Pediatrics comprehensive summary

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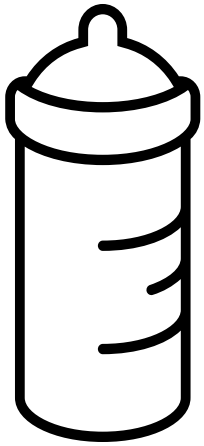

References: Slides

*Note: This file contains a comprehensive summary of the **other 18 files, excluding those mentioned** in the post.*

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Approach to a Child with Short Stature

Module 1: Normal Pediatric Growth Milestones

- ***Weight:*** *Term neonates may lose up to* ***10% of their birth weight****, but typically regain it by* ***10 to 14 days*** *of age. Infants grow rapidly, gaining roughly 30 g/day for the first three months, 20 g/day between 3-6 months, and 10 g/day between 6-12 months. By* ***4 months****, infants double their birth weight, and they* ***triple it by 1 year****.*
- ***Height/Length:*** *The average birth length is* ***50 cm****. A child will grow* ***25 cm*** *in their first year. They grow 10 cm between 12-24 months, 7.5 cm between 24-36 months, and another 7.5 cm between 36-48 months. Crucially, children reach* ***one-half of their adult height by 24 to 30 months****. From age 4 until puberty, children grow at a steady rate of* ***5 cm per year****.*
- ***Head Circumference:*** *The average at birth is* ***35 cm****. The brain grows most rapidly in the first 6 months, with the head circumference increasing by 2 cm in the first month alone. Brain weight doubles by 4 to 6 months, triples by one year, and most head growth is completely finished by* ***4 years of age****.*

Module 2: The ICP Model of Growth

Human linear growth is mathematically broken down into three distinct hormonal phases known as the ICP concept:

1. ***Infantile Phase (0-2 years):*** *A period of rapid but decelerating growth that continues from intrauterine life. This phase is largely* ***independent of growth hormone*** *and depends heavily on nutrition, insulin, and insulin-like growth factors (IGFs). Birth size reflects the intrauterine environment, but by age 2, the infant will "track" onto their true genetic centile.*
2. ***Childhood Phase (2-12 years):*** *A slower, slightly decelerating growth curve that is heavily dependent on* ***growth hormone (GH) and thyroxine****. A healthy child will stay on their genetic centile throughout this phase.*
3. ***Pubertal Phase (~12 years to adulthood):*** *This phase is driven by* ***sex steroids (estrogen)****, which are aromatized from testosterone. Estrogen increases GH secretion, causing a growth spurt, but it is also responsible for eventually fusing the epiphyses to halt growth in both sexes.*

Module 3: Defining and Evaluating Short Stature

- ***Short Stature Definition:*** *A child is formally considered short if their height is* ***below 2 standard deviations (SD)*** *from the mean for their age and sex, which correlates to the* ***3rd centile****.*
- ***Growth Failure:*** *This is defined as a failure to maintain an appropriate height velocity (HV). Growth failure is highly likely if a child's growth curve deviates downwards across* ***two major percentile curves****. It is also diagnosed if the child grows slower than the minimum thresholds:* ***<5.5 cm/year*** *(ages 2-4),* ***<5 cm/year*** *(ages 4-6), or* ***<4 cm/year*** *(age 6 to puberty).*
- ***Target Height (Mid-Parental Height):*** *To predict adult height, you must calculate the target height.*
    - ***Boys:*** *(Mother's height + 13 cm + Father's height) / 2.*
    - ***Girls:*** *(Father's height - 13 cm + Mother's height) / 2.*
    - *Target Range: 95% of normal individuals will reach a final height within the mid-parental height* ***± 8.5 cm****.*
- ***Puberty & Growth Spurts (Tanner Staging):***
    - ***Girls:*** *Peak height velocity occurs early in puberty at* ***Tanner stage B3*** *(when the breast mound forms). Height velocity slows down at B4, coinciding with the onset of menstruation (menarche).*
    - ***Boys:*** *Peak height velocity occurs later in puberty at* ***Tanner stage G4*** *(penis lengthens and broadens, testes reach ~12-15 ml).*

Module 4: Differential Diagnosis (Causes of Short Stature)

When a child presents with short stature, the causes are divided into normal variants and pathological conditions:

- ***Familial (Genetic) Short Stature:*** *The child is short, parents are short, but the child is entirely healthy with a* ***normal bone age****.*
- ***Constitutional Delay:*** *The child is short and looks younger than their chronological age. They have a* ***delayed bone age*** *and will experience late puberty and a late catch-up growth spurt.*
- ***Small for Gestational Age (SGA) / IUGR:*** *Most catch up by age 2, but about* ***10% fail to experience catch-up growth****. Symmetric IUGR (head, length, and weight all equally small) often results in a short adult.*
- ***Dysmorphic Syndromes:*** *Includes Turner, Noonan, Silver-Russell, Williams, and Prader-Willi syndromes.*
- ***Skeletal Dysplasias:*** *Diseases causing frank skeletal abnormalities, such as* ***Achondroplasia*** *(a gain-of-function mutation in the FGFR3 gene that disrupts normal endochondral ossification). This often presents with an advanced bone age and a disproportionately low adult height.*
- ***Chronic Diseases:*** *Any chronic issue (GI, cardiac, respiratory, renal, CNS) can stunt growth. Conditions like* ***coeliac disease or chronic renal disease can be completely silent*** *and present solely as short stature.*
- ***Psychosocial Deprivation:*** *Severe emotional abuse, neglect, or poor social circumstances can result in genuine growth failure.*
- ***Endocrine Disorders:*** *GH deficiency, thyroxine deficiency, cortisol excess, and precocious puberty.*

Module 5: Diagnostic Workup

- ***Clinical Evaluation:*** *Always use accurate measuring equipment with the child's head in the Frankfurt plane to measure stretched height. Measure sitting height and leg length if you suspect disproportionate growth (like skeletal dysplasia). Plot the growth chart carefully and check the pubertal status and dysmorphic features.*
- ***Bone Age:*** *Always order an X-ray of the* ***left hand and wrist*** *to assess bone maturation (using methods like Greulich & Pyle).*

- ***Screening Labs:*** *Initial blood work should include a full blood count, ESR, coeliac screening (TTG antibodies), liver function, blood gas, electrolytes, calcium, phosphate, urinalysis, thyroid function (TFT), IGF-1, IGFBP3, prolactin, and cortisol. Have a* ***low threshold for ordering a karyotype in girls*** *to rule out Turner syndrome.*
- ***Further Investigation:*** *If indicated, perform endocrine stimulation testing (such as ITT, arginine, clonidine, or glucagon tests) and pituitary MRI imaging.*

Module 6: Growth Hormone Deficiency and Treatment

- ***GH Physiology:*** *GH is an anterior pituitary protein that antagonizes insulin, promotes lipolysis (fat breakdown), and augments protein synthesis. It is secreted in pulses, stimulated by sleep and exercise, and inhibited by glucose and leptin.*
- ***GH Deficiency (GHD):*** *Can be congenital (idiopathic, genetic, Prader-Willi, midline defects like septo-optic dysplasia) or acquired (tumors like craniopharyngiomas, surgery, cranial radiotherapy, or trauma).*
- ***Treatment:*** *The primary treatment is* ***Recombinant Human Growth Hormone (RHGH)*** *administered via daily bedtime subcutaneous injections. Patients must be followed up every 3 months.*
- ***Indications for GH Treatment:*** *Approved for GH deficiency, Turner syndrome, Noonan syndrome, SHOX deficiency, Prader-Willi syndrome, SGA without catch-up by 4 years, chronic renal insufficiency, and idiopathic short stature.*
- ***Adverse Effects of GH Therapy:*** *You must monitor for hyperglycemia, slipped capital femoral epiphysis, progression of pre-existing scoliosis, pseudotumor cerebri, transient gynecomastia, hypothyroidism, pancreatitis, and the risk of second neoplasms like meningiomas.*

Development Assessment

1. Core Principles & Screening

Child development is a dynamic, sequential process that spans four main domains: ***Gross Motor, Fine Motor/Adaptive, Social/Behavioral, and Language****.*

- ***Screening Importance:*** *Without screening, 70% of developmental disabilities go unnoticed. With standardized screening tools, 70-80% of developmental disabilities and 80-90% of mental health problems are correctly identified.*
- ***AAP Recommendations:*** *Clinicians should screen children for general development using standardized tools at* ***9, 18, and 24 or 30 months****, and specifically for autism at* ***18 and 24 months****.*

2. Normal Developmental Milestones (High-Yield for Memorization)

Vision and Hearing Basics:

- ***Hearing*** *is the dominant sense in the first 10 months of life. Infants turn to noise sources by 3 months.*
- ***Vision*** *fixation begins at 35 weeks gestation. By 2 months, infants follow objects 180 degrees and prefer human faces. Visual acuity reaches adult levels by 1 year of age.*

Key Milestone Groupings by Age:

- ***3 Months:***
    - *Motor: Raises head above the plane of the body when prone; opens hands spontaneously and reaches for objects.*
    - *Communication/Social: Says "aah, ngah"; exhibits sustained social contact.*
- ***4 Months:***
    - *Motor: Has full head control and sits with full truncal support; reaches for objects and puts them in the mouth.*
    - *Communication/Social: Laughs loudly.*

- ***6-7 Months:***
    - *Motor: Sits with pelvic support, rolls over; transfers objects from hand to hand.*
    - *Communication/Social: Forms polysyllabic vowel sounds; prefers mother, enjoys mirrors.*
- ***10 Months:***
    - *Motor: Sits alone, crawls, pulls to a standing position; utilizes a thumb-index pincer grasp.*
    - *Communication/Social: Says "mama" and "dada"; waves bye-bye, plays peek-a-boo, and responds to their name.*
- ***12 Months (1 Year):***
    - *Motor: Walks with one hand held; releases objects on demand.*
    - *Communication/Social: Plays simple ball games.*
- ***15 Months:***
    - *Motor: Walks alone, crawls upstairs; builds a tower of 3 cubes.*
    - *Communication/Social: Follows simple commands; indicates desires by pointing, hugs parents.*
- ***18 Months (1.5 Years):***
    - *Motor: Runs stiffly, walks upstairs holding a hand; builds a tower of 4 cubes, imitates a vertical stroke.*
    - *Communication/Social: Speaks ~10 words, identifies body parts; feeds self.*
- ***2 Years (24 Months):***
    - *Motor: Runs well, walks up and downstairs one step at a time; builds a tower of 7 cubes, imitates a horizontal stroke.*
    - *Communication/Social: Puts 3 words together; listens to stories with pictures.*
- ***3 Years:***
    - *Motor: Rides a tricycle, stands momentarily on 1 foot; copies a circle.*
    - *Communication/Social: Washes hands, unbuttons clothing, puts on shoes.*

- ***4 Years:***
    - *Motor: Hops on 1 foot, throws a ball overhand; copies a cross and a square, draws a person.*
    - *Communication/Social: Tells a story; goes to the toilet alone, engages in role-playing.*
- ***5 Years:***
    - *Motor: Skips.*
    - *Communication/Social: Counts 10 pennies; dresses and undresses self.*

3. Identifying Delays & Red Flags

- ***Global Developmental Delay (GDD):*** *Defined as significant delay in* ***two or more*** *developmental domains.*
- ***Delay vs. Regression:*** *Delay is a slow progress in attaining milestones, while psychomotor regression is the alarming loss of milestones previously attained.*
- ***Crucial Red Flags:*** *Loss of any skills at any age, no babbling or gesturing by 12 months, no single words by 16 months, and no spontaneous two-word phrases by 24 months.*

4. Diagnostic Approach: Central vs. Peripheral

When evaluating motor delay, determine the origin:

- ***Central (Upper Motor Neuron):*** *Features include normal or brisk reflexes, fisting, scissoring on vertical suspension, seizures, and dysmorphic features.*
- ***Peripheral (Lower Motor Neuron):*** *Features include absent or depressed reflexes, profound weakness, muscle atrophy, fasciculations, and an awake/alert child with intact brain function.*

5. Cerebral Palsy (CP)

CP is a non-progressive (static) disorder of motor function resulting from damage to the developing brain.

