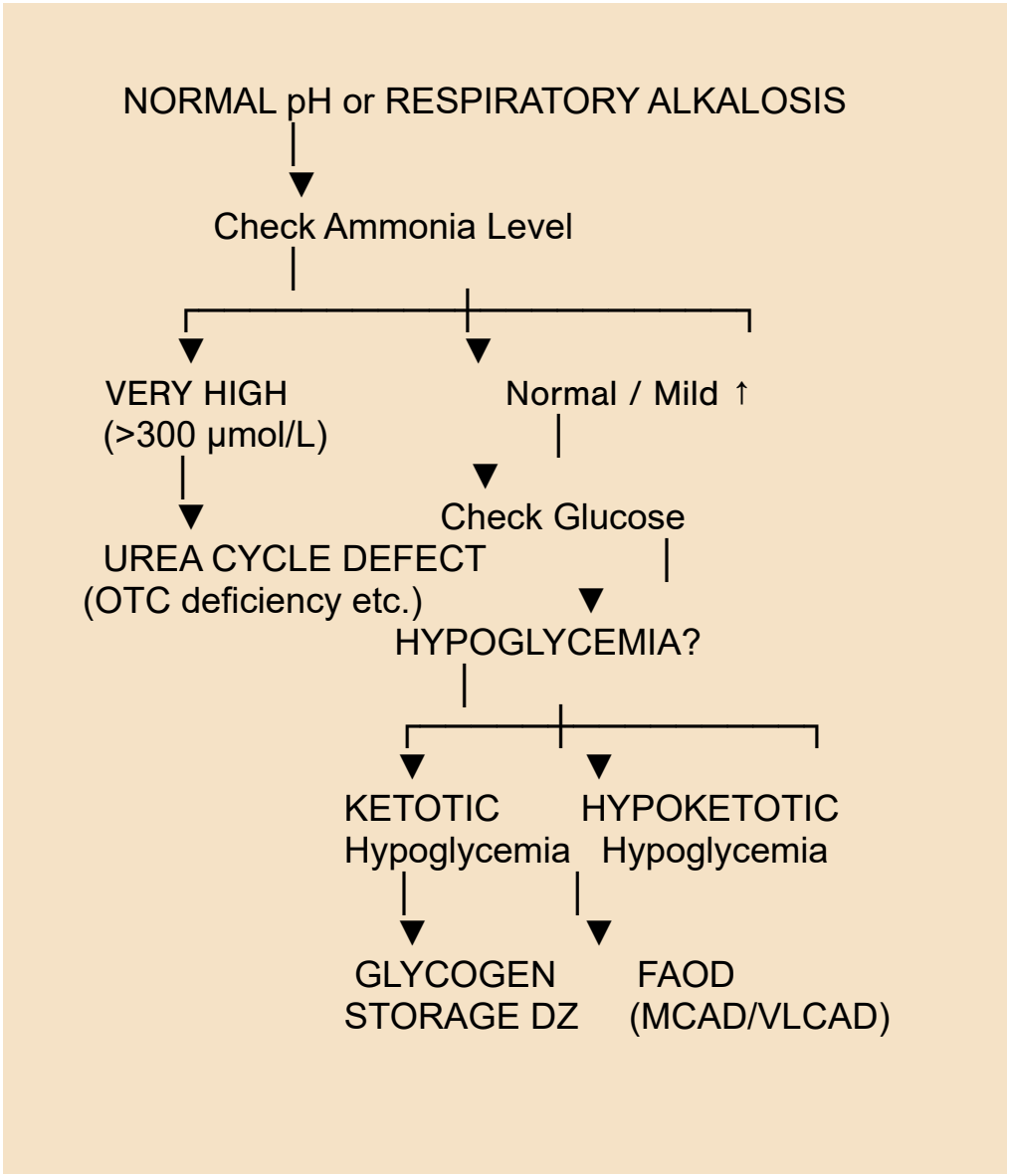
Metabolism

- **Approach to IEM**



- **High AG + ketones → Organic acidemia**
- **High AG + no ketones + high lactate → Mitochondrial disease**

Metabolism



- **No acidosis + respiratory alkalosis + very high ammonia → Urea Cycle Defect**
- **Hypoglycemia + no ketones → FAOD**
- **Hypoglycemia + ketones → GSD (except GSD I = no ketones)**

Nephrology

- **Nephrotic syndrome**

Category	ISPN High-Yield Points (Exam Focused)
Definition	Proteinuria: UP/Cr ≥ 2 mg/mg OR 3+ urine dipstick Hypoalbuminemia: <2.5 g/dL Edema present Hyperlipidemia.
Most Common Type	Minimal Change Disease (MCD) (85–90%).
Steroid Regimen (Initial Episode)	Prednisolone 2 mg/kg/day (max 60 mg) × 6 weeks, THEN 1.5 mg/kg alternate days × 6 weeks → total 12 weeks (ISPN 2021).
Response Definitions	Steroid Sensitive: Remission within 4 weeks. Steroid Dependent: Relapse during taper or within 14 days of stopping. Frequent Relapser: ≥2 relapses in 6 months OR ≥4/year. Steroid Resistant: No remission after 6 weeks daily steroids.
Indications for Renal Biopsy	Steroid-resistant NS Age <1 year or >12 years Hematuria, low C3, hypertension AKI, persistent renal dysfunction Before calcineurin inhibitor therapy sometimes.
Relapse Treatment	Prednisolone 2 mg/kg/day until remission (usually 5–7 days) → then 1.5 mg/kg alternate days × 4 weeks.
Management of Frequent Relapsers / Steroid-Dependent	**1. Levamisole** (2 mg/kg/day or alternate days). S/e- **Agranulocytosis (neutropenia)**, ANCA-positive vasculitis (skin necrosis, purpura) **2. Cyclophosphamide** 2 mg/kg/day × 12 weeks. S/e- Gonadal toxicity (infertility), Hemorrhagic cystitis (acrolein metabolite), **Myelosuppression (neutropenia)**, Secondary malignancies (TCC bladder, leukemia) **3. MMF** (600 mg/m² twice daily). S/e- **Bone marrow suppression**, GI toxicity, Opportunistic infections , Teratogenic **4. Calcineurin inhibitors (CNI):** Tacrolimus preferred, Cyclosporine also used.
Steroid-Resistant NS (SRNS)	Trial of CNI (Tacrolimus / Cyclosporine) for 6 months. Supportive: RAAS blockade, BP control **Tacrolimus: fewer cosmetic side effects (no hirsutism/gingival hyperplasia)** Cyclosporine: more cosmetic toxicity; requires drug levels.
Rituximab Use	For SRNS unresponsive to CNI AND frequent relapsers/steroid dependent NS.
Infection Risk	High risk of SBP (Strep pneumo), cellulitis. **Give Pneumococcal PCV13 + PPSV23; annual influenza vaccine.**
Thrombosis Risk	Albumin <2 g/dL, immobilization, hemoconcentration → high risk of DVT, RVT Consider LMWH if high-risk.
Hypertension Management	RAAS blockade is ideal but avoid ACEi in AKI/hypovolemia.

Nephrology

- **Vaccines in nephrotic syndrome**

Clinical State	Recommendation
Remission + off immunosuppression	All routine vaccines, including live vaccines
On low-dose steroids (<2 mg/kg/day or <20 mg/day)	Live vaccines allowed
On high-dose steroids (≥2 mg/kg/day or ≥20 mg/day for ≥14 days)	**No live vaccines Inactivated vaccines allowed**
On CNI (Tacrolimus/Cyclosporine)	**Avoid live vaccines Inactivated vaccines OK**
On MMF	**Avoid live vaccines**
After Rituximab	**Avoid live vaccines Inactivated vaccines ineffective for 6 months**

- **PCV → PPSV → Flu yearly → Varicella in remission → HepB booster if needed.**

Vaccine	When to Give?
PCV13 (Pneumococcal Conjugate)	Give if not received earlier
PPSV23 (Pneumococcal Polysaccharide)	8 weeks after PCV13, repeat in 5 years
Influenza (Inactivated) Most important relapse-preventing vaccine	Annually
Varicella (Live)	Only in remission, not on immunosuppression Post-exposure: VZIG within 72 hours
Hepatitis B	Booster if anti-HBs <10 mIU/mL

Respiratory

- **Pneumonia**

Type of Pneumonia	Key Features / Clues	Common Organisms	X-ray Pattern
Lobar Pneumonia	High fever, chills, sudden onset, pleuritic chest pain; consolidation localized to a lobe	**Streptococcus pneumoniae (most common), Klebsiella**	Homogeneous consolidation, air bronchogram
Bronchopneumonia	Patchy symptoms, occurs in very young/old; follows viral infection	**Staph aureus, H. influenzae, Gram-negatives**	Patchy infiltrates, multifocal, peribronchial
Interstitial (Atypical) Pneumonia	Dry cough, low-grade fever, headache, minimal sputum	Mycoplasma, Chlamydophila, Viruses (RSV, influenza)	Diffuse interstitial pattern, reticulonodular
Round Pneumonia (Pediatrics)	Seen in children due to underdeveloped pores of Kohn; mimics mass	**S. pneumoniae**	Round, well-defined opacity
Necrotizing Pneumonia	Severe, cavitation, hemoptysis	**Staph aureus (PVL+), Klebsiella**	Cavitary lesions, pneumatoceles
Viral Pneumonia	Wheeze + interstitial findings; infants	RSV, influenza, adenovirus	Perihilar streakiness, hyperinflation