- ***Etiology:*** *70-80% of cases are prenatal in origin. While prematurity is the most common known antecedent, the absolute majority of children who develop CP are actually born at term.*

- ***Key Subtypes:***
    - *Spastic (70-80%): Most common type.*
    - *Hemiplegic: Arm is more affected than the leg. Children often tip-toe walk and swing the affected leg.*
    - *Spastic Quadriplegic: Legs are more affected than arms. Associated with opisthotonic posture in infancy, and high rates of mental retardation and seizures.*
    - *Diplegic: Bilateral leg involvement with scissoring.*
- ***Clinical Clues:*** *A primary motor deficit, delayed milestones, and* ***hand preference before 3 years of age****, which indicates a relative weakness on the opposite side.*

6. Physical Exam Clues for Underlying Syndromes

Specific physical features strongly guide the diagnosis of underlying genetic or metabolic conditions:

- ***Skin:*** *Neurofibromatosis Type 1 (macules/spots); Tuberous Sclerosis (ash leaf spots, shagreen patches, sebaceous adenomas).*
- ***Hair:*** *Menkes disease (colorless, friable, kinky hair with low copper); Gricelli syndrome (silvery hair).*
- ***Eyes:*** *Cataracts (Galactosemia, Zellweger); Dislocated lenses (Homocystinuria); Cherry red spot on the retina (lipid storage diseases like Niemann-Pick and Tay-Sachs).*

7. Evaluation & Investigations

If no obvious clinical clues are present, a structured workup is required:

- ***Vision and Hearing:*** *Assessment must be done for* ***all*** *children with developmental delay.*
- ***Lab Tests:*** *Metabolic workup (including thyroid testing).*
- ***Genetic Testing:*** *High-resolution chromosomal microarray is a routine screen (15-20% yield).* ***Whole Exome Sequencing (WES)*** *is highly recommended when initial investigations are negative, boasting a powerful diagnostic yield of approximately 40-44.8%.*
- ***Imaging & EEG:*** *MRI of the brain detects abnormalities in 48-65% of cases. An EEG is* ***not recommended*** *in routine evaluation unless the child is having seizures.*

Common endocrine disorders

1. Diabetes Mellitus (Focus on Type 1)

Definition & Diagnosis *Diabetes mellitus is a metabolic disorder defined by hyperglycemia caused by defective insulin secretion, action, or both. Diagnosis requires meeting at least one of the following criteria:*

- *Fasting Plasma Glucose (FPG) $\ge$ 126 mg/dL.*
- *Random Plasma Glucose $\ge$ 200 mg/dL accompanied by symptoms of diabetes.*
- *2-hour Plasma Glucose during an Oral Glucose Tolerance Test (OGTT) $\ge$ 200 mg/dL.*

Pathophysiology & Genetics *Type 1 Diabetes (T1DM) is an autoimmune disease where immune dysregulation and environmental triggers lead to the destruction of pancreatic beta cells.*

- ***Insulin's Normal Role:*** *Insulin is secreted by pancreatic beta cells. It lowers blood sugar by stimulating protein synthesis and lipogenesis while inhibiting glycogenolysis, gluconeogenesis, lipolysis, and proteinolysis.*
- ***Absence of Insulin:*** *Without insulin, the body improperly breaks down fat and protein and produces excess glucose, leading to high blood sugar. Counter-regulatory hormones (epinephrine, cortisol, growth hormone, and glucagon) further increase glucose levels.*
- ***Progression:*** *Symptoms of T1DM typically appear when only about 10% of beta cells remain functional.*
- ***Genetics & Environment:*** *T1DM has a familial link (30-65% concordance in monozygotic twins, 6% in siblings), but environmental factors heavily influence it, as evidenced by a 50% discordance rate in identical twins and varying incidences in different geographic areas.*

Clinical Manifestations & Complications

- ***Classic Symptoms:*** *Polyuria (excessive urination), polydipsia (excessive thirst), polyphagia (excessive hunger), weight loss, and fatigue.*
- ***Diabetic Ketoacidosis (DKA):*** *A 100% preventable, severe consequence of absolute insulin deficiency. It is diagnosed by blood glucose > 200 mg/dL, pH < 7.3, serum bicarbonate < 18 mmol/L, and the presence of ketones in urine or blood. Symptoms include Kussmaul breathing (deep, labored breathing), lethargy, abdominal pain, vomiting, and confusion.*

- ***Cerebral Edema (CE) Risk in DKA:*** *CE is a dangerous complication. Risk factors include being under 5 years old, new-onset diabetes, high initial urea, low arterial CO2, and the rapid administration of hypotonic fluids or IV insulin boluses.*
- ***Hypoglycemia (Low Blood Sugar):*** *A complication of treatment, characterized by hunger, trembling, sweating, extreme mood changes, dizziness, and blurred vision. It must be confirmed (blood sugar < 72 mg/dL) and treated immediately with carbohydrates.*

Management & Monitoring

- ***Insulin Therapy:*** *Managed via pens or continuous subcutaneous insulin infusion (pumps). Regimens utilize a mix of rapid-acting (Aspart, Lispro, Glulisine), short-acting (Regular), intermediate (NPH), and long-acting (Detemir, Glargine) insulins.*
- ***Monitoring:*** *Regular glucose and ketone monitoring is vital.* ***HbA1c*** *is used to measure the average blood glucose over the preceding 2-3 months.*
- ***Sick Day Rules:*** *During intercurrent illnesses, patients must check ketones early, maintain hydration, and* ***never omit insulin****.*
- ***Long-Term Screening:*** *Screen for retinopathy and nephropathy annually starting at age 11 (after 2 years of disease duration) or at age 9 (after 5 years of duration).*

Other Forms of Diabetes

- ***MODY (Maturity-Onset Diabetes of the Young):*** *A rare (1-2% of cases) genetic defect causing β-cell dysfunction. It features autosomal dominant inheritance, onset between 9-25 years, and a family history spanning at least three generations.*
- ***Wolfram Syndrome:*** *Characterized by the acronym* ***DIDMOAD*** *(Diabetes Insipidus, Diabetes Mellitus, Optic Atrophy, and Deafness). Other issues include neurogenic bladder and neurodegenerative problems like ataxia.*

2. Congenital Adrenal Hyperplasia (CAH)

While CAH involves multiple pathways, the sources specifically highlight the critical presentation of a ***Salt-Losing Crisis in Infancy****:*

- ***Clinical Presentation:*** *This is a life-threatening emergency causing severe hyponatremic dehydration (low sodium), hyperkalemia (high potassium), and metabolic acidosis.*
- ***Management:*** *It requires immediate, aggressive treatment to prevent death.*

- ***Differential Diagnosis:*** *When an infant presents with a salt-losing crisis, causes to consider include CAH, congenital adrenal hypoplasia, isolated aldosterone deficiency, and pseudohypoaldosteronism.*

3. Congenital Hypothyroidism (CH)

Role of Thyroid Hormones *Thyroid hormones are essential for life and development. They increase oxidative metabolism (oxygen consumption, basal metabolic rate, and glucose/fat metabolism), promote growth, augment cardiac function, maintain reproductive health, and are absolutely vital for normal central nervous system (CNS) development and myelination.*

Causes of Congenital Hypothyroidism *Causes are categorized into permanent (primary or central) and transient.*

- ***Permanent Primary Hypothyroidism*** *(Low T4, High TSH):*
    1. ***Thyroid Dysgenesis (85% of cases):*** *Malformation of the gland, including ectopy (abnormal location), agenesis (absence), or hypoplasia (underdevelopment).*
    2. ***Thyroid Dyshormogenesis:*** *Defective hormone synthesis (e.g., TPO mutation), often presenting with a goiter.*
    3. ***TSH Resistance:*** *Rare mutations in the TSH receptor.*
- ***Permanent Central Hypothyroidism*** *(Low T4, Low/Normal TSH):*
    1. ***Developmental Defects:*** *Issues in the pituitary or hypothalamus, sometimes associated with midline defects.*
    2. ***Inactivating Mutations:*** *Involving the TRH receptor, TSH β subunit, or pituitary transcription factors.*
- ***Transient Hypothyroidism*** *(Temporary):*
    1. *Severe iodine deficiency or acute iodine overload.*
    2. *Maternal use of antithyroid drugs (clears 3-4 days after birth).*
    3. *Transplacental transfer of maternal TSH-receptor blocking antibodies.*
    4. ***Hypothyroxinemia of prematurity:*** *Low T4/T3 with normal TSH, which is an adaptation to premature birth rather than true central hypothyroidism.*

Treatment *The standard treatment for hypothyroidism is* ***Levothyroxine*** *replacement to restore normal hormone levels and ensure proper neurological and physical development.*

Vaccines

1. Core Concepts: Vaccination vs. Immunization

- ***Vaccination*** *is the physical act of administering a vaccine (an antigen) into the body.*
- ***Immunization*** *is the body's internal process of building an immune response after being exposed to that antigen.*
- ***Goals:*** *The immediate goal is to prevent disease, while the ultimate goals are the total control and eradication of the disease. Historically, vaccines have reduced diseases like smallpox, polio, and diphtheria by 100% since the 20th century.*

2. Types of Immunity

Immunity is divided into two main categories: Active and Passive.

- ***Active Immunity:*** *The body builds its own protective antibodies after being exposed to an immunogenic antigen. It provides long-term, sometimes lifelong, protection.*
    - *Natural: Getting a disease (infection).*
    - *Artificial: Receiving a vaccination.*
- ***Passive Immunity:*** *The body is given ready-made antibodies (immune globulin) from human or animal sources. This provides temporary protection that decreases over time.*
    - *Natural: A mother passing antibodies to her infant via the placenta or breast milk (protects for about 6 months).*
    - *Artificial: Administering specific immunoglobulins, such as Hepatitis B IG or Varicella IG.*

3. Vaccine Classifications (The "How They're Made" List)

- ***Live Attenuated (Weakened):*** *BCG (Tuberculosis), MMR (Measles, Mumps, Rubella), OPV (Oral Polio), Rotavirus, Varicella (Chickenpox), Yellow fever.*
- ***Killed/Inactivated (Whole Cell):*** *Pertussis, IPV (Inactivated Polio), Influenza (Flu), Hepatitis A.*
- ***Toxoids (Inactivated toxins):*** *Tetanus, Diphtheria.*
- ***Conjugated:*** *Hib, Pneumococcal, Meningococcal.*
- ***Recombinant:*** *Hepatitis B, COVID-19, HPV.*

4. Routes of Administration

- ***Intramuscular (IM):*** *DTaP, Hib, Hepatitis B, Hepatitis A.*
- ***Subcutaneous (Under the skin):*** *MMR, Varicella, IPV.*
- ***Intradermal (Into the skin):*** *BCG.*
- ***Oral (By mouth):*** *OPV, Rotavirus.*
- ***Intranasal (Nose spray):*** *Nasal Influenza vaccine.*

5. General Side Effects

- ***Timing:*** *Most reactions occur early, within 24 to 48 hours of vaccination.*
- ***Common Symptoms:*** *Fever, syncope (fainting), or local reactions at the injection site like pain, swelling, and redness.*
- ***Live Vaccine Specifics:*** *Live attenuated vaccines (like MMR) might cause a mild, delayed version of the disease 5 to 7 days later.*
- ***Severe Reactions:*** *Anaphylaxis (severe allergic reaction) to the vaccine or its components can occur but is rare.*

6. Crucial Details on Specific Vaccines

BCG (Bacillus Calmette–Guérin) - For Tuberculosis

- ***Purpose:*** *Highly effective (80%) at preventing life-threatening, disseminated TB in young children, such as TB meningitis and miliary TB.*
- ***Side Effects:*** *Generally not serious. About 1% experience a localized abscess and swollen lymph nodes. Rarely, it can cause bone infection (osteitis) years later or disseminated infection (2 in 1 million).*

Hepatitis B

- ***Efficacy:*** *90–95% effective, providing protection for 20 years to life.*
- ***Special Protocol:*** *Infants born to Hepatitis B-positive mothers require special management, receiving both the vaccine and Hepatitis B immunoglobulins at birth.*

Hib (Haemophilus Influenzae type B)

- *There are types A through H, but the vaccine specifically targets type B.*

- *Highly recommended for patients with compromised immune systems or splenic dysfunction.*

DTaP Family (Diphtheria, Tetanus, Pertussis)

- ***Variations: DTP** (whole-cell pertussis), **DTaP** (acellular pertussis), **Td** (tetanus + small dose diphtheria), and **Tdap** (tetanus + small dose diphtheria and pertussis).*
- ***Efficacy:** 98–100% effective after 5 doses. Needs a Tetanus/Diphtheria booster every 10 years.*
- ***Side Effects:***
    - *Mild: Fussiness, poor appetite, vomiting (1 in 50).*
    - *Moderate: Non-stop crying for 3+ hours (1 in 1,000), high fever, or seizures (1 in 14,000).*
    - *Severe: Serious allergic reaction (1 in a million), long-term seizures, coma, or permanent brain damage.*
- ***Contraindications:** Do not give if the patient had an anaphylactic reaction or developed encephalopathy (coma, decreased consciousness) within 7 days of a previous dose.*

Pneumococcal

- *Comes in **Conjugated** forms and a **Polysaccharide** form (which protects against 23 different bacteria strains).*
- ***Special Indications:** Highly recommended for high-risk patients, including those with cystic fibrosis, chronic lung disease, cochlear implants, sickle cell disease, missing/removed spleens (asplenia), nephrotic syndrome, cerebrospinal fluid leaks, or compromised immune systems.*

Meningococcal

- ***Coverage:** Tetravalent vaccines (both polysaccharide MPSV4 and conjugate MCV4) protect against Neisseria Meningitis serogroups A, C, Y, and W-135. Notably, serogroup B is the most lethal.*
- ***Special Indications:** Recommended for travelers going to endemic/epidemic areas, and individuals with impaired immunity (e.g., complement deficiencies, splenectomy, or nephrotic syndrome causing immunoglobulin loss).*

Well Newborn and Exam

The Golden Hour (First 60 Minutes)

The first hour of a newborn's life is highly critical for ensuring a healthy transition from intrauterine to extrauterine life. The foundational care protocol follows seven chronological steps:

- ***Step 1: Thermal Care.*** *Immediately dry the baby, provide a hat, and use very gentle stimulation. The goal is to maintain a normal body temperature between* ***36.5–37.5°C****.*
- ***Step 2: Delayed Cord Clamping (DCC).*** *Wait 1–3 minutes (or until pulsation stops) before clamping the cord. This provides the infant with up to 80% of the placental cord blood, resulting in* ***increased red cell volume, higher iron stores****, and a significantly lower risk of anemia, necrotizing enterocolitis (NEC), and intraventricular hemorrhage (IVH).*
- ***Step 3: Skin-to-Skin Contact.*** *Place the unclothed newborn directly on the mother's bare chest for at least 60 minutes. This prevents hypothermia and hypoglycemia, significantly increases exclusive breastfeeding rates, and reduces newborn crying and maternal stress.*
- ***Step 4: Initial Newborn Assessment.*** *While the baby is skin-to-skin, conduct the* ***Apgar score at 1 and 5 minutes****, take vital signs, and perform a routine exam. An Apgar score of 7 to 10 is normal; it helps assess the infant's transition to life but should not be used to predict future neurological outcomes.*
- ***Step 5: Early Initiation of Breastfeeding.*** *Begin breastfeeding within the first hour to establish a strong feeding relationship.*
- ***Step 6: Postpone Non-Urgent Procedures.*** *Delay weighing, bathing, and injections until after the first 60 minutes to preserve uninterrupted bonding and avoid stressing or cooling the baby.*
- ***Step 7: Vitamin K Administration.*** *Administer an intramuscular Vitamin K injection. All neonates are born with dangerously low Vitamin K levels due to immature gut bacteria, poor placental transfer, and low levels in breast milk. This injection is crucial to prevent* ***Vitamin K-Deficiency Bleeding (VKDB)****, which can cause fatal intracranial hemorrhages.*

Routine Assessment and Growth (First 24–48 Hours)

Following the initial delivery room care, the pediatric clinician conducts a comprehensive examination within the first 24 to 48 hours to identify any anomalies, assess risks, and reassure the parents.

- ***Growth Measurements:*** *You must measure weight, length (average 46–56 cm), and head circumference (average 33–35 cm).*
- ***Growth Charts:*** *Use appropriate charts depending on the infant:* ***WHO charts*** *for breastfed term infants,* ***CDC charts*** *for formula-fed term infants, and* ***Fenton or Intergrowth charts*** *for premature infants.*
- ***Weight Fluctuations:*** *It is completely normal for newborns to lose weight initially (up to 5% for vaginal births and 10% for C-section births). Babies should* ***regain their birth weight by 10 to 14 days*** *of life. After reaching their lowest weight, healthy term babies gain an average of about 30 grams per day in the first month.*

Normal Bodily Functions: Voiding and Stooling

A newborn should never be discharged until the passage of both urine and stool has been officially documented.

- ***Urine Pattern:*** *Infants should have their first urination within the first 12 hours; if there is no urine by 24 hours, it requires investigation. Afterward, passing urine 4–6 times per day indicates adequate hydration. It is common to see* ***"brick dust"*** *in the diaper—these are harmless urate crystals and should not be mistaken for blood.*
- ***Stool Pattern:*** *The first stool is called* ***meconium*** *(black, tarry, and sticky) and usually passes within the first 48 hours. If delayed beyond 48 hours, the infant must be evaluated for colonic obstructions like Hirschsprung disease or an imperforate anus. As breast milk intake increases, the stool transitions to a seedy, mustard-yellow appearance.*

Common Risks and Daily Hygiene Care

- ***Hypoglycemia (Low Blood Sugar):*** *High-risk infants include those born to diabetic mothers (IDM), those who are small or large for gestational age (SGA/LGA), preterm infants, and infants who experienced perinatal stress. Symptomatic babies (sweating, jittery, hypotonic) must be screened.*

- ***Skin Care and Bathing:*** *The World Health Organization recommends* ***delaying the first bath for at least 6 to 24 hours****. This prevents hypothermia and retains the* ***vernix caseosa*** *(the white substance on newborn skin), which acts as a natural moisturizer, prevents water loss, and has antimicrobial properties. When bathing, keep it under 10 minutes and limit baths to 2-3 times per week to prevent skin drying.*
- ***Umbilical Cord Care:*** *Keep the cord clean and dry.* ***Avoid routine antiseptics*** *like alcohol or chlorhexidine (unless in a high-mortality region), as these actually increase the time it takes for the cord to separate.*
- ***Genital Care:*** *For uncircumcised males,* ***never retract the foreskin****, as this will cause pain, bleeding, and adhesions.*

Discharge, Safety, and Anticipatory Guidance

Before a baby goes home, parents must be thoroughly educated on safety and preventative care.

- ***Safe Sleep (SIDS Prevention):*** *Babies must sleep* ***alone, on their backs, in a bare crib****. Parents should share a room with the infant for at least the first 6 months, but* ***bed-sharing is strictly prohibited****. Keep the crib entirely free of quilts, sleep positioners, and soft toys, and never allow the baby to sleep routinely in sitting devices like car seats.*
- ***Environmental Hazards:*** *Absolutely* ***no tobacco smoke exposure****, as it drastically increases the risk of Sudden Infant Death Syndrome (SIDS), respiratory infections, and asthma.*
- ***Car Seat Safety:*** *Car seats must be appropriately sized, rear-facing, and secured in the back seat at the correct angle.*
- ***Mandatory Newborn Screening:*** *Before discharge, babies must undergo:*
    - ***Hearing Screening****.*
    - ***CCHD (Critical Congenital Heart Disease) Screening:*** *Pulse oximetry is placed on the right hand and either foot to check for cyanotic heart defects.*
    - ***Metabolic Screening:*** *A blood spot test to catch metabolic disorders (like PKU, G6PD, and TSH) early before significant morbidities occur.*
    - ***Immunizations:*** *The Hepatitis B vaccine should be administered within 24 hours of birth to prevent chronic infection.*

Pre-term

The Basics: Understanding Prematurity and its Global Burden

*A **preterm baby** is defined as an infant born alive before completing 37 weeks of gestation. These births are categorised by gestational age into **extremely preterm** (less than 28 weeks), **very preterm** (28 to less than 32 weeks), and **moderate to late preterm** (32 to 37 weeks).*

Weight classifications are equally important and include:

- ***Low Birth Weight (LBW):** Less than 2,500 grams.*
- ***Very Low Birth Weight (VLBW):** Less than 1,500 grams.*
- ***Extremely Low Birth Weight (ELBW):** Less than 1,000 grams. Infants are further assessed by comparing their weight to their gestational age, being classed as Appropriate for Gestational Age (AGA), Small for Gestational Age (SGA), or Large for Gestational Age (LGA). To determine gestational maturity physically and neurologically, clinicians use the **New Ballard Score**, which is most accurate when performed within the first 12 hours for babies under 26 weeks, or up to 96 hours for older preterm infants.*

*The burden of prematurity is massive: an estimated 13.4 million babies were born preterm globally in 2020. It is the **leading cause of death among children under 5**, causing around 900,000 deaths in 2019, though 75% of these deaths could be prevented with cost-effective interventions. In Jordan, it is the second leading cause of neonatal mortality, resulting in staggering healthcare costs and high rates of disability (60% disability rate for babies born before 26 weeks).*

Physical Characteristics of a Preterm Baby

- *Small size (often under 2,500g) with very little body fat.*
- *Pink or red, almost transparent skin with visible veins.*
- *Abundant fine body hair (**lanugo**) but little scalp hair.*
- *Weak cry and poor muscle tone.*
- *Underdeveloped genitals and soles of the feet without well-formed creases.*
- *Small or absent breast nodules.*

Causes and Risk Factors *Prematurity is triggered by various factors spanning three main categories:*

- ***Maternal Factors:*** *Previous preterm deliveries, maternal infections (such as group B streptococcus or UTIs), chronic illnesses, preeclampsia, malnutrition, anemia, and drug abuse.*
- ***Uterine/Pregnancy Factors:*** *Placental issues (placenta previa or abruption), premature rupture of membranes (pPROM), excess amniotic fluid (polyhydramnios), or cervical incompetence.*
- ***Fetal Factors:*** *Fetal distress, multiple gestations (twins/triplets), congenital malformations, and intrauterine (TORCH) infections.*

Prevention and Management Before Birth

If a mother is at risk of preterm labor, specific medical interventions can delay delivery and dramatically improve the baby's survival chances:

- ***Cervical Cerclage:*** *Offered if a transvaginal ultrasound shows the cervix has shortened to under 25 mm before 24 weeks.*
- ***Progesterone:*** *Given to women carrying a single baby with a history of preterm birth and a shortened cervix to reduce preterm delivery rates.*
- ***Antibiotics:*** *Administered for prolonged rupture of membranes (pPROM) to delay delivery and reduce newborn illness.*
- ***Neuroprotection: Magnesium Sulphate (MgSO4)*** *is given to mothers before 32 weeks if delivery is imminent; it reduces the baby's risk of cerebral palsy by about 30%.*
- ***Antenatal Steroids:*** *A single course is given between 24 and 34 weeks (ideally 24 hours before birth) to accelerate fetal lung development.*
- ***In Utero Transfer:*** *Mothers at risk of delivering before 29-30 weeks should be transferred to a hospital with a Level III Neonatal Intensive Care Unit (NICU).*

Delivery Room Management (The Golden Minutes)

Immediate care upon delivery focuses heavily on stabilizing temperature and breathing:

- ***Temperature Control:*** *The delivery room should be 24-26°C. For babies under 28 weeks, clinicians use plastic bags or occlusive wrapping under radiant warmers and hats to prevent hypothermia.*

- ***Delayed Cord Clamping (DCC):*** *Clamping the umbilical cord should be delayed by* ***20 to 45 seconds*** *after the initiation of respiration. This is a crucial step that reduces intraventricular hemorrhage (brain bleeding) by 2-to-3-fold, reduces the need for blood transfusions, improves blood pressure, and significantly cuts mortality rates.*
- ***Respiratory Support:*** *If the baby is in respiratory distress but breathing spontaneously with a heart rate over 100,* ***CPAP*** *(Continuous Positive Airway Pressure) is applied via a mask or nasal prongs. Target oxygen saturation levels are strictly monitored, starting at 60-65% at 1 minute of life and gradually rising.*

The Unstable Stage (Birth to 3-5 Days)

- ***Hypothermia Risk:*** *Preterm babies lose heat rapidly due to a high surface-area-to-volume ratio, lack of insulating fat, inability to shiver, and immature skin. Hypothermia (temp <36.5°C) can be fatal, triggering hypoxia, hypoglycemia, and metabolic acidosis. They must be kept in a neutral thermal environment (36.5-37.4°C).*
- ***Respiratory Distress Syndrome (RDS):*** *Caused by immature lungs and a lack of* ***surfactant*** *(a substance that keeps air sacs open). Management requires ventilation, oxygen, and directly administering surfactant (such as via the LISA method). Complications can include pulmonary hemorrhage or air leaks (pneumothorax).*
- ***Cardiovascular Issues:*** *Often suffer from hypotension and* ***Patent Ductus Arteriosus (PDA)****, a blood vessel that fails to close, leading to congestive heart failure. PDA is treated with fluid restriction, NSAIDs, Paracetamol, or surgical closure.*
- ***Metabolic & Skin Fragility:*** *Highly prone to fluid loss, electrolyte imbalances (Sodium, Potassium, Calcium), and glucose instability. Their skin is so fragile that standard medical tapes cannot be used; hydrogel tapes are required.*
- ***Infections:*** *Extremely susceptible to hospital-acquired infections due to compromised immune systems (decreased T-cells, B-cells, and antibodies).*
- ***Nutrition & Gastrointestinal Risks:*** *Because they cannot coordinate sucking and swallowing until 34 weeks, they are fed via tubes or IV (parenteral nutrition). Early "trophic" feeding (tiny amounts of breast milk) primes the gut. They are at severe risk for* ***Necrotizing Enterocolitis (NEC)****, a deadly condition where intestinal tissue dies due to hypoxia, poor blood flow, and bacterial infections.*

The Stable Stage and Long-Term Complications (> 5 Days)

- ***Apnea of Prematurity:*** *Cessation of breathing for more than 20 seconds, or a shorter pause accompanied by a dropped heart rate or oxygen level. It is treated with caffeine/theophylline (for central apnea) or CPAP (for obstructive apnea).*
- ***Neurological Damage:*** *High risk of* ***Intraventricular Hemorrhage (IVH)*** *(bleeding in the brain), which can lead to hydrocephalus (fluid buildup), and* ***Periventricular Leukomalacia (PVL)*** *(softening of brain tissue), leading to long-term neurodevelopmental delays.*
- ***Retinopathy of Prematurity (ROP):*** *Incomplete retinal blood vessel growth that can lead to blindness.*
- ***Chronic Lung Disease (CLD) / Bronchopulmonary Dysplasia (BPD):*** *Long-term lung damage caused by prolonged mechanical ventilation, oxygen toxicity, and early lung inflammation.*
- ***Metabolic Bone Disease of Preterm (MBDP):*** *Weak bones due to a lack of calcium and phosphorus usually acquired in the final trimester. Treated with vitamin D, breast milk fortifiers, and careful physical handling.*
- ***Anemia of Prematurity:*** *Driven by shortened red blood cell lifespans, frequent blood draws, and inadequate new cell production.*

Going Home: Discharge Criteria and Follow-up

A preterm baby is only ready to be discharged from the hospital when several strict milestones are met:

- *All serious illnesses are resolved.*
- *They can maintain a stable body temperature in an open crib without a warming incubator.*
- *They can take all necessary feedings by breast or bottle.*
- *There are no recent episodes of apnea or low heart rate.*
- *They have reached at least 35 weeks corrected gestational age and weigh more than 1.8 to 2 kg.*
- *Parents are fully trained to provide care, administer medications, and have received CPR training.*

Infant Nutrition

1. Breast Milk

The Gold Standard *Breast milk is uniquely composed to promote healthy growth, immunity, and development.*

- ***Key Components:*** *It consists of roughly 87% water, 7% lactose, 4% fat, 0.9% protein (a specific ratio of whey to casein), and 0.5%–1.5% Human Milk Oligosaccharides (HMOs). It is packed with hormones, digestive enzymes, anti-inflammatory and antimicrobial factors (like Secretory IgA), and growth factors.*
- ***Human Milk Oligosaccharides (HMOs):*** *HMOs are the third most common solid constituent in human milk after lactose and lipids. Built on lactose, there are over 200 identified structures, with* ***2'-Fucosyllactose (2'-FL)*** *being the most abundant.*

2. The Unmatched Benefits of Breast Milk For the Infant:

- ***Infection Protection:*** *Breastfed children have a substantially lower risk of suffering and dying from infectious diarrhea and respiratory infections.*
- ***Premature Infant Survival:*** *Preterm infants receiving human milk have a 6- to 10-fold lower risk of developing necrotizing enterocolitis (NEC), a deadly intestinal disorder.*
- ***Lifelong Health:*** *Lowers the risk of overweight/obesity, type 1 diabetes, childhood asthma, and childhood leukemia, while contributing to a higher IQ.*

For the Mother & Family:

- ***Maternal Health:*** *Aids in post-delivery weight loss, uterine contraction, and clearing pregnancy hormones. It lowers the mother's risk of diabetes, cardiovascular disease (CVD), and breast cancer.*
- ***Practicality:*** *It acts as a natural contraceptive (Lactational Amenorrhea Method - LAM), is available 24/7, is cheaper than formula, requires no preparation, and promotes mother-baby bonding.*

3. Infant Formulas:

Breaking Down the Basics *Infants require at least* ***100 kcal/kg/day****. Regular formula and breast milk generally contain* ***67 kcal/100 ml*** *(or 20 Kcal/oz). Formulas are classified by their three main macronutrients:*

- ***Protein Content:*** *Progresses from least to most hypoallergenic:*
    1. *Cow's milk-based: Standard for a normal GI tract.*
    2. *Soy: For cow's milk allergy (in older babies), lactose malabsorption, or galactosemia.*
    3. *Casein Hydrolysate (extensive vs. partial): For cow's milk/soy allergies or malabsorption.*
    4. *Amino Acid-based: For severe protein allergies not responsive to hydrolysate formulas.*
- ***Carbohydrate Content:*** *Includes lactose, sucrose, or glucose polymers. Crucial rule: Patients with galactosemia must use lactose-free formulas.*
- ***Fat Content:*** *Utilizes Long-Chain Triglycerides (LCTs) or Medium-Chain Triglycerides (MCTs). MCTs are highly beneficial for infants with impaired fat absorption or lymphatic abnormalities.*

4. Cow's Milk Protein Allergy (CMPA)

CMPA is the leading cause of food allergy in children under 3 years old.

- ***Symptoms:*** *Involves the GI tract (vomiting, diarrhea, blood in stool), skin (urticaria, eczema, angioedema), and respiratory tract (wheezing, runny nose). The involvement of more than two systems greatly increases the probability of CMPA.*
- ***Diagnosis:*** *Primarily based on history and physical exams. Skin prick tests (SPT) or specific IgE tests might be negative, especially in kids who only have GI symptoms. An oral challenge test is required to confirm the diagnosis.*
- ***Treatment:***
    - *Breastfed infants: The mother must start a strict cow's milk protein-free diet.*
    - *Formula-fed infants: Switch to an extensively hydrolyzed protein formula (or amino-acid based for severe cases). Soy formula is an option, but only if the infant is older than 6 months.*

- ***Prognosis:*** *Patients should be reevaluated every 6 to 12 months. The allergy is usually outgrown; >75% develop tolerance by age 3, and >90% by age 6. Unnecessarily long dietary eliminations should be avoided as they impair quality of life and growth.*

5. Practical Approach to Choosing Formulas

Choosing a formula is exactly like choosing an antibiotic.

1. ***Identify the target:*** *Determine the exact cause of the baby's intolerance (Is it the protein, the carbohydrate/sugar, or the fat?).*
2. ***Pick the coverage:*** *Switch to a formula tailored to that condition (e.g., modified protein, lactose-free, or MCT fat).*
3. ***Evaluate:*** *Assess the infant's response to the new formula and decide if further changes are needed.*

Neonatal Jaundice

1. Core Definitions & The Main Danger

- ***Neonatal Jaundice:*** *Yellowish discoloration of the skin and/or conjunctiva caused by bilirubin deposition.*
- ***Significant Hyperbilirubinemia:*** *Total serum bilirubin (TSB) > 95th percentile on the hour-specific Bhutani nomogram.*
- ***Severe Hyperbilirubinemia:*** *TSB > 25 mg/dL (425 micromol/L) in term newborns.*
- ***The Danger (BIND):*** *High levels of unconjugated bilirubin can cross the blood-brain barrier and cause* ***Bilirubin-Induced Neurologic Dysfunction (BIND)****.*
    - ***Acute Bilirubin Encephalopathy (ABE):*** *The acute phase, presenting with altered consciousness, lethargy, poor feeding, shrill cry, hypertonia (retrocollis, opisthotonus), and seizures.*
    - ***Kernicterus:*** *The permanent, chronic sequelae of irreversible brain damage caused by BIND.*

2. The "Why": Bilirubin Metabolism in Neonates

To understand jaundice, you must understand how newborns process bilirubin differently than adults:

- ***Increased Production:*** *80-90% of bilirubin comes from the breakdown of hemoglobin in old or injured red blood cells (RBCs). Neonates have more RBCs and a shorter RBC lifespan.*
- ***Decreased Clearance (Immature Liver):*** *Unconjugated (lipid-soluble) bilirubin must be bound to albumin to travel to the liver. In the liver, the enzyme* ***UGT1A1*** *converts it into conjugated (water-soluble) bilirubin. Neonates have immature liver function with less UGT1A1 activity.*
- ***Increased Enterohepatic Circulation (EHC):*** *Conjugated bilirubin moves to the intestines. However, neonates have an enzyme called beta-glucuronidase in their gut that deconjugates it back into unconjugated bilirubin, which is then reabsorbed into the bloodstream.*

3. The 4 Main Types of Neonatal Jaundice

A. Physiologic Jaundice (Normal & Harmless)

- ***Cause:*** *Due to normal newborn physiology (high RBC turnover, immature liver, increased EHC).*
- ***Pattern:*** *Appears after 24 hours. Peaks at 3-4 days in term infants (5-7 days in preterms) and disappears by 1-2 weeks.*
- ***Rules:*** *The baby is otherwise healthy, the TSB rise is slow (< 0.2 mg/dL/hour or < 5 mg/dL/day), and peak levels usually stay below 15 mg/dL.*

B. Breastfeeding Jaundice (BFJ)

- ***Onset:*** *First few days of life.*
- ***Cause:*** *Not enough milk! It is a misnomer and actually means "suboptimal intake". It leads to mild dehydration, delayed bowel movements (which increases EHC), and significant weight loss (>8% of birth weight).*
- ***Management:*** *Improve and increase breastfeeding frequency (every 2-3 hours). Do not give water or dextrose.*

C. Breast Milk Jaundice (BMJ)

- ***Onset:*** *Develops later, typically after 4-7 days, and can last for 3 to 12 weeks.*
- ***Cause:*** *Believed to be caused by inhibitory substances in breast milk that interfere with the UGT1A1 enzyme, or high levels of beta-glucuronidase in the milk.*
- ***Note:*** *Very rarely causes BIND unless TSB > 25 mg/dL, and affects healthy, thriving babies.*

D. Pathologic Jaundice (Medical Emergency)

- ***Red Flags (How to Recognize):***
    1. *Appears in the **first 24 hours of life**.*
    2. *Rises rapidly (> 0.2 mg/dL/hour or > 5 mg/dL/day).*
    3. *Direct (conjugated) bilirubin is > 1 mg/dL.*
    4. *Jaundice lasts longer than 2 weeks in a term newborn.*
    5. *The baby shows signs of illness (lethargy, vomiting, temperature instability).*

- ***Common Causes:***
    - *Increased Production: Hemolysis (Rh/ABO incompatibility), G6PD deficiency, spherocytosis, polycythemia, or enclosed bleeding (cephalohematoma).*
    - *Decreased Clearance: Crigler-Najjar syndrome, Gilbert syndrome, Galactosemia, or Congenital Hypothyroidism.*

4. Prolonged Jaundice (Lasting > 14 days)

- *If jaundice persists beyond 14 days, you **must check direct (conjugated) bilirubin**.*
- ***Why it's crucial:** Elevated direct bilirubin (> 1 mg/dL) indicates cholestatic liver disease, such as **Biliary Atresia**, which requires urgent surgical intervention for a good outcome. Other causes include neonatal hepatitis and metabolic/infectious diseases.*

5. Risk Assessment & Monitoring

- ***Universal Screening:** All babies ≥12 hours old must have TSB or transcutaneous bilirubin (TcB) measured before discharge. If discharged before 12 hours, a check must be arranged for the first day.*
- ***The Nomogram:** Doctors use the hour-specific **Bhutani Nomogram** to plot bilirubin levels and determine if the baby is in a Low, Intermediate, or High-Risk zone.*
- ***The "Rule of 30":** Used to calculate the difference (Δ-TSB) between the baby's actual bilirubin and the threshold for phototherapy to decide if urgent action, early follow-up, or routine follow-up is needed.*
- ***Major Risk Factors:** Gestational age < 38 weeks, previous sibling needing phototherapy, visible jaundice in the first 24 hours, exclusive breastfeeding with poor intake, hemolytic disease (e.g., G6PD), and significant bruising/cephalohematoma.*

6. Management & Treatment Strategies

- ***Phototherapy:*** *The primary treatment. It exposes the baby's skin to blue/cool white light (425-475 nm wavelength). This light causes an isomerization reaction, changing the toxic bilirubin into a non-toxic, water-soluble form (lumirubin) that can be excreted.*
- ***Intravenous Immune Globulin (IVIG):*** *Used for isoimmune hemolytic disease (like ABO/Rh incompatibility) if bilirubin keeps rising despite phototherapy, or if levels are close to exchange transfusion thresholds.*
- ***Exchange Transfusion:*** *A critical intervention used when TSB > 25 mg/dL, when the baby is not responding to phototherapy, or if there are clinical signs of Acute Bilirubin Encephalopathy.*

Neonatal Ophthalmologic Emergencies

- ***Congenital Nasolacrimal Duct Obstruction (CNLDO):*** *Common (20% of infants). Presents as a watery eye with mucoid discharge. Usually resolves by 1 year. Treated with observation, cleaning, and Crigler massage (10 times, 3x/day).*
- ***Primary Congenital Glaucoma (PGG): Urgent surgical emergency.*** *Triad of symptoms: tearing, photophobia (light sensitivity), and blepharospasm. Cornea appears cloudy and enlarged (buphthalmos).*
- ***Abnormal Red Reflex:*** *A white spot (leukocoria) or asymmetrical reflex is highly abnormal. It can indicate a* ***Congenital Cataract*** *(caused by genetics, rubella, or metabolic disorders like galactosemia) or* ***Retinoblastoma*** *(eye cancer). Red reflex must be checked before newborn discharge.*

Seizures

PART 1: SEIZURES & EPILEPSY

Definitions & Core Concepts

- ***Seizure:*** *A transient occurrence of signs or symptoms caused by abnormal, excessive, or synchronous neuronal brain activity.*
- ***Epilepsy:*** *A brain disease defined by any of the following:*
    1. *At least two unprovoked seizures occurring >24 hours apart.*
    2. *One unprovoked seizure with a >60% risk of recurrence over the next 10 years.*
    3. *Diagnosis of a specific epilepsy syndrome.*
- ***Evaluation:*** *Focus on history (video recordings, tone changes, altered awareness, triggers) and look for provoking factors like hypoglycemia or infections.*
    - ***EEG:*** *Needed for unprovoked seizures to determine recurrence risk (40% if normal, 60% if abnormal).*
    - ***Brain MRI:*** *Indicated if there is a concern for a focal issue based on history or exam.*

Epilepsy Syndromes (Organized by Age of Onset)

- ***1. Infantile Spasms (West Syndrome)***
    - ***Age/Symptoms:*** *Infancy; presents with sudden spasms (jackknife seizures).*
    - ***EEG: Hypsarrhythmia*** *(disorganized, discontinuous, high amplitude, multifocal spikes).*
    - ***Treatment:*** *Steroids (ACTH, Prednisone) or Vigabatrin.*
    - ***Prognosis:*** *Frequently leads to developmental delay; early treatment improves outcomes.*

- ***2. Lennox-Gastaut Syndrome (LGS)***
    - ***Age/Symptoms:*** *Childhood (<8 years); features multiple seizure types (tonic, myoclonic, atypical absence).*
    - ***EEG:*** *Severely abnormal (slow spike-wave).*
    - ***Treatment:*** *Valproate, Clobazam, Lamotrigine. Refractory to treatment and associated with severe intellectual dysfunction.*
- ***3. Childhood Absence Epilepsy (CAE)***
    - ***Age/Symptoms:*** *Childhood (4-8 years); sudden staring and activity interruption lasting seconds; easily provoked by* ***hyperventilation****.*
    - ***EEG:*** *Generalized* ***3 Hz spike-wave*** *discharges.*
    - ***Treatment/Prognosis:*** *Ethosuximide, Lamotrigine, Valproate; typically resolves by adolescence.*
- ***4. Benign Rolandic Epilepsy (BECTS)***
    - ***Age/Symptoms:*** *Childhood (4-11 years); focal seizures causing mouth/tongue paresthesia, face jerking, and excessive drooling, usually when awake or upon arousal from sleep.*
    - ***EEG:*** *Centrotemporal spikes.*
    - ***Treatment/Prognosis:*** *Carbamazepine, Oxcarbazepine; good prognosis, typically resolving by puberty.*
- ***5. Juvenile Myoclonic Epilepsy (JME)***
    - ***Age/Symptoms:*** *Adolescence (>12 years); myoclonic jerks mostly affecting the arms upon awakening; provoked by* ***sleep deprivation and flashing lights****.*
    - ***EEG:*** *Fast spike-wave (4-6 Hz).*
    - ***Treatment:*** *Lamotrigine, Valproate; requires life-long treatment.*

Special Seizure Categories

- ***Neonatal Seizures:*** *Highest incidence in life, presenting in 5 main types: subtle (common in preterm babies, like eye deviations or apnea), clonic, tonic, spasms, and myoclonic. Etiologies vary by day of life (e.g., HIE in days 1-4, infection in days 4-14).* ***Phenobarbital*** *is the first-line treatment.*
- ***Febrile Seizures (Ages 6 months to 5 years):***
    - ***Simple:*** *Lasts <15 minutes, generalized, does not recur within 24 hours.*
    - ***Complex:*** *Lasts >15 minutes, focal, or recurs within 24 hours.*
    - *Management: Treat the underlying fever etiology; daily prophylactic anti-seizure medications and scheduled antipyretics are not recommended. Rescue Diazepam is used for seizures >4 minutes.*
- ***Status Epilepticus (SE):*** *Ongoing seizure for* ***>5 minutes*** *or recurrent seizures with no return to baseline for* ***>30 minutes****.*
    - *Timeline: 0-5 mins: IV Benzodiazepine (Diazepam/Midazolam) -> 5-10 mins: 2nd dose -> 10-15 mins: Fosphenytoin/Phenobarbital -> >30 mins: Anesthesia consult.*

Epilepsy Treatment Overview

- ***Medications:*** *Aim for the lowest effective dose of monotherapy. 70% of patients become seizure-free on one drug, 15% on polypharmacy, and 15% remain drug-resistant (failing 2+ appropriate drugs).*
- ***Drug-Resistant Epilepsy (DRE) Treatments:*** *Ketogenic diet, Vagal nerve stimulation, or Epilepsy surgery.*

PART 2: EPILEPSY IMITATORS (Paroxysmal Events)

Conditions that mimic seizures but are fundamentally different:

- ***Syncope/Breath-Holding Spells:*** *Often seen in pre-schoolers; triggered by crying, leading to apnea, cyanosis, or pallor, and sometimes transient syncope/tonic posturing. Worsened by* ***iron deficiency anemia****.*
- ***Cardiac Syncope:*** *Warrants investigation (like Long QT syndrome) if syncope happens during sleep, swimming, fright, or with a strong family history of sudden death.*
- ***Infantile Gratification:*** *Rhythmic hip flexion and adduction accompanied by a flushed face, commonly seen in pre-school girls.*

- ***Tics vs. Stereotypies:***
    - *Tics: Semi-voluntary, rapid movements/vocalizations with an urge, can be suppressed.*
    - *Stereotypies: Semi-voluntary but non-rhythmic, often involve arms/legs, have no urge, can be interrupted by distraction, and are common in autism.*
- ***Sandifer Syndrome:*** *Associated with GERD; presents as arching of the back, dystonic posturing, and head tilting, often after feeding.*
- ***Episodic Migraine Syndromes:*** *Includes cyclical vomiting (predictable, severe N/V resolving by age 10) and benign paroxysmal vertigo (brief attacks of vertigo, nystagmus, ataxia).*

PART 3: PEDIATRIC HEADACHE

Headache affects up to 80% of children by age 15.

Primary Headaches

- ***Migraine:*** *Affects 5% of children. Presents as a throbbing headache lasting hours to days, accompanied by nausea, vomiting, photophobia, phonophobia, and fatigue.*
    - *Treatment: Lifestyle modifications (sleep, hydration, no caffeine), rescue medications (NSAIDs, Triptans), and prophylactic meds (Propranolol, Topiramate, Amitriptyline).*
- ***Tension Headache:*** *Affects 5-10% of children. Presents as a "featureless" bilateral, pressing/tightening headache of mild to moderate intensity.*
    - *Treatment: Lifestyle modifications and NSAIDs; prophylaxis is usually not needed.*

Secondary Headaches (Red Flags) *Always investigate headaches presenting with the following warning signs:*

- *Headache that is side-locked, wakes the child from sleep, or worsens with lying down, standing up, coughing, or Valsalva maneuvers.*
- *Accompanying focal neurological deficits, visual field defects, or altered mental status.*
- *Signs of high brain pressure (Idiopathic Intracranial Hypertension, Tumors, Bleeding, Sinus Thrombosis) which may show papilledema, empty sella, or slit-like ventricles on imaging.*

Meningitis

1. Pathophysiology & Risk Factors

- ***How it Happens:*** *Bacteria enter the subarachnoid space either through the bloodstream (from respiratory, gastrointestinal, or genital tracts) or via direct entry (e.g., trauma, neurosurgery, sinusitis).*
- ***Why it Damages the Brain:*** *The cerebrospinal fluid (CSF) has very low levels of immune defenses (immunoglobulins and complement) compared to blood. This allows bacteria to multiply, triggering a massive inflammatory response that injures the blood-brain barrier, causing brain edema, localized ischemia, and neuronal damage.*
- ***Who is at Risk:*** *The highest incidence is in babies under 2 months old. Other risk factors include immunodeficiency, lack of a spleen, anatomical brain/spinal defects, recent respiratory or ear infections, and exposure to endemic areas.*

2. The Bugs (Microbiology by Age)

- ***Neonates (under 3 months): Group B Streptococcus (GBS)*** *and* ***E. coli*** *are the most common culprits, causing 65–75% of early-onset cases.*
- ***Older Infants & Children: Streptococcus pneumoniae****,* ***Neisseria meningitidis****, and* ***Haemophilus influenzae (Hib)*** *are the primary causes.*
- ***Adolescents: N. meningitidis*** *is the most common pathogen.*

3. Clinical Presentation (Symptoms)

- ***Neonates & Young Infants:*** *Symptoms are often non-specific. Look for poor feeding, vomiting, temperature instability, irritability, lethargy, respiratory distress, or seizures.* ***Note:*** *Classic signs like a bulging fontanelle and nuchal rigidity (stiff neck) are uncommon in neonates.*
- ***Children & Adolescents:*** *Watch for classic signs of meningeal irritation:*
    - *Fever, headache, and photophobia.*
    - ***Nuchal rigidity:*** *Inability to touch the chin to the chest.*
    - ***Kernig sign:*** *Inability to extend the knee past 135° when the hip is flexed.*
    - ***Brudzinski sign:*** *Attempting to flex the neck causes the patient to involuntarily flex their hips/knees.*

- ***Other Red Flags:*** *Rashes (especially the purpuric/petechial rash seen with meningococcal infections), altered mental status, signs of increased intracranial pressure (ICP), and focal neurologic deficits.*

4. Diagnosis & CSF Profile

A complete evaluation includes blood cultures, blood counts, and critically, a Lumbar Puncture (LP) to analyze the CSF. An LP is contraindicated if the patient is unstable, has signs of increased ICP, papilledema, or a focal neurologic deficit (in which case, neuroimaging must be done first).

- ***Bacterial Meningitis CSF Profile:***
    - ***WBCs:*** *Very high (>1000–5000 cells/microL) with mostly* ***neutrophils (>80%)****.*
    - ***Protein:*** *Elevated (100–500 mg/dL).*
    - ***Glucose:*** *Very low (<40 mg/dL, or a CSF-to-serum glucose ratio of <0.4).*

5. Treatment & Management

Antibiotics must be initiated ***immediately*** *after the LP.*

- ***Empiric Antibiotics by Age:***
    - *Neonates (<72 hours): Ampicillin + Aminoglycoside.*
    - *Infants (8 to 28 days): IV Ampicillin + Ceftazidime.*
    - *Infants (29 to 60 days): Ceftriaxone + Vancomycin.*
    - *Children: Vancomycin + Ceftriaxone (or Cefotaxime).*
- ***Duration of Therapy:*** *Depends on the bug. N. meningitidis (5-7 days), Hib (7-10 days), S. pneumoniae (10-14 days), GBS (14 days), and Listeria/Gram-negative bacilli (21+ days).*

6. The Crucial Role of Dexamethasone (Steroids)

- ***Who gets it:*** *Infants $\geq$ 6 weeks old and children. It is* ***not*** *used in neonates due to the risk of brain toxicity outweighing benefits.*
- ***Why:*** *It significantly reduces the risk of hearing loss and mortality, particularly for Hib and S. pneumoniae infections.*
- ***Timing:*** *Must be given* ***10 to 20 minutes before or at the same time*** *as the first antibiotic dose. Stop the steroids early if CSF testing confirms a viral or non-bacterial cause.*

7. Complications & Prognosis

Even with treatment, meningitis carries a death rate of 3–5%. Major complications include:

- ***Hearing deficits:*** *Occur in 7–30% of survivors.*
- ***Neurological damage:*** *Decreased IQ (30–50%), seizures, hemiparesis, and subdural effusions.*
- ***Secondary fevers:*** *Occur in 15-20% of patients and may indicate inadequate treatment, drug fever, or a secondary hospital-acquired infection.*

8. Prevention & Prophylaxis

- ***Vaccines:*** *Highly effective vaccines exist and are recommended for Hib, S. pneumoniae (PCV), and N. meningitidis (MenACWY and MenB).*
- ***Chemoprophylaxis (Preventative Meds):*** *Given to close contacts to prevent spread.*
    - ***Meningococcal:*** *Give Rifampin, Ceftriaxone, or Ciprofloxacin to close contacts (household members, daycare contacts, flight passengers seated nearby for >8 hours, or anyone exposed to oral secretions) ideally within 24 hours.*
    - ***Hib:*** *Give Rifampin daily for 4 days to all household contacts if there is an unimmunized child under 4 years old present. This reduces bacterial carriage by >95%.*

Approach to child with febrile illness

Definition and Classification

- ***Fever:*** *A rise in internal body temperature above the normal 37.5°C. It occurs when the hypothalamus increases the body's temperature "set-point".*
- ***Hyperthermia:*** *A dangerous rise in core body temperature that exceeds the hypothalamus's set-point and regulation capabilities (>41°C).*
- ***Temperature Categories:*** *Fevers are classified as low-grade (37.3–38.0°C), moderate-grade (38.1–39.0°C), high-grade (39.1–41.0°C), and hyperthermia (>41.0°C).*
- ***Measurement Variances:*** *Normal baseline readings vary by the measurement site. Axillary is typically 35.97°C, oral is 36.5°C, tympanic is 36.6°C, and rectal is 37.0°C. Relying solely on "feeling warm" is inaccurate and unreliable.*

The Pathogenesis and Benefits of Fever *Fever is generally beneficial because it inhibits the growth of bacteria and the replication of viruses. It also enhances immunological processes, including T-cell and B-cell activity, immunoglobulin synthesis, and the phagocytic killing of bacteria by leukocytes (which is significantly greater at temperatures >40°C).*

The process begins with ***exogenous pyrogens*** *(like microorganisms, foreign proteins, or bacterial toxins). These trigger monocytes, macrophages, and neutrophils to release* ***endogenous pyrogens*** *(such as IL-1, IL-6, and TNF). These cytokines increase PGE2 synthesis, which directly tells the hypothalamus to increase its temperature set-point.*

Clinical Assessment and Crucial History

- ***Emergency Questions:*** *Ask about the child's ability to swallow (pharyngitis/tonsillitis), vomiting (which could indicate increased intracranial pressure), and convulsions (meningitis).*
- ***Vaccination History:*** *Post-vaccination fever is common. For DPT, it typically occurs after 24 hours, and for MMR, after 5-7 days. However, in a visibly unwell child, the fever should not be attributed to vaccination alone.*
- ***Important Context:*** *Check for recent travel, sick contacts, incomplete immunizations, and prior antibiotic use, which can mask the signs of a bacterial infection.* ***Note:*** *Teething does not cause fever.*
- ***High-Risk Patients:*** *Children with prematurity, immunosuppression, central lines, chronic lung disease, or congenital heart disease require special attention.*

Red Flags: Factors Suggesting Serious Infection *Immediate life-threatening features must be ruled out using a "Traffic light system" to predict the risk of serious illness. "Red" warning signs include:*

- *Age younger than one month or under three months with a temperature ≥38°C.*
- *Poor arousal, no response to social cues, or inability to stay awake.*
- *Petechial (non-blanching) rash.*
- *Delayed capillary refill or poor perfusion (mottling, pallor, cyanosis, ashen color).*
- *Increased respiratory effort (grunting or severe chest indrawing).*
- *Neurological signs like neck rigidity, bulging fontanelle, focal seizures, or status epilepticus.*

Age-Specific Approaches

- ***Under 2 Months:*** *Serious infections in this group include neonatal sepsis, severe pneumonia, meningitis, urinary tract infections (UTIs), and enterocolitis. The most common bacterial pathogen in this age group is Group B beta-streptococcus.*
- ***2 Months to 5 Years:*** *Causes usually range from respiratory (otitis media, pneumonia) and GI infections to UTIs and CNS infections.* ***Crucial Protocol:*** *If a patient in this age group looks visibly sick, they require a complete sepsis work-up, intravenous antibiotics, and cultures of the blood, urine, and stool (if blood/mucus is present).*

Differentiating Specific Conditions

- ***Pneumonia (Viral vs. Bacterial):*** *Clinical judgment is the best guide.* ***Viral pneumonia*** *starts gradually with general malaise, wheezing, watery cough, and diffuse bilateral lung involvement on an X-ray.* ***Bacterial pneumonia*** *starts abruptly with high fever, chills, a productive cough containing pus or blood, and localized lung consolidation. Both are characterized by tachypnea: >60 breaths/min (<2 months), >50 breaths/min (2-12 months), and >40 breaths/min (>12 months).*
- ***Fever of Unknown Origin (FUO):*** *Defined as a documented fever lasting over one week with no apparent diagnosis after one week of hospital investigation. It is often caused by atypical presentations of common diseases, systemic infectious, or collagen vascular diseases like Juvenile Rheumatoid Arthritis and Systemic Lupus Erythematosus.*
- ***Kawasaki Disease:*** *Requires clinical judgment as it often presents with concurrent infections. Diagnosis requires a fever lasting at least 5 days combined with at least 4 of the following: bilateral bulbar conjunctival injection, oral mucous membrane changes*

(strawberry tongue, injected/fissured lips), peripheral extremity changes (edema or desquamation), polymorphous rash, and cervical lymphadenopathy (>1.5 cm).

Febrile Illnesses Accompanied by Rash

- ***1st Disease:*** *Rubella.*
- ***2nd Disease (Scarlet Fever):*** *Caused by Group A streptococcus, usually affecting ages 5-15. Starts with flu-like symptoms and swollen neck glands, followed 12-48 hours later by a rash of small raised bumps on the chest/tummy that feels rough like sandpaper.*
- ***3rd Disease:*** *Chickenpox (causes a vesicular rash).*
- ***4th Disease:*** *Measles (features Koplik spots inside the mouth).*
- ***5th Disease (Erythema Infectiosum):*** *Affects school-age children. Starts with a low-grade fever followed by a "slapped cheek" appearance. The rash fades with central clearing, giving a reticulated look.*
- ***6th Disease:*** *The sources refer to both* ***Roseola infantum*** *(characterized by a high fever for 1-5 days followed by an abrupt drop in temperature and the immediate appearance of a quickly fading rash) and* ***Typhoid fever*** *(presenting with "rose spots") under the umbrella of the 6th disease.*

Investigations and Management

- ***Labs:*** *For ill children, check absolute neutrophil counts (ANC), white blood cells (WBC), and perform cultures of blood, urine, CSF, and sputum. Check blood glucose, ensuring it is >40mg/dl in newborns and >60 mg/dl in children.*
- ***Procalcitonin (PCT):*** *This is a highly specific, promising biomarker for early detection of systemic bacterial infections. It rises much more rapidly than CRP or ANC in response to bacterial infections and is not elevated in viral infections.*
- ***Antibiotic Stewardship:*** *Only prescribe antibiotics when a bacterial infection is highly suspected or confirmed. Liberal use promotes dangerous antibiotic resistance.*
- ***Advice to Families:*** *For fever management at home, advise caregivers to offer plenty of fluids, give lukewarm sponge baths, dress the child in lightweight/breathable clothes, use a light sheet for comfort, and use recommended fever-reducing medications. Caregivers must* ***always*** *call a pediatrician if a baby under 3 months of age develops a fever.*

Upper Respiratory Tract Infections

1. The Common Cold (Viral Rhinosinusitis)

- ***Definition & Etiology:*** *A viral illness dominated by rhinorrhea and nasal obstruction, with mild or absent systemic symptoms like fever. The most frequent culprits are rhinoviruses, though coronaviruses, respiratory syncytial virus (RSV), influenza, parainfluenza, and adenoviruses also cause it.*
- ***Epidemiology:*** *Children average 6–7 colds per year (decreasing to 2–3 in adults), with higher rates in daycare. Rhinovirus peaks in early fall and late spring, while parainfluenza peaks in late fall, and RSV/influenza peak in winter.*
- ***Pathogenesis:*** *Transmission occurs via small and large particle aerosols, as well as direct contact. Rhinoviruses and coronaviruses cause no apparent histologic damage to the nasal epithelium, whereas influenza and adenoviruses actively destroy the nasal epithelial lining.*
- ***Clinical Presentation:*** *Symptoms begin 1–3 days post-infection, starting with a scratchy throat, followed by nasal obstruction and rhinorrhea. Cough develops in about 30% of cases, while fever is more common with influenza, adenoviruses, and RSV.*
- ***Diagnosis & Treatment:*** *Diagnosis involves ruling out allergic rhinitis, foreign bodies, and sinusitis; routine labs and viral detection are not helpful. Treatment is purely symptomatic: topical decongestants like oxymetazoline (which can cause rebound rhinitis medicamentosa if overused) and first-generation antihistamines to reduce rhinorrhea. Aspirin is contraindicated in children due to Reye syndrome risk, and cough suppressants/expectorants, Vitamin C, and zinc are largely ineffective.*
- ***Complications:*** *Otitis media is the most common (5–30%), followed by sinusitis (5–13% in children) and asthma exacerbation.*

2. Acute Bacterial Sinusitis

- ***Pathophysiology:*** *Often occurs as a complication of a viral URTI when inflammation and edema block sinus drainage and impair mucociliary function. Nose blowing forces virus-laden nasal secretions into the normally sterile sinus cavities.*
- ***Etiology:*** *The primary bacterial agents are Streptococcus pneumoniae (30%), non-typable Haemophilus influenzae (20%), and Moraxella catarrhalis (20%).*
- ***Clinical Presentation:*** *Nonspecific symptoms include nasal congestion, cough, and fever. It is diagnosed clinically based on symptoms persisting for >10–15 days without improvement, or severe symptoms (temperature >39°C and purulent nasal discharge) lasting 3–4 days. Headache and facial pain are rare in children.*
- ***Treatment & Complications:*** *Initial treatment is Amoxicillin (45 mg/kg/day); frontal sinusitis initially requires Ceftriaxone. Severe complications include periorbital/orbital cellulitis, intracranial infections (meningitis, brain abscess, cavernous sinus thrombosis), and bone infections like osteomyelitis of the frontal bone (Pott Puffy Tumor).*

3. Acute Pharyngitis

- ***Etiology:*** *Viruses cause the majority of cases, while Group A Beta-hemolytic strep (GABHS) is the primary bacterial cause.*
- ***Clinical Manifestation (GABHS vs. Viral):***
    - *GABHS: Rapid onset of sore throat, fever, headache, and GI symptoms. Signs include a red pharynx, enlarged tonsils with yellow/blood-tinged exudate, palatal petechiae, and tender anterior cervical lymph nodes. A specific manifestation is scarlet fever, characterized by a strawberry tongue, circumoral pallor, and a fine red "sandpaper" rash.*
    - *Viral: Gradual onset with concurrent rhinorrhea, cough, and diarrhea. Specific viruses have unique signs: Coxsackievirus causes grayish vesicles (herpangina), EBV causes massive tonsils and hepatosplenomegaly (infectious mononucleosis), and Primary Herpes Simplex causes high fever and gingivostomatitis.*
- ***Diagnosis & Treatment:*** *Throat culture or rapid antigen testing is needed to identify GABHS. The primary goal of antibiotic therapy (Penicillin V or Amoxicillin) is to prevent Acute Rheumatic Fever (ARF), which is highly effective if given within 9 days of illness onset. Tonsillectomy is considered for frequent, severe recurrent infections.*

4. Tonsillar and Adenoidal Disease

- ***Chronic Infection:*** *Polymicrobial infections (often involving beta-lactamase-producing organisms and anaerobes) cause accumulated debris in tonsillar crypts, leading to halitosis, chronic sore throats, and a foreign body sensation.*
- ***Airway Obstruction:*** *Enlarged tonsils and adenoids can cause chronic mouth breathing, hyponasal speech, loud snoring, apnea, and poor school performance. Treatment is adenotonsillectomy.*

5. Deep Neck Space Infections (Abscesses)

- ***Retropharyngeal Abscess:*** *Typically seen in boys under 3–4 years old, presenting with fever, irritability, drooling, neck stiffness (torticollis), and a bulging posterior pharyngeal wall.*
- ***Lateral Pharyngeal Abscess:*** *Presents with fever, dysphagia, and a prominent lateral wall bulge that may displace the tonsil medially.*
- ***Peritonsillar Cellulitis/Abscess:*** *A deep infection commonly seen in adolescents following acute tonsillitis, marked by severe sore throat, trismus (lockjaw), dysphagia, and an asymmetrical bulging of the tonsil that displaces the uvula.*
- ***Treatment:*** *These require IV antibiotics (e.g., third-generation cephalosporins + clindamycin/ampicillin-sulbactam) and frequently surgical incision/drainage, especially if airway distress is present.*

6. Infectious Upper Airway Obstruction (Croup vs. Epiglottitis)

- ***Croup (Laryngotracheobronchitis):***
    - *Overview: The most common cause of infectious upper airway obstruction, typically viral (Parainfluenza types 1-3 account for 75%), affecting children aged 5 months to 5 years.*
    - *Presentation: Starts as a mild URTI with low-grade fever, evolving over 1-3 days into a characteristic "barking" cough, hoarseness, and inspiratory stridor. Symptoms worsen at night and with agitation/crying.*
    - *Diagnosis & Treatment: X-rays may show a characteristic "steeple sign". Treatment involves cool mist, nebulized epinephrine (if stridor is present at rest or hypoxia occurs), and a single dose of dexamethasone.*
- ***Acute Epiglottitis:***
    - *Overview: A dramatic, potentially lethal bacterial infection historically caused mostly by H. influenzae type b, though now S. pyogenes and S. pneumoniae are rising causes due to vaccines.*
    - *Presentation: Fulminating (rapid) onset over mere hours. The child is highly toxic, with high fever, difficult swallowing, severe drooling, and dyspnea. They often adopt a "tripod position" (sitting upright, leaning forward, chin up) to maintain an airway. Stridor is a late sign of near-complete airway obstruction.*
    - *Diagnosis & Treatment: A lateral X-ray shows a classic "thumb sign", and laryngoscopy reveals a "cherry-red" swollen epiglottis.* ***Crucial Rule:*** *Avoid all anxiety-provoking actions (e.g., forcing them supine, inspecting the mouth, starting IVs prematurely) as this can trigger fatal airway spasms. It is a medical emergency requiring an immediate artificial airway (nasotracheal intubation in an ICU/theater setting) regardless of distress level, followed by IV Ceftriaxone or Cefotaxime. Contacts may require Rifampin chemoprophylaxis.*

Lower respiratory disorders & infections

Part 1: Pediatric Pneumonia

1. Definition and Classifications

- ***Definition****: An acute infection of the lung's pulmonary parenchyma. It accounts for 14% of all deaths in children under 5 worldwide.*
- ***Anatomical Types****:*
    - ***Bronchopneumonia****: Patchy consolidation usually in the basal (dependent) lung zones.*
    - ***Lobar Pneumonia****: Affects one or more lobes, often leaving bronchi air-filled (air bronchograms).*
    - ***Interstitial Pneumonia****: Diffuse inflammation of the lung interstitium, characterized by lymphocyte and macrophage infiltration.*
    - ***Congenital Pneumonia****: Presents within the first 24 hours of birth.*

2. Causes (Etiology) by Age Group *The cause of pneumonia heavily depends on the child's age:*

- ***First 2 Months****: Klebsiella, E. coli, and Staphylococci.*
- ***3 Months to 3 Years****: S. pneumoniae, H. influenzae, and Staphylococci.*
- ***Over 3 Years****: S. pneumoniae and Staphylococci.*
- ***Atypical Organisms****: Mycoplasma (most common in ages 5–20 with gradual onset of headache, fever, and sore throat) and Chlamydia.*
- ***Immunocompromised****: Pneumocystis carinii.*

3. Signs and Symptoms

- ***Respiratory Distress****: Hallmarks include retractions, grunting, nasal flaring, and hypoxia (SpO2 < 90%).*

- ***Tachypnea (Fast Breathing)**: Defined by the WHO as:*
    - *0–2 months: > 60 breaths/min.*
    - *2–12 months: > 50 breaths/min.*
    - *1–5 years: > 40 breaths/min.*
    - *5 years: > 20 breaths/min.*
- ***Physical Exam**: Signs of consolidation include bronchial breathing, dullness to percussion, and increased tactile vocal fremitus. Note: Pneumonia can also present with acute abdominal pain referred from the pleura.*

4. Diagnosis

- ***Chest X-Ray (CXR)**: Confirms diagnosis. Viral pneumonia shows hyperinflation and interstitial infiltrates, pneumococcal pneumonia shows lobar consolidation, and staphylococcal pneumonia can cause pneumatoceles (cavities).*
- ***Blood Tests**: Help differentiate between viral (WBC normal or slightly elevated < 20,000 with lymphocytosis) and bacterial (WBC elevated 15,000–40,000 with neutrophilia).*

5. Management and Treatment

- ***Hospitalization Criteria**: Admit children with moderate-to-severe disease (SpO2 < 90%), infants under 3–6 months with suspected bacterial pneumonia, cases involving highly virulent pathogens (like MRSA), or cases with home observation concerns.*
- ***Outpatient Antibiotics**: **Amoxicillin** (or amoxicillin-clavulanate) for bacterial; **Azithromycin** or other macrolides for atypical.*
- ***Inpatient Antibiotics**: **Ampicillin or Penicillin G** (alternatives: ceftriaxone/cefotaxime) for bacterial; add Vancomycin/Clindamycin if MRSA is suspected. Macrolides are used for atypical cases.*

Part 2: Cystic Fibrosis (CF)

1. Definition and Pathophysiology

- ***What it is**: A multisystem, inherited genetic disorder causing chronic, progressive obstructive lung disease and severe nutritional issues.*
- ***Genetics**: Caused by a mutation in the **CFTR gene** on the long arm of chromosome 7 (most common mutation: delta F508).*

- ***The Core Problem****: Defective ion transport leads to depleted airway surface liquid, thickened mucus, and defective clearance. This thick mucus obstructs organs, leading to chronic infection and inflammation.*

2. Diagnosis *Diagnosis requires clinical features, family history, or a positive newborn screen,* ***PLUS*** *evidence of CFTR dysfunction:*

- ***Sweat Chloride Test****: The most useful diagnostic test. A level of* ***≥ 60 mmol/L confirms CF****.*
- ***Other tests****: Newborn screening (looks for high IRT levels), genetic testing, or abnormal nasal potential difference.*

3. Clinical Manifestations and Complications *CF affects multiple organ systems:*

- ***Respiratory****: Chronic sinusitis, nasal polyps (25% need surgery), and a persistent cough with thick, green sputum. Lung infections initially involve H. influenzae and S. aureus, but eventually, Pseudomonas aeruginosa becomes dominant. Severe complications include pneumothorax, hemoptysis, and respiratory failure.*
- ***Gastrointestinal (GI)****:*
    - ***Exocrine Pancreatic Insufficiency*** *(>90% of patients), which leads to severe protein/fat malabsorption, bulky foul-smelling stools, and deficiency in fat-soluble vitamins (A, D, E, K).*
    - *Infants may have* ***Meconium ileus*** *(failure to pass stool, abdominal distention).*
    - *Older children may develop* ***DIOS*** *(Distal Intestinal Obstruction Syndrome) which mimics appendicitis.*
- ***Genitourinary****: Delayed puberty. Over* ***95% of males are infertile*** *(azoospermia due to absent vas deferens), and 20% of females are infertile.*

4. Treatment and Management *Treatment requires a high daily burden and focuses on clearing secretions, fighting infections, and improving nutrition:*

- ***Lungs****: Long, high-dose antibiotic courses. For Pseudomonas, two drugs are used to prevent resistance (e.g., Ceftazidime + an aminoglycoside). Mucus clearance is improved with* ***DNAse treatments****, inhaled bronchodilators, and chest physiotherapy.*
- ***Gastrointestinal****:* ***Pancreatic enzyme replacement*** *(Creon) and fat-soluble vitamin (A, D, E, K) supplementation are crucial. Insulin is used if CF-related diabetes develops. Liver transplants are considered for end-stage liver disease.*

UTI in Children

1. Epidemiology and Pathogenesis

- ***Prevalence:*** *By age 11, UTIs affect 8% of girls and 2% of boys.*
- ***Age and Sex Factors:*** *The highest incidence occurs during the first year of life. In the neonatal period, males (especially uncircumcised) are more affected than females.*
- ***Recurrence:*** *Approximately 30% of children will experience a recurrent UTI within the first 6 to 12 months after the initial infection.*
- ***Pathogenesis:*** *Infections typically ascend from periurethral colonization, while hematogenous (bloodborne) spread is rare. The bacteria usually originate from the bowel or from under the foreskin in boys.*
- ***Causative Organisms: E. coli*** *causes 80% of infections, largely due to its strong adhesive capacity. Other typical organisms include Klebsiella, Enterobacter, Enterococcus, Proteus, and Pseudomonas.*

2. Clinical Assessment (History & Physical Exam)

Key History to Gather:

- ***Urinary Symptoms:*** *Dysuria (burning/pain on urination), urgency, frequency, and urge incontinence.*
- ***Other Symptoms:*** *Hematuria (blood in urine), abdominal or loin pain, and fever.*
- ***Habits & Associations:*** *Bowel habits (frequency/consistency), voiding volume, holding maneuvers, relation to toilet training, history of previous UTIs, and vaginal discharge or itching.*
- ***Family History:*** *History of UTIs in siblings, stones, or specific dietary habits.*

Physical Examination Focus:

- *Temperature and signs of renal angle or suprapubic tenderness.*
- *Genitalia exam checking for vulvitis, discharge, meatal stenosis, or labial adhesions.*
- *Inspection of the back for a sacral dimple (which can indicate neurological bladder dysfunction).*
- *Palpation for a distended bladder or impacted stool.*

3. Differential Diagnosis for Dysuria

1. ***Genitourinary infections:*** *Pyelonephritis, cystitis, urethritis.*
2. ***Vulvovaginitis****.*
3. ***Chemical irritation:*** *From soaps, poor hygiene, or improper wiping.*
4. ***Stones:*** *Associated with hypercalciuria, hyperoxaluria, or hyperuricosuria.*
5. ***Other causes:*** *Labial adhesions, trauma, or sexual abuse.*

4. Diagnosis and Testing

Specimen Collection:

- ***Recommended methods:*** *Urethral catheterization, suprapubic aspiration (SPA), or a clean catch midstream sample in toilet-trained children.*
- ***Not recommended:*** *Urine bags have a high risk of contamination and yield high false-positive results.*

Urinalysis & Culture:

- ***Pyuria:*** *Defined as more than 5 white blood cells per High Power Field (HPF).*
- ***AAP Guidelines for Diagnosis:*** *A confirmed UTI requires* ***both*** *a urinalysis suggesting infection (pyuria and/or bacteriuria)* ***and*** *at least 50,000 colony-forming units (CFUs) per mL of a uropathogen from a catheterized or SPA specimen.*
- ***Important Caveat:*** *A negative urinalysis in a symptomatic patient does* ***not*** *completely rule out a UTI.*

Predictive Risk Factors for UTI: *A clinical calculator uses specific risk factors to estimate the probability of a UTI: age under 12 months, female sex or uncircumcised male, temperature $\ge$ 39°C, fever lasting $\ge$ 48 hours, and the absence of another source of fever.*

5. Diagnostic Pitfalls & Special Conditions

- ***Common Diagnostic Errors:*** *Using contaminated bag specimens (feces/skin), accepting cultures with "mixed growth" (more than 2 pathogens), or treating typical skin contaminants like viridians streptococci or coagulase-negative staphylococcus as true infections.*
- ***Asymptomatic Bacteriuria:*** *This is harmless bladder colonization without inflammation, occurring in 1-3% of children. It usually resolves spontaneously, is common in children with neurogenic bladders (e.g., those using catheters or with bladder augmentation), and* ***antibiotic treatment is not recommended****.*
- ***Sterile Pyuria:*** *White blood cells in the urine without bacterial growth can be caused by appendicitis, viral infections, partially treated UTIs, immunologic conditions (like glomerulonephritis or Kawasaki disease), stones, interstitial nephritis, vaginitis, or general febrile illness.*

6. Complications: VUR and Renal Scarring

Vesicoureteral Reflux (VUR):

- ***What is it?*** *The backward flow of urine. It is found in 33% of UTI cases.*
- ***Causes:*** *Can be primary or secondary to posterior urethral valves (PUV) or neurogenic bladder. It is also associated with renal agenesis, ectopia, and duplex kidney.*
- ***Genetics:*** *Siblings of a child with VUR have a 27-45% incidence of having it as well.*
- ***Management:*** *Treated with antimicrobial prophylaxis, or surgical correction (open reimplantation or endoscopic treatment) for high-grade VUR, recurrent UTIs despite prophylaxis, or worsening renal scars.*

Renal Scarring:

- *10-40% of patients develop renal scarring.*
- *Scarring can ultimately lead to serious long-term issues: proteinuria, hypertension, and chronic kidney disease.*
- ***Imaging:*** *A DMSA scan done acutely helps confirm pyelonephritis, but to assess for permanent scarring, it should be done 4-6 months after the UTI resolves. Furthermore, 30% of prenatal VUR cases show abnormal DMSA scans (dysplasia).*

7. Management and Surveillance

- ***Clinical Course:*** *Patients typically become afebrile after 48 hours of treatment. If fever persists, providers should consider upgrading antibiotics to cover resistant strains or evaluate for complications.*
- ***Treatment Transitions:*** *It is acceptable to transition from intravenous to oral antibiotics.*
- ***Follow-up:*** *Routine follow-up urine cultures after symptom resolution are* ***not*** *necessary.*
- ***Prophylaxis Note:*** *Long-term antibiotic prophylaxis carries a major risk of antibiotic resistance. There is no clinical evidence supporting the use of cranberry products to prevent UTIs in children. Ensure underlying constipation and bladder dysfunction are treated.*
- ***Action Statement:*** *Parents should be instructed to seek prompt medical evaluation (ideally within 48 hours) for any future febrile illnesses to catch and treat recurrent infections early.*

Acute Kidney Injury (AKI) and (CKD) in children

PART 1: ACUTE KIDNEY INJURY (AKI)

The "Sudden Stop" of Kidney Function

1. Definition and Diagnosis

- ***What it is:*** *AKI is an abrupt loss of kidney function leading to a decline in Glomerular Filtration Rate (GFR), which causes the retention of urea and nitrogenous wastes, along with dysregulation of fluid volume and electrolytes.*
- ***Why it matters:*** *It is an independent risk factor for prolonged ICU stays, requires longer mechanical ventilation, and has a high risk of progressing to CKD, hypertension, and proteinuria.*
- ***Diagnosis:*** *Clinicians rely on clinical signs, serum creatinine levels, and urine output monitoring, utilizing classification systems like pRIFLE and KDIGO.*
- ***The Biomarker Problem:*** *Serum creatinine is actually a poor and delayed marker; it may not rise until up to 50% of kidney function is already lost, and it is affected by a child's muscle mass and hydration status. Novel biomarkers (like NGAL, KIM-1, IL-18, and Cystatin C) show promise for early detection but are not frequently used due to their high cost.*

2. The Three Classifications (Causes)

- ***Prerenal (Most Common):*** *Caused by reduced blood flow to the kidneys.*
    - *Causes: True volume depletion (bleeding, burns, gastroenteritis) or decreased effective blood volume (heart failure, septic shock).*
    - *Mechanism: The kidney tubules still work fine; they just aren't getting blood. The kidney tries to compensate by using prostaglandins to dilate incoming vessels and Angiotensin II to constrict outgoing vessels to keep filtration pressure up.* ***Crucial Point:*** *NSAIDs (like Ibuprofen) block prostaglandins, and ACE inhibitors block Angiotensin II; using these medications during hypoperfusion can precipitate AKI.*
- ***Intrinsic:*** *Caused by direct structural damage to the kidney tissue.*
    - *Causes: Prolonged lack of blood flow leading to Acute Tubular Necrosis (ATN), sepsis, vasculitis, or glomerular diseases like Hemolytic Uremic Syndrome (HUS) and Post-Streptococcal Glomerulonephritis (PSGN).*

- ***Postrenal:*** *Caused by a physical blockage preventing urine from leaving the body.*
    - *Causes: Congenital anomalies (like Posterior Urethral Valves), stones, blood clots, or strictures.*

3. Clinical Presentation & Investigations

- ***Symptoms:*** *Edema (fluid accumulation), high blood pressure, decreased or absent urine output (anuria/oliguria), and hematuria (blood in urine). Oliguria is defined as urine output <1 mL/kg/hr in infants and <0.5 mL/kg/hr in children.*
- ***Key Labs to Order:*** *Kidney Function Tests (creatinine, urea), electrolytes (watch for dangerous hyperkalemia, hyponatremia, hypocalcemia, hyperphosphatemia), Venous Blood Gas for metabolic acidosis, Complete Blood Count, and Urinalysis looking for casts or protein.*

4. Management of AKI *There are no medications that cure established AKI; management is entirely supportive and preventative.*

- ***Fluid Management:*** *This is critical. If hypovolemic (dry), give a normal saline bolus (10-20 mL/kg). If euvolemic, replace insensible losses and give diuretics. If hypervolemic (fluid overloaded by >20%), restrict fluids, use Furosemide, and consider dialysis.*
- ***Hyperkalemia Emergency:*** *High potassium (>7.0 meq/L) causes dangerous ECG changes, starting with tall peaked T waves.* ***Treatment steps:*** *Give Calcium gluconate to stabilize the heart immediately, followed by glucose/insulin or albuterol to push potassium into cells, and ultimately use Furosemide or dialysis to remove potassium from the body.*
- ***Other Treatments:*** *Treat acidosis with Sodium Bicarbonate, manage hypertension with Calcium Channel Blockers, and adjust all drug doses to match the reduced kidney function.*

PART 2: CHRONIC KIDNEY DISEASE (CKD)

The "Long-Term Decline" of Kidney Function

1. Definition and Epidemiology

- ***What it is:*** *Structural or functional kidney abnormalities persisting for at least* ***3 months****, with or without a decreased GFR. Note: This definition is not used for children under 2 years old.*
- ***Who gets it:*** *It is more common in males due to a higher frequency of congenital anomalies. The main causes in children include obstructive uropathy, renal hypoplasia/dysplasia, and childhood nephritis syndromes like FSGS.*

2. Pathophysiology and Progression

- ***The "Adaptive Hyperfiltration" Trap:*** *When kidneys are damaged, the remaining healthy nephrons work overtime (hyperfiltration) to compensate. This keeps labs (like creatinine and electrolytes) normal in mild CKD. However, over time, this overwork destroys the remaining glomeruli, leading to proteinuria and irreversible failure.*
- ***Outcomes:*** *About 70% of children with CKD will develop End-Stage Renal Disease (ESRD) by age 20. The most common cause of death in these children is cardiovascular disease.*

3. Staging of CKD *Staging is based on GFR levels:*

- ***Stage 1:*** *GFR $\ge$ 90 (Kidney damage but normal filtration)*
- ***Stage 2:*** *GFR 60-89 (Mild)*
- ***Stage 3A/3B:*** *GFR 30-59 (Moderate)*
- ***Stage 4:*** *GFR 15-29 (Severe)*
- ***Stage 5:*** *GFR < 15 (Kidney Failure / ESRD requiring dialysis)*

4. Complications & How to Treat Them

- ***Anemia:*** *The failing kidneys stop producing Erythropoietin (EPO), leading to early and near-universal anemia in severe CKD.* ***Treatment:*** *Give EPO injections and Iron supplements to maintain a Hemoglobin level of 11-12 g/dL.*
- ***CKD-Mineral and Bone Disorder (CKD-MBD):*** *Kidneys fail to excrete phosphate and fail to activate Vitamin D. This leads to low calcium, triggering Secondary Hyperparathyroidism (high PTH) which pulls calcium from bones, causing skeletal deformities, fractures, and poor growth.* ***Treatment:*** *Restrict dietary phosphate, give calcium-based phosphate binders, and provide active Vitamin D. Check PTH every 3 months (target PTH in Stage 5 is 200-300 pg/mL).*
- ***Hypertension:*** *A massive predictor of CKD progression.* ***Treatment:*** *The target is to keep blood pressure below the 90th percentile for age. ACE inhibitors or ARBs are the drugs of choice because they slow renal injury and reduce proteinuria.*
- ***Metabolic Acidosis:*** *Occurs mostly when GFR drops below 30 (Stage 4).* ***Treatment:*** *Supplement with Sodium Bicarbonate to keep HCO3 levels around 22 mmol/L.*
- ***Growth Retardation:*** *Children with CKD suffer from short stature and failure to thrive.* ***Treatment:*** *Address feeding issues, give Growth Hormone supplements, and optimize dialysis.*

PART 3: RENAL REPLACEMENT THERAPY (RRT)

When the Kidneys Give Out completely

When to start RRT (Indications): *Dialysis is required when medical therapy fails. Triggers include fluid overload unresponsive to diuretics, unmanageable hyperkalemia, severe metabolic acidosis, BUN reaching 80-100 mg/dL, or life-threatening complications like pulmonary edema.*

The Three Modalities: *There is no proof that one type is universally better than the others for AKI outcomes, so the choice depends on the specific child.*

1. ***Hemodialysis (HD):*** *Uses a machine to filter blood. It is very difficult for very small children and infants because it requires central vascular access and a large volume of blood to be outside the body at once.*
2. ***Peritoneal Dialysis (PD):*** *Uses the lining of the child's abdomen as a filter. It is easy to perform, doesn't require complex equipment or blood thinners, and is the* ***therapy of choice for neonates and small infants****.*
3. ***Continuous Renal Replacement Therapy (CRRT):*** *A slow, continuous 24/7 dialysis. It is the best choice for patients in the ICU with hemodynamic instability (unstable blood pressure) because it prevents the massive, rapid fluid shifts seen in standard HD.*

Anemia

1. Core Concepts & Classification

Anemia is defined as a reduction in red blood cell (RBC) volume or hemoglobin (Hb) concentration below the normal range for a healthy person of the same age.

In infants, normal physiology dictates that newborns have higher Hb, experience "physiologic anemia" at 2-3 months, and premature babies suffer from an exaggerated form of this anemia.

Anemias are broadly classified into four categories:

- ***Inadequate Intake:*** *Deficiency in Iron, Vitamin B12, or Folic Acid.*
- ***Production (Bone Marrow) Defects:*** *Congenital pure red cell anemia, ineffective erythropoiesis, or anemia secondary to infections, renal failure, or cancer.*
- ***Hemolytic Anemias:*** *Caused by RBC membrane defects (Spherocytosis, Elliptocytosis), Enzyme defects (G6PD, Pyruvate Kinase), Hemoglobin synthesis defects (Sickle cell/HbS, Thalassemia), or Immunologic destruction (Rh/ABO incompatibility).*
- ***Blood Loss:*** *Hemorrhage from any site.*

2. Nutritional Anemias

Iron Deficiency Anemia (IDA)

- ***Epidemiology:*** *The most common pediatric anemia (5.5% of children), peaking between 9–24 months of age.*
- ***Daily Requirement:*** *2 mg/kg/day.*
- ***Causes:*** *Inadequate diet (e.g., cow's milk), impaired absorption (e.g., Celiac disease), or chronic blood loss (e.g., Meckel's diverticulum, cow's milk allergy).*
- ***Lab Findings:*** *Microcytic, hypochromic RBCs, anisocytosis, and polychromasia.*
- ***Systemic Tissue Effects:***
    - *GI: Anorexia, pica, atrophic glossitis, dysphagia, guaiac-positive stool.*
    - *CNS: Irritability, fatigue, decreased activity, breath-holding spells.*
    - *Cardiovascular: Tachycardia, cardiac hypertrophy, heart failure.*
    - *Immune: Increased infection rates, impaired granulocyte killing.*

- ***Treatment Milestones:*** *The very* ***first response*** *to iron therapy is increased appetite and activity because essential metabolic enzymes require iron. This is followed by a rise in the reticulocyte count (Retic) after 3–5 days.*

Megaloblastic Anemia *Characterized by macrocytes (large RBCs) in the blood and megaloblasts in the bone marrow, accompanied by hypersegmented neutrophils and pancytopenia.*

- ***Folic Acid Deficiency:*** *Onset at 4–7 months. Caused by inadequate diet (e.g., drinking goat's milk), poor absorption, increased requirement (hemolysis), or metabolic disorders (MTHFR deficiency). Treatment: 5 mg Folic Acid tablet/day.*
- ***Vitamin B12 Deficiency:*** *Onset later, between 9 months and 10 years. Caused by diet (including maternal diet), defective intrinsic factor, GI malabsorption (e.g., Crohn's disease, surgery), defective transport (congenital TCII deficiency), or drugs.*
- ***Clinical Presentation:*** *Failure to thrive, generalized weakness, glossitis, anorexia, pallor, jaundice, and importantly,* ***neurologic manifestations*** *(ataxia, paresthesia, hyporeflexia).*
- ***Treatment:*** *1 mg Vitamin B12 IM daily for 2 weeks (especially if neurological defects are present), then monthly. Alternatively, 200-1000 microgram/week for 4 weeks, then monthly.*

3. Hemolytic Anemias

Hemolysis is the premature destruction of RBCs. Evidence of hemolysis includes decreased Packed Cell Volume (PCV), increased indirect bilirubin, increased reticulocyte count, and decreased haptoglobin.

Hereditary Spherocytosis (HS)

- ***Pathology:*** *Defect in RBC cell membrane proteins (spectrin, ankyrin). It is 75% autosomal dominant, 25% genetic mutation.*
- ***Clinical Features:*** *Ranges from mild to severe (10% of cases are severe). Key signs include splenomegaly, gallstones, and newborn jaundice/hemolytic anemia.*
- ***Diagnosis:*** *Low PCV/MCV, High MCHC/RDW/Retic, spherocytes on blood smear. Confirmed by a positive Osmotic Fragility test or flow cytometry (highly sensitive and specific).*

4. Chronic Transfusion & Chelation (e.g., Thalassemia)

*For severe anemias like Thalassemia, a **Hypertransfusion Protocol** is used to maintain a pre-transfusion Hb level > 10.5 g/dl.*

- ***Goals:** Maximize growth, minimize skeletal abnormalities and extramedullary hematopoiesis, reduce gut iron absorption, and reduce splenomegaly.*
- ***Iron Overload Risk:** Stems from ongoing transfusions, increased gut absorption, and chronic hemolysis.*
- ***Chelation Therapy:** Used to bind free iron, remove intracellular iron, and achieve a negative iron balance.*
    - *Desferal: Given SC/IV. Dose 40mg/kg. Highly selective and safe, but poor compliance.*
    - *Ferriprox: Given orally 3x/day. Dose 75mg/kg. Risks include neutropenia and joint problems.*
    - *Deferasirox (Exjade): Given orally 1x/day. Dose 20mg/kg. Risks include skin rashes and kidney toxicity.*

5. Aplastic Anemia & Transfusion Risks

- ***Aplastic Anemia Management:** Treated with Steroids, Androgens, Antithymocyte Globulin (ATG) + cyclosporine, or Bone Marrow Transplant (BMT). BMT is also used for Thalassemia, but faces challenges with rejection and regimen toxicity.*
- ***Blood Transfusion Side Effects:** Includes allergies, non-hemolytic febrile reactions, viral transmission, hemolytic reactions, and Transfusion Graft-Versus-Host Disease (GVHD).*

6. Diagnostic Quick-Reference Pearls

- *Pale 20-month-old: Key history elements include diet, GI symptoms/diarrhea, and family history. Basic labs to order are Hb, PCV, WBC, Platelets, MCV, RDW, and Retic count.*
- *Brown pigmentation on lips/gums + Iron Deficiency: Think Peutz-Jeghers Syndrome.*
- *Newborn with jaundice/anemia since birth (Mother AB+, Baby B+): Think ABO Incompatibility (differential includes Rh incompatibility, G6PD, spherocytosis, infection, or alpha thalassemia).*
- *Severe microcytic anemia + high HbS & HbF on electrophoresis: Diagnosis points toward Sickle Cell disease variants (e.g., HbSS).*

Rheumatology

1. The Basics of Pediatric Rheumatology

- ***The Focus:*** *It involves diagnosing and treating autoimmune and autoinflammatory conditions (e.g., Juvenile Idiopathic Arthritis, Systemic Lupus Erythematosus, Scleroderma, and Systemic Vasculitis).*
- ***The Golden Rule:*** *Most rheumatologic conditions lack a single definitive diagnostic test; therefore, a thorough clinical history and physical exam are the most essential tools.*
- ***Key Screening Tool:*** *The* ***pGALS*** *(pediatric Gait, Arms, Legs, and Spine) is a simple, quick, and validated musculoskeletal exam to help recognize joint problems early.*

2. Inflammatory vs. Mechanical Joint Pain

Distinguishing between these two types of pain is the first major step in diagnosis:

- ***Inflammatory Pain:***
    - *Worse in the morning; features significant morning stiffness or "gelling" after rest.*
    - *Movement and activity actually ease the symptoms.*
    - *Swelling is persistent (days to weeks) and may be accompanied by systemic features like fever, weight loss, or anorexia.*
- ***Mechanical Pain:***
    - *Usually worse with and after activity, often peaking in the evening or after school.*
    - *Morning stiffness is usually absent.*

3. Common Differential Diagnoses for Joint Pain

- ***Reactive Arthritis:*** *Very common following a viral illness, features joint swelling that lasts 2–3 weeks, and resolves on its own.*
- ***Septic Arthritis:*** *A medical emergency presenting with high fever and a hot, swollen joint; requires immediate joint aspiration and IV antibiotics.*
- ***Hypermobility-Related Pain:*** *Widespread pain (especially in the knees after exercise), normal inflammatory markers, and hypermobile joints. Treated with reassurance and physiotherapy.*

4. Juvenile Idiopathic Arthritis (JIA): The Core Definition

JIA is the most common chronic rheumatic childhood illness.

- ***Definition:*** *Arthritis in* ***one or more joints*** *lasting for* ***at least 6 weeks*** *in a child* ***under 16 years old****.*
- ***Nature:*** *It is primarily a clinical diagnosis of exclusion.*

5. The Major Subtypes of JIA

The ILAR classifies JIA into distinct subtypes. Here are the key features to memorize for each:

A. Oligoarticular JIA (The Most Common Subtype)

- ***Features:*** *Affects $\le$ 4 joints, asymmetrically. Usually involves medium-to-large joints like knees and ankles.*
- ***Demographic:*** *Typically affects females aged 1–3 years.*
- ***Crucial Risk:*** *Patients (especially the ~70% who are* ***ANA positive****) are at a high risk for* ***chronic uveitis*** *(eye inflammation). Because this eye disease is often asymptomatic, they require frequent slit-lamp eye screenings to prevent vision loss.*

B. Polyarticular JIA

- ***Features:*** *Affects $\ge$ 5 joints, including both large and small joints (commonly the C-spine, jaw/TMJ, shoulders, and fingers).*
- ***Sub-categories:***
    - *RF Negative: Affects mostly girls, has a biphasic age onset, and is ANA positive in ~50% of cases.*
    - *RF Positive: Affects older girls (ages 9–11). These patients often have positive anti-CCP antibodies (~60%) and are at risk for aggressive, erosive joint damage.*

C. Enthesitis-Related Arthritis (ERA)

- ***Features:*** *Involves both arthritis and* ***enthesitis*** *(inflammation where tendons and ligaments insert into bone, like the Achilles tendon or patella).*
- ***Key Markers:*** *Associated with being male (onset >6 years),* ***HLA-B27 positive****, sacroiliac joint (SIJ) tenderness, lumbosacral pain, and acute symptomatic uveitis.*

D. Psoriatic JIA

- ***Features:*** *Arthritis accompanied by clinical psoriasis, OR arthritis plus two minor criteria:* ***dactylitis*** *(swollen "sausage-like" fingers/toes),* ***nail pitting****, or a first-degree relative with psoriasis. Often affects the distal interphalangeal (DIP) joints of the hand.*

E. Systemic JIA (Still's Disease)

- ***Features:*** *Driven by severe systemic inflammation rather than isolated joint issues.*
- ***Classic Triad:***
    1. ***Quotidian fevers:*** *Spiking fevers 1–2 times a day, typically in the evening/night.*
    2. ***Rash:*** *A salmon-pink, evanescent (comes and goes), migratory rash that often appears alongside the fever.*
    3. ***Organ Involvement:*** *Hepatosplenomegaly (enlarged liver/spleen) and generalized lymphadenopathy.*
- ***Labs:*** *Extremely high inflammatory markers (ESR, CRP), high ferritin, and high fibrinogen.*

6. The Most Dangerous JIA Complication: MAS

Macrophage Activation Syndrome (MAS) *is a severe, life-threatening complication, most heavily associated with Systemic JIA.*

- ***Clinical Picture:*** *Persistent fever, coagulopathy (bleeding issues), and hemophagocytosis (macrophages eating other blood cells) visible on a bone marrow biopsy.*
- ***Paradoxical Labs to Memorize:*** *While ferritin and CRP stay dangerously high, the previously high* ***ESR and fibrinogen will suddenly drop*** *(due to consumption/liver dysfunction), accompanied by falling blood cell counts (cytopenia).*

هذه المحاضرات غير مشمولة بالملخص

Neonatology

1- Failure to Thrive done
2- Physical exam and physical signs in pediatrics
3- Approach to genetic & metabolic diseases 1&2
4- Pediatric History

GI

5-Acute GE done
6-Chronic diarrhea

Cardiology

7-HF
8-CHD

Neurology

9- Approach to Hypotonia

Respiratory

10- Stridor
11- Approach to Wheeze In Children

Nephrology

12- Dehydration and Electrolytes
13- Nephrotic syndrome