Feature	Recurrent Pneumonia	Persistent Pneumonia
Definition	≥ 2 episodes in 1 year OR ≥ 3 episodes ever, with radiographic clearing between episodes	Pneumonia lasting >6 weeks without radiologic clearing
Key Concept	New episodes with complete recovery in between	Never fully resolves
Pattern Suggests	Underlying predisposition	A fixed lesion or ongoing process

- Recurrent in SAME lobe → obstruction (foreign body).
- Recurrent in DIFFERENT lobes → CF, immunodeficiency, aspiration.
- Persistent pneumonia → think TB, fungal, airway obstruction, congenital anomalies.
- Chronic wet cough + situs inversus → Primary ciliary dyskinesia.
- Failure to thrive + steatorrhea → Cystic fibrosis

Primary ciliary dyskinesia-
- **Bronchiectasis**
- **Recurrent sinusitis**
- Nasal polyposis
- Recurrent otitis media
- Infertility

50% children with PCD will have kartagener

Kartagener (subset of primary ciliary dyskinesia)-
- **Bronchiectasis**
- **Chronic sinusitis**
- Situs inversus

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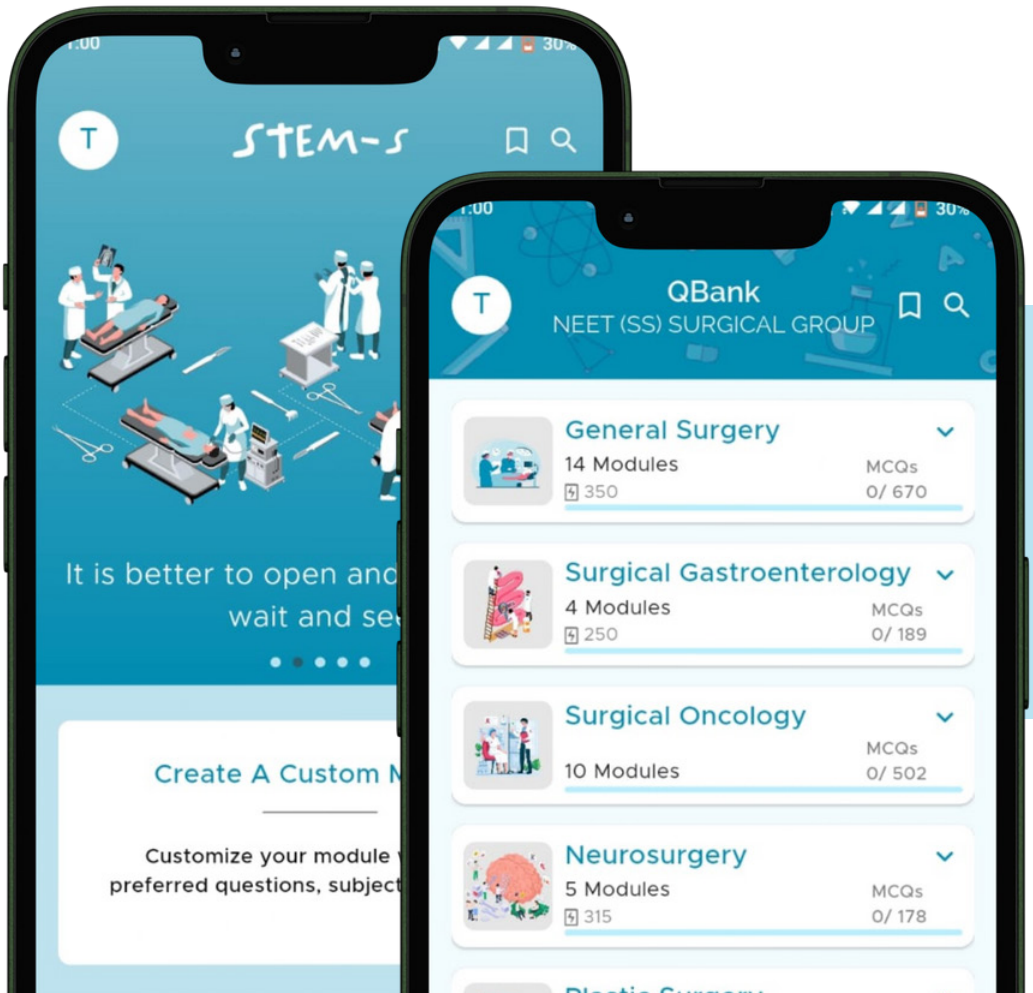

